

THE WILEY ENCYCLOPEDIA OF

HEALTH PSYCHOLOGY



VOLUME ONE
Biological Bases of Health Behavior

Editor-in-Chief: Lee M. Cohen, Ph.D.
Edited by Robert H. Paul, Ph.D., Lauren E. Salminen, Ph.D.
& Jodi Heaps, Ph.D.

WILEY Blackwell

Editor-in-Chief
Cohen
Edited by
Cohen

THE WILEY ENCYCLOPEDIA OF
HEALTH PSYCHOLOGY

VOLUME
FOUR

WILEY
Blackwell

Editor-in-Chief
Cohen
Edited by
Cohen

THE WILEY ENCYCLOPEDIA OF
HEALTH PSYCHOLOGY

VOLUME
THREE

WILEY
Blackwell

Editor-in-Chief
Cohen
Edited by
Cohen

THE WILEY ENCYCLOPEDIA OF
HEALTH PSYCHOLOGY

VOLUME
TWO

WILEY
Blackwell

Editor-in-Chief
Cohen
Edited by
Cohen

THE WILEY ENCYCLOPEDIA OF
HEALTH PSYCHOLOGY

VOLUME
ONE

WILEY
Blackwell

The Wiley Encyclopedia of Health Psychology

The Wiley Encyclopedia of Health Psychology

Volume 1
Biological Bases of Health Behavior

Edited by

Robert H. Paul

Co edited by

**Lauren E. Salminen
Jodi Heaps**

Editor-in-Chief

Lee M. Cohen

WILEY Blackwell

This edition first published 2021
© 2021 John Wiley & Sons Ltd

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by law. Advice on how to obtain permission to reuse material from this title is available at <http://www.wiley.com/go/permissions>.

The right of Lee M. Cohen to be identified as the author of the editorial material in this work has been asserted in accordance with law.

Registered Offices

John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Office

111 River Street, Hoboken, NJ 07030, USA

For details of our global editorial offices, customer services, and more information about Wiley products visit us at www.wiley.com.

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Limit of Liability/Disclaimer of Warranty

While the publisher and authors have used their best efforts in preparing this work, they make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives, written sales materials or promotional statements for this work. The fact that an organization, website, or product is referred to in this work as a citation and/or potential source of further information does not mean that the publisher and authors endorse the information or services the organization, website, or product may provide or recommendations it may make. This work is sold with the understanding that the publisher is not engaged in rendering professional services. The advice and strategies contained herein may not be suitable for your situation. You should consult with a specialist where appropriate. Further, readers should be aware that websites listed in this work may have changed or disappeared between when this work was written and when it is read. Neither the publisher nor authors shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

Library of Congress Cataloging-in-Publication Data

Names: Paul, Robert D., editor.

Title: The Wiley encyclopedia of health psychology / edited by Robert Paul,
St. Louis MO, USA ; editor-in-chief, Lee Cohen, The University of
Mississippi, MS, USA.

Description: First edition. | Hoboken : Wiley-Blackwell, 2021. | Includes
index. | Contents: volume. 1. Biological bases of health behavior.

Identifiers: LCCN 2020005459 (print) | LCCN 2020005460 (ebook) | ISBN
9781119057833 (v. 1 ; cloth) | ISBN 9781119057833 (v. 2 ; cloth) | ISBN
9781119057833 (v. 3 ; cloth) | ISBN 9781119057833 (v. 4 ; cloth) | ISBN
9781119675785 (adobe pdf) | ISBN 9781119675761 (epub)

Subjects: LCSH: Clinical health psychology—Encyclopedias.

Classification: LCC R726.7 .W554 2021 (print) | LCC R726.7 (ebook) | DDC
616.001/903—dc23

LC record available at <https://lcn.loc.gov/2020005459>

LC ebook record available at <https://lcn.loc.gov/2020005460>

Cover image and design: Wiley

Set in 10/12.5pt ITC Galliard by SPi Global, Pondicherry, India

10 9 8 7 6 5 4 3 2 1

Contents

List of Contributors	ix
Foreword	xxvii
Preface Volume 1: Biological Bases of Health	xxix
Preface Volume 1: Biological Basis of Behavior	xxxi
Editor-in-Chief Acknowledgments	xxxiii
Brain Development from Conception to Adulthood <i>Jodi M. Heaps-Woodruff and David Von Nordheim</i>	1
Functional Anatomy: Types of Cells/Physiology <i>Patrick W. Wright</i>	5
Functional Neuroanatomy: Cortical–Subcortical Distinctions and Pathways <i>David Von Nordheim and Jodi M. Heaps-Woodruff</i>	13
Neurobiology of Stress–Health Relationships <i>R.L. Spencer, L.E. Chun, M.J. Hartsock, and E.R. Woodruff</i>	21
Stress, Neurogenesis, and Mood: An Introduction to the Neurogenic Hypothesis of Depression <i>Jacob Huffman and George T. Taylor</i>	37
An Overview of Neuropsychiatric Symptoms and Pathogenesis in MS <i>Emily Evans, Elizabeth Silbermann, and Soe Mar</i>	43
Psychoneuroimmunology: Immune Markers of Psychopathology <i>Rachel A. Wamser-Nanney and Brittany F. Goodman</i>	49

Genetic Polymorphisms <i>Mikhail Votinov and Katharina Sophia Goerlich</i>	59
Epigenetics in Developmental Disorders <i>Tiffany S. Doherty and Tania L. Roth</i>	75
Epigenetics in Behavior and Mental Health <i>Tiffany S. Doherty and Tania L. Roth</i>	83
Diffusion MRI: Introduction <i>Naiara Demnitz and Claire E. Sexton</i>	91
Structural Neuroimaging of Hippocampal Subfields in Healthy Aging, Alzheimer's Disease, Schizophrenia, and Major Depressive Disorder <i>Lok-Kin Yeung and Adam M. Brickman</i>	99
DTI Tractography Metrics: Fiber Bundle Length <i>Stephen Correia</i>	109
The Use of Diffusional Kurtosis Imaging (DKI) to Define Pathological Processes in Stroke and Dementia <i>Rachael L. Deardorff, Jens H. Jensen, and Joseph A. Helpert</i>	117
Functional Magnetic Resonance Imaging: fMRI <i>Tricia Z. King</i>	125
Resting-State Functional Magnetic Resonance Imaging and Neuropsychiatric Conditions <i>Carissa L. Philippi, Tasheia Floyd, and Gregory Dahl</i>	131
The Event-Related Potential (ERP) Method and Applications in Clinical Research <i>Amanda C. Eaton, Rebecca A. Meza, Kirk Ballew, and Suzanne E. Welcome</i>	141
Magnetic Resonance Spectroscopy (MRS) in Psychiatric Diseases <i>Alexander P. Lin, Sai Merugumala, Huijun Liao, and Napapon Sailasuta</i>	149
Artificial Intelligence in Medicine: Can Computers Aid the Diagnosis of Alzheimer's Disease? <i>Christian Salvatore, Petronilla Battista, and Isabella Castiglioni</i>	163
Therapeutic Effects of Repetitive Transcranial Magnetic Stimulation (rTMS) in Stroke: Moving Toward an Individualized Approach <i>Jason L. Neva, Kathryn S. Hayward, and Lara A. Boyd</i>	169
Connectomics and Multimodal Imaging Techniques in Clinical Research <i>Olusola Ajilore and Alex Leow</i>	181
Asperger Syndrome <i>Laurent Motttron</i>	187

Neurobiology of Down Syndrome <i>Stella Sakhon and Jamie Edgin</i>	197
Vascular Dementia <i>Paola García-Egan, Jenna Haddock, and Robert H. Paul</i>	205
Neurodegenerative Conditions: FTD <i>Abigail O. Kramer, Andrea G. Alioto, and Joel H. Kramer</i>	209
Traumatic Brain Injury (TBI) <i>Matthew R. Powell and Michael McCrea</i>	219
Chronic Traumatic Encephalopathy: A Long-Term Consequence of Repetitive Head Impacts <i>Michael L. Alosco, Alyssa Phelps, and Robert A. Stern</i>	227
Neuropsychological Assessment of Cortical and Subcortical Cognitive Phenotypes <i>Robert H. Paul, Carissa L. Philippi, and Gregory Dahl</i>	237
Mild Traumatic Brain Injury and Posttraumatic Stress Disorder: Diagnostic and Neurobiological Signatures of Comorbidity <i>Jacob Bolzenius</i>	243
Caloric Restriction, Cognitive Function, and Brain Health <i>Patrick J. Smith</i>	253
Psychosocial: Substance Abuse—Street Drugs <i>J. Megan Ross, Jacqueline C. Duperronzel, Ileana Pacheco-Colón, and Raul Gonzalez</i>	261
The Effects of Prenatal Stress on Offspring Development <i>Megan R. Gunnar and Colleen Doyle</i>	275
Psychophysiology of Traumatic Stress and Posttraumatic Stress Disorder <i>Michael G. Griffin, Brittany F. Goodman, Rebecca E. Chesher, and Natalia M. Kecala</i>	287
Cognitive Reserve <i>Sarah A. Cooley</i>	293
APOE as a Risk Factor for Age-Related Cognitive Impairment: Neuropsychological and Neuroimaging Findings <i>Laura E. Korthauer and Ira Driscoll</i>	299
The Serotonin Transporter-Linked Polymorphic Region (5-HTTLPR) Polymorphism, Stress, and Depression <i>Christina Di Iorio, Kimberly Johnson, Daniel Sheinbein, Samantha Kahn, Patrick England, and Ryan Bogdan</i>	311

The Neuroscience of Well-Being: Part 1 (Conceptual Definitions) <i>Justine Megan Gatt</i>	325
Gene × Environment Interactions: BDNF <i>Lauren E. Salminen and Robert H. Paul</i>	331
The Insula in Women with Posttraumatic Stress Disorder <i>Steven E. Bruce, Katherine R. Buchholz, and Wilson J. Brown</i>	341
Language and Literacy Development <i>Laura O'Hara, Rebecca A. Meza, and Amanda C. Eaton</i>	353
The Neuroscience of Well-Being: Part 2 (Potential Neural Networks) <i>Justine Megan Gatt</i>	361
Early Life Stress <i>Madeline B. Harms and Seth D. Pollak</i>	373
Alcohol-Related Brain Disorders <i>Rebecca N. Preston-Campbell, Stephanie A. Peak, Paola García-Egan, and Robert H. Paul</i>	395
Index	403

List of Contributors

- Amanda M. Acevedo**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Zeba Ahmad**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- Leona S. Aiken**, Department of Psychology, Arizona State University, Tempe, AZ, USA
- Olusola Ajilore**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Damla E. Aksen**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Kristi Alexander**, Department of Psychology, University of Central Florida, Orlando, FL, USA
- Andrea G. Alioto**, Palo Alto University, Palo Alto, CA, USA
Department of Neurology, University of California, Davis, CA, USA
- Michael L. Alosco**, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA
- Christina M. Amaro**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Judith Andersen**, Department of Psychology, University of Toronto, Mississauga, ON, Canada
- Aviva H. Ariel**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Danielle Arigo**, Psychology Department, The University of Scranton, Scranton, PA, USA
- Jamie Arndt**, Psychology Department, University of Missouri System, Columbia, MO, USA
- Robert C. Arnold**, Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
- Melissa V. Auerbach**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Lisa Auster-Gussman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Aya Avishai**, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Hoda Badr**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- David E. Balk**, Department of Health and Nutrition Sciences, Brooklyn College of the City University of New York, Brooklyn, NY, USA

- Kirk Ballew**, Department of Communication, Fontbonne University, St. Louis, MO, USA
 Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
 Department of Communication, University of Illinois at Urbana-Champaign College of
 Liberal Arts and Sciences, Urbana, IL, USA
- Diletta Barbiani**, Department of Neuroscience, University of Turin Medical School, Turin,
 Italy
 Hypoxia Medicine, Plateau Rosà, Switzerland
- Jeffrey E. Barnett**, College of Arts and Sciences, Loyola University Maryland, Baltimore,
 MD, USA
- Petronilla Battista**, Istituti Clinici Scientifici Maugeri IRCCS, Pavia, Institute of Bari, Italy
- Brittney Becker**, Psychology Department, Texas A&M University System, College Station,
 TX, USA
- Alexandra A. Belzer**, Colby College, Waterville, ME, USA
- Fabrizio Benedetti**, Department of Neuroscience, University of Turin Medical School,
 Turin, Italy
 Hypoxia Medicine, Plateau Rosà, Switzerland
- Danielle S. Berke**, Massachusetts Veterans Epidemiology Research and Information Center
 (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Mamta Bhatnagar**, Department of Medicine, University of Pittsburgh Medical Center,
 Pittsburgh, PA, USA
- Kelly Biegler**, Department of Psychological Science, University of California, Irvine, CA,
 USA
- Susan J. Blalock**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill,
 Chapel Hill, NC, USA
- Brittany E. Blanchard**, Department of Psychological Sciences, Texas Tech University,
 Lubbock, TX, USA
- Jennifer B. Blossom**, Clinical Child Psychology Program, University of Kansas, Lawrence,
 KS, USA
- Ryan Bogdan**, BRAIN Lab, Psychological and Brain Sciences, Washington University in
 St. Louis, St. Louis, MO, USA
- Jacob Bolzenius**, Missouri Institute of Mental Health, University of Missouri-St. Louis,
 St. Louis, MO, USA
- Bruce Bongar**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Dianna Boone**, Clinical Psychology Program, Department of Psychological Sciences, Texas
 Tech University, Lubbock, TX, USA
- Jerica X. Bornstein**, Department of Human Development and Family Sciences, University of
 Texas at Austin, Austin, TX, USA
- Joaquín Borrego Jr.**, Department of Psychological Sciences, Texas Tech University, Lubbock,
 TX, USA
- Kyle J. Bourassa**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Lara A. Boyd**, Department of Physical Therapy, University of British Columbia, Vancouver,
 BC, Canada
 Djavad Mowafaghian Centre for Brain Health, University of British Columbia, Vancouver,
 BC, Canada
- Patrick Boyd**, Department of Psychology, University of South Florida, Tampa, FL, USA
- Adam M. Brickman**, Columbia University Medical Center, New York, NY, USA
- Athena Brindle**, Department of Psychology, College of Science, The University of Texas at
 Arlington, Arlington, TX, USA

- Jennifer L. Brown**, Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati College of Medicine, Cincinnati, OH, USA
- Kathleen S. Brown**, Private Practice, Fort Myers, FL, USA
- Wilson J. Brown**, Pennsylvania State University, The Behrend College, Erie, PA, USA
- Steven E. Bruce**, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Angela D. Bryan**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Katherine R. Buchholz**, VA Boston Healthcare System, Boston, MA, USA
National Center for Post Traumatic Stress Disorder, White River Junction, VT, USA
- Claire Burgess**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Edith Burns**, Medical College of Wisconsin, Milwaukee, WI, USA
- Rachel J. Burns**, Douglas Mental Health University Institute, McGill University, Montreal, QC, Canada
- Johannes B.J. Bussmann**, Erasmus MC University Medical Center Rotterdam, CA Rotterdam, South Holland, The Netherlands
- Fawn C. Caplandies**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Angela L. Carey**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Brandon L. Carlisle**, Department of Psychiatry, University of California, San Diego, CA, USA
- Delesha M. Carpenter**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Allison J. Carroll**, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL, USA
- Charles S. Carver**, Department of Psychology, University of Miami, Coral Gables, FL, USA
Center for Advanced Study in the Behavioral Sciences, Stanford University, Stanford, CA, USA
- Isabella Castiglioni**, Dipartimento di Fisica “G. Occhialini”, Università degli Studi di Milano-Bicocca – Piazza della Scienza, Milan, Italy
- Gretchen B. Chapman**, Department of Psychology, Rutgers University–New Brunswick, New Brunswick, NJ, USA
- Alyssa C.D. Cheadle**, Psychology Department, Hope College, Holland, MI, USA
- Rebecca E. Chesher**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- William J. Chopik**, Psychology Department, Michigan State University, East Lansing, MI, USA
- Nicholas J.S. Christenfeld**, Department of Psychology, University of California San Diego, La Jolla, CA, USA
- L.E. Chun**, University of Colorado, Boulder, CO, USA
- Briana Cobos**, Department of Psychology, Texas State University, San Marcos, TX, USA
- Alex S. Cohen**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Jenna Herold Cohen**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Lee M. Cohen**, Department of Psychology, College of Liberal Arts, The University of Mississippi, Oxford, MS, USA
- Catherine Cook-Cottone**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA

- Sarah A. Cooley**, Department of Neurology, School of Medicine, Washington University in Saint Louis, St. Louis, MO, USA
- Stephen Correia**, Department of Psychiatry and Human Behavior, Alpert Medical School, Brown University, Providence, RI, USA
- Patrick Corrigan**, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
- Juliann Cortese**, School of Information, Florida State University, Tallahassee, FL, USA
- Riley Cropper**, Stanford University Counseling and Psychological Services, Stanford Hospital and Clinics, Stanford, CA, USA
- Robert T. Croyle**, National Cancer Institute, Bethesda, MD, USA
- Tegan Cruwys**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Sasha Cukier**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Gregory Dahl**, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Beth D. Darnall**, Department of Anesthesiology, Perioperative, and Pain Medicine, Stanford University School of Medicine, Stanford, CA, USA
- Savannah Davidson**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Elizabeth L. Davis**, Department of Psychology, University of California, Riverside, CA, USA
- Rachael L. Deardorff**, Center for Biomedical Imaging, Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Janet Deatrick**, Department of Family & Community Health, University of Pennsylvania School of Nursing, Philadelphia, PA, USA
- Lauren Decaportale-Ryan**, Departments of Psychiatry, Medicine, & Surgery, University of Rochester Medical Center, Rochester, NY, USA
- Jordan Degelia**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Anita Delongis**, Department of Psychology, University of British Columbia, Vancouver, BC, Canada
- Naiara Demnitz**, Department of Psychiatry, University of Oxford, Oxford, UK
- Eliot Dennard**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Kristine M. Diaz**, Walter Reed National Military Medical Center, Bethesda, MD, USA
- Sally S. Dickerson**, Department of Psychology, Pace University, New York, NY, USA
- M. Robin DiMatteo**, Department of Psychology, University of California Riverside, Riverside, CA, USA
- Sona Dimidjian**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Genevieve A. Dingle**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Laura J. Dixon**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Michelle R. Dixon**, Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Tiffany S. Doherty**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Michael D. Dooley**, Department of Psychology, University of California, Riverside, Riverside, CA, USA

- Tessa L. Dover**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Colleen Doyle**, Institute of Child Development, College of Education and Human Development, University of Minnesota, Minneapolis, MN, USA
- Ira Driscoll**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Claudia Drossel**, Department of Psychology, Eastern Michigan University College of Arts and Sciences, Ypsilanti, MI, USA
- Sean P.A. Drummond**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Christine Dunkel Schetter**, Department of Psychology, University of California, Los Angeles, CA, USA
- William L. Dunlop**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Jacqueline C. Duperrouzel**, Florida International University, Miami, FL, USA
- Allison Earl**, Department of Psychology, University of Michigan, Ann Arbor, MI, USA
- Amanda C. Eaton**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Ulrich W. Ebner-Priemer**, Karlsruhe Institute of Technology, Karlsruhe, Germany
- Robin S. Edelstein**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Jamie Edgin**, Department of Psychology, The University of Arizona, Tucson, AZ, USA
- Bradley A. Edwards**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Naomi I. Eisenberger**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Amber S. Emanuel**, Psychology Department, University of Florida, Gainesville, FL, USA
- Patrick England**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Ipek Ensari**, Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA
- Emily Evans**, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
- James Evans**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Anne M. Fairlie**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Angelica Falkenstein**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Zachary Fetterman**, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
- Christopher Fink**, Durham Veterans Affairs Medical Center, Durham, NC, USA
- North Carolina State University, Raleigh, NC, USA
- Alexandra N. Fisher**, Psychology Department, University of Victoria, Victoria, BC, Canada
- Tabitha Fleming**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Tasheia Floyd**, Washington University School of Medicine, St. Louis, MO, USA

- Lauren A. Fowler**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- Elinor Flynn**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Heather Walton Flynn**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Elizabeth S. Focella**, Department of Psychology, University of Wisconsin Oshkosh, Oshkosh, WI, USA
- Amy Frers**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA
- Howard S. Friedman**, Psychology Department, University of California Riverside, Riverside, CA, USA
- Paul T. Fuglestad**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Kentaro Fujita**, Department of Psychology, The Ohio State University, Columbus, OH, USA
- Kristel M. Gallagher**, Thiel College, Greenville, PA, USA
- Andrea M. Garcia**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Paola García-Egan**, Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA
- Robert García**, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA
- Casey K. Gardiner**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Robert J. Gatchel**, Endowment Professor of Clinical Health Psychology Research, Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA
- Justine Megan Gatt**, Neuroscience Research Australia, Sydney, NSW, Australia
School of Psychology, University of New South Wales, Sydney, NSW, Australia
- Nicole K. Gause**, Department of Psychology, University of Cincinnati, Cincinnati, OH, USA
- Ashwin Gautam**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Fiona Ge**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Andrew L. Geers**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Margaux C. Genoff**, Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, NY, USA
- James I. Gerhart**, Department of Psychosocial Oncology, Cancer Integrative Medicine Program, Rush University Medical Center, Chicago, IL, USA
- Meg Gerrard**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Frederick X. Gibbons**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Arielle S. Gillman**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Marci E.J. Gleason**, Department of Human Development and Family Sciences, University of Texas at Austin, Austin, TX, USA
- Teresa Gobble**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

- Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA
Katharina Sophia Goerlich, Department of Psychiatry, Psychotherapy and Psychosomatics,
RWTH Aachen University, Aachen, Germany
Jamie L. Goldenberg, Department of Psychology, University of South Florida, Tampa,
FL, USA
Cesar A. Gonzalez, Mayo Clinic Minnesota, Rochester, MN, USA
Raul Gonzalez, Florida International University, Miami, FL, USA
Brittany F. Goodman, Department of Psychological Sciences, Center for Trauma Recovery,
University of Missouri-St. Louis, St. Louis, MO, USA
Janna R. Gordon, San Diego State University and SDSU/UC San Diego Joint Doctoral
Program in Clinical Psychology, San Diego, CA, USA
Edward C. Green, Department of Anthropology, The George Washington University,
Washington, DC, USA
Joseph P. Green, Department of Psychology, Ohio State University at Lima, Lima, OH,
USA
Michael G. Griffin, Department of Psychological Sciences and Center for Trauma Recovery,
University of Missouri–St. Louis, St. Louis, MO, USA
Steven Grindel, Medical College of Wisconsin, Milwaukee, WI, USA
Kristina Gryboski, Project Hope, Millwood, VA, USA
Suzy Bird Gulliver, Baylor and Scott and White Hospitals and Clinics, Waco, TX, USA
School of Public Health, Texas A&M University, College Station, TX, USA
Megan R. Gunnar, Institute of Child Development, College of Education and Human
Development, University of Minnesota, Minneapolis, MN, USA
Jenna Haddock, Department of Psychological Sciences, University of Missouri-St. Louis,
St. Louis, MO, USA
Martin S. Hagger, Health Psychology and Behavioral Medicine Research Group, School of
Psychology and Speech Pathology, Faculty of Health Sciences, Curtin University, Perth,
WA, Australia
Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland
School of Applied Psychology and Menzies Health Institute Queensland, Behavioural Bases
for Health, Griffith University, Brisbane, QLD, Australia
Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central
Queensland University, Rockhampton, QLD, Australia
Jada G. Hamilton, Department of Psychiatry and Behavioral Sciences, Memorial Sloan
Kettering Cancer Center, New York, NY, USA
Paul K.J. Han, Center for Outcomes Research and Evaluation, Maine Medical Center
Research Institute, Portland, ME, USA
Grace E. Hanley, Psychology Department, University of California, Riverside, Riverside,
CA, USA
Madeline B. Harms, Department of Psychology, University of Wisconsin-Madison, Madison,
WI, USA
Adam Harris, Department of Psychology and University Health Sciences Center, University
of Colorado Denver, Denver, CO, USA
Lauren N. Harris, VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA
Peter R. Harris, School of Psychology, University of Sussex, Brighton & Hove, UK
Philine S. Harris, School of Psychology, University of Sussex, Brighton & Hove, UK
William Hart, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
M.J. Hartsock, University of Colorado, Boulder, CO, USA

- Kelly B. Haskard-Zolnierrek**, Department of Psychology, Texas State University, San Marcos, TX, USA
- S. Alexandar Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Catherine Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Karen Hasselmo**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Kathryn S. Hayward**, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada
- Stroke Division, Florey Institute of Neuroscience and Mental Health, University of Melbourne, Melbourne, VIC, Australia
- NHMRC Centre for Research Excellence in Stroke Rehabilitation and Recovery, Parkville, VIC, Australia
- Jodi M. Heaps-Woodruff**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Dietlinde Heilmayr**, Psychology Department, Moravian College, Bethlehem, PA, USA
- Vicki S. Helgeson**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Joseph A. Helpern**, Center for Biomedical Imaging, Department of Neuroscience, Department of Neurology, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Garrett Hisler**, Department of Psychology, Iowa State University, Ames, IA, USA
- Molly B. Hodgkins**, Department of Psychological and Brain Sciences, University of Maine, Orono, ME, USA
- Michael Hoerger**, Department of Psychology, Tulane University, New Orleans, LA, USA
- Jeanne S. Hoffman**, US Department of the Army, Washington, DC, USA
- Stephanie A. Hooker**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Vera Hoorens**, Center for Social and Cultural Psychology, Katholieke Universiteit Leuven, Leuven, Belgium
- Arpine Hovasapian**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jennifer L. Howell**, Psychology Department, Ohio University, Athens, OH, USA
- Robert M. Huff**, Department of Health Sciences, California State University, Northridge, Morgan Hill, CA, USA
- Jacob Huffman**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Department of Psychiatry, Washington University in St. Louis School of Medicine, St. Louis, MO, USA
- Lauren J. Human**, Department of Psychology, McGill University, Montreal, QC, Canada
- Jeffrey M. Hunger**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Tanya Hunt**, Department of Clinical Psychology, Palo Alto University, Palo Alto, California, USA
- Steven Hyde**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- Christina Di Iorio**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA

- Jamie M. Jacobs**, Department of Psychiatry, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA
- Lauren James**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Rebekah Jazdzewski**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Brooke N. Jenkins**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jens H. Jensen**, Center for Biomedical Imaging, Department of Neuroscience, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Donna C. Jessop**, School of Psychology, University of Sussex, Brighton & Hove, UK
- Jolanda Jetten**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Kimberly Johnson**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Doerte U. Junghaenel**, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA
- Vanessa Juth**, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA
- Samantha Kahn**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- David A. Kalkstein**, Department of Psychology, New York University, New York, NY, USA
- Alexander Karan**, Department of Psychology, University of California, Riverside, CA, USA
- Natalia M. Kecala**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri–St. Louis, St. Louis, MO, USA
- Robert G. Kent de Grey**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- Margaret L. Kern**, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia
- Nathan A. Kimbrel**, Department of Veterans Affairs' VISN 6 Mid-Atlantic MIRECC, Durham, NC, USA
Durham Veterans Affairs Medical Center, Durham, NC, USA
Psychiatry and Behavioral Sciences, Duke University School of Medicine, Durham, NC, USA
- Tricia Z. King**, The Neuroscience Institute, Georgia State University, Atlanta, GA, USA
- Nancy Kishino**, West Coast Spine Restoration, Riverside, CA, USA
- Ekaterina Klyachko**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Brian Konecky**, Boise VAMC Virtual Integrated Multisite Patient Aligned Care Team Hub, Boise, ID, USA
- Gerald P. Koocher**, Department of Psychology, DePaul University, Chicago, IL, USA
- Laura E. Korthauer**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Abigail O. Kramer**, Palo Alto University, Palo Alto, CA, USA
Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Joel H. Kramer**, Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Zlatan Krizan**, Department of Psychology, Iowa State University, Ames, IA, USA

- Monica F. Kurylo**, Departments of Medicine and Public Health, University of Kansas Medical Center, Kansas City, KS, USA
- Onawa P. LaBelle**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa H. Laitner**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Mark J. Landau**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Shane Landry**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Kevin T. Larkin**, Department of Psychology, West Virginia University, Morgantown, WV, USA
- Jason M. Lavender**, Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA
- Kristin Layous**, Department of Psychology, California State University East Bay, Hayward, CA, USA
- Thanh Le**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Thad R. Leffingwell**, Department of Psychology, Oklahoma State University, Stillwater, OK, USA
- Angela M. Legg**, Department of Psychology, Pace University, New York, NY, USA
- Jana Lembke**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Elizabeth Lemon**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Richie L. Lenne**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Alex Leow**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Department of Bioengineering, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Howard Leventhal**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Linda J. Levine**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Melissa A. Lewis**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Meng Li**, Department of Health and Behavior Sciences, University of Colorado Denver, Denver, CO, USA
- Huijun Liao**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Alexander P. Lin**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Wendy P. Linda**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Nikolette P. Lipsey**, Psychology Department, University of Florida, Gainesville, FL, USA
- Dana M. Litt**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

- Andrew K. Littlefield**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Brett Litz**, Department of Psychology, Boston University & VA Boston Healthcare System, Boston, MA, USA
- Marci Lobel**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Todd Lucas**, C. S. Mott Endowed Professor of Public Health Associate Professor of Epidemiology and Biostatistics Division of Public Health College of Human Medicine Michigan State University, Wayne State University, Detroit, MI, USA
- Mia Liza A. Lustria**, School of Information, Florida State University, Tallahassee, FL, USA
- Steven Jay Lynn**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Salvatore R. Maddi**, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
- Renee E. Magnan**, Washington State University Vancouver, Vancouver, WA, USA
- Brenda Major**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Vanessa L. Malcarne**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
UC San Diego Moores Cancer Center, San Diego, CA, USA
Department of Psychology, San Diego State University, San Diego, CA, USA
- Kelsey A. Maloney**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Andrew W. Manigault**, Psychology Department, Ohio University, Athens, OH, USA
- Traci Mann**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Soe Mar**, Neurology and Pediatrics, Division of Pediatric Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Shannon M. Martin**, Department of Psychology, Central Michigan University, Mount Pleasant, MI, USA
- Allison Marziliano**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Katilyn Mascatelli**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Tyler B. Mason**, Department of Psychology, University of Southern California, Los Angeles, CA, USA
Neuropsychiatric Research Institute, Fargo, ND, USA
- Kevin S. Masters**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Kevin D. McCaul**, North Dakota State University, Fargo, ND, USA
- Tara P. McCoy**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Michael McCrea**, Brain Injury Research Program, Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA
- Scout N. McCully**, Department of Psychological Sciences, Kent State University, Kent OH, USA
- Don McGeary**, Department of Psychiatry, Department of Family and Community Medicine, The University of Texas Health Science Center San Antonio, San Antonio, TX, USA

- Jessica McGovern**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Robert E. McGrath**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Michael P. Mead**, North Dakota State University, Fargo, ND, USA
- Alan Meca**, Department of Psychology, Old Dominion University, Norfolk, VA, USA
- Matthias R. Mehl**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Timothy P. Melchert**, Department of Psychology, Marquette University, Milwaukee, WI, USA
- Paola Mendoza-Rivera**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Maria G. Mens**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Sai Merugumala**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Rebecca A. Meza**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Dara Mickschl**, Medical College of Wisconsin, Milwaukee, WI, USA
- Tricia A. Miller**, Santa Barbara Cottage Hospital, Santa Barbara, CA, USA
- Department of Psychology, University of California Riverside, Riverside, CA, USA
- Kyle R. Mitchell**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Mona Moieni**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Phillip J. Moore**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- DeAnna L. Mori**, VA Boston Healthcare System, West Roxbury, MA, USA
- Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
- Sandra B. Morissette**, Department of Psychology, The University of Texas at San Antonio College of Sciences, San Antonio, TX, USA
- Amber Morrow**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Ariel J. Mosley**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Robert W. Motl**, Department of Physical Therapy, University of Alabama at Birmingham, Birmingham, AL, USA
- Laurent Mottron**, Department of Psychiatry, Faculty of Medicine, Universite de Montreal, Montreal, QC, Canada
- Anne Moyer**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Elaine M. Murphy**, Population Reference Bureau, Washington, DC, USA
- Carolyn B. Murray**, Psychology, University of California, Riverside, CA, USA
- Christopher S. Nave**, Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA
- Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Eve-Lynn Nelson**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Steven M. Nelson**, VISN 17 Center of Excellence for Research on Returning War Veterans, Waco, TX, USA
- Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA

Center for Vital Longevity, School of Behavioral and Brain Sciences, University of Texas at Dallas, Dallas, TX, USA

Elizabeth Neumann, Medical College of Wisconsin, Milwaukee, WI, USA

Jason L. Neva, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada

Jody S. Nicholson, Department of Psychology, University of North Florida, Jacksonville, FL, USA

Christina Nisson, Department of Psychology, University of Michigan, Ann Arbor, MI, USA

Mary-Frances O'Connor, Psychology Department, University of Arizona, Tucson, AZ, USA

Laura O'Hara, Department of Communication, Fontbonne University, St. Louis, MO, USA

Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA

Gbolahan O. Olanubi, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA

Nils Olsen, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA

Yimi Omofuma, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA

Elizabeth Ortiz-Gonzalez, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Ileana Pacheco-Colón, Florida International University, Miami, FL, USA

Aliza A. Panjwani, Graduate School and University Center, City University of New York, New York, NY, USA

Mike C. Parent, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA

Thomas J. Parkman, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Robert H. Paul, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

Missouri Institute of Mental Health, St. Louis, MO, USA

Stephanie A. Peak, Battelle Memorial Institute, Columbus, OH, USA

Michael G. Perri, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

Laura Perry, Department of Psychology, Tulane University, New Orleans, LA, USA

Alyssa Phelps, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Carissa L. Philippi, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

L. Alison Phillips, Iowa State University, Ames, IA, USA

Paula R. Pietromonaco, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA

Craig P. Pollizi, Department of Psychology, Binghamton University, Binghamton, NY, USA

Seth D. Pollak, Department of Psychology, University of Wisconsin–Madison, Madison, WI, USA

David B. Portnoy, US Food and Drug Administration, Silver Spring, MD, USA

Rachel Postupack, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

- Matthew R. Powell**, Division of Neurocognitive Disorders, Department of Psychiatry & Psychology, Mayo Clinic Minnesota, Rochester, MN, USA
- Candice Presseau**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Sarah D. Pressman**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Rebecca N. Preston-Campbell**, Missouri Institute of Mental Health, St. Louis, MO, USA
- Isabel F. Ramos**, Department of Psychology, University of California, Los Angeles, CA, USA
- Christopher T. Ray**, College of Health Sciences, Texas Woman's University, Denton, TX, USA
- Amanda L. Rebar**, Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia
- Allecia E. Reid**, Colby College, Waterville, ME, USA
- May Reinert**, Department of Psychology, Pace University, New York, NY, USA
- Kelly E. Rentscher**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Tracey A. Revenson**, Department of Psychology, Hunter College, New York, NY, USA
- C. Steven Richards**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Laura River**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Megan L. Robbins**, Department of Psychology, University of California, Riverside, CA, USA
- Michael C. Roberts**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- John D. Robinson**, Department of Surgery and Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine/Hospital, Washington, DC, USA
- Theodore F. Robles**, Psychology Department, University of California Los Angeles, CA, USA
- Catherine Rochefort**, Department of Psychology, Southern Methodist University, Dallas, TX, USA
- Daniel G. Rogers**, Department of Psychology, University of Mississippi, MS, USA
- John L. Romano**, Department of Educational Psychology, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Karen S. Rook**, Department of Psychological Science, University of California, Irvine, CA, USA
- Ann Rosenthal**, Medical College of Wisconsin, Milwaukee, WI, USA
- J. Megan Ross**, Florida International University, Miami, FL, USA
- Tania L. Roth**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Alexander J. Rothman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Ronald H. Rozensky**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Kristen L. Rudd**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Kirstie K. Russell**, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada
- Arseny A. Ryazanov**, Department of Psychology, University of California San Diego, La Jolla, CA, USA

- Napapon Sailasuta**, Centre for Addiction and Mental Health, Toronto, ON, Canada
Stella Sakhon, Department of Psychology, The University of Arizona, Tucson, AZ, USA
Lauren E. Salminen, Imaging Genetics Center, University of Southern California, Marina del Rey, CA, USA
Christian Salvatore, Scuola Universitaria Superiore IUSS Pavia, Piazza della Vittoria, Pavia, Italy
DeepTrace Technologies S.R.L., Via Conservatorio, Milan, Italy
Peter M. Sandman, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
Keith Sanford, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
David A. Sbarra, Department of Psychology, University of Arizona, Tucson, AZ, USA
Michael F. Scheier, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
Adam T. Schmidt, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Elana Schwartz, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
Esther N. Schwartz, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
Seth J. Schwartz, Department of Public Health Sciences, University of Miami Miller School of Medicine, Miami, FL, USA
Ralf Schwarzer, Health Psychology, Freie Universität Berlin, Berlin, Germany
Health Psychology, Australian Catholic University, Melbourne, VIC, Australia
Richard J. Seime, Mayo Clinic Minnesota, Rochester, MN, USA
Claire E. Sexton, Department of Psychiatry, University of Oxford, Oxford, UK
Louise Sharpe, School of Psychology, Faculty of Science, The University of Sydney, Sydney, NSW, Australia
Lindsay Sheehan, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
Paschal Sheeran, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
Daniel Sheinbein, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
James A. Shepperd, Psychology Department, University of Florida, Gainesville, FL, USA
Brittany N. Sherrill, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
Molin Shi, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Emily W. Shih, Department of Psychology, University of California, Riverside, CA, USA
Ben W. Shulman, Psychology Department, University of California, Los Angeles, CA, USA
Elizabeth Silberman, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
Department of Neurology, Oregon Health and Science University, Portland, OR, USA
Roxane Cohen Silver, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
Colleen A. Sloan, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA

Rachel Smallman, Psychology Department, Texas A&M University System, College Station, TX, USA

Kathryn E. Smith, Neuropsychiatric Research Institute, Fargo, ND, USA
Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA

Patrick J. Smith, Duke University Medical Center, Durham, NC, USA

Todd A. Smitherman, Department of Psychology, University of Mississippi, University, MS, USA

Joshua M. Smyth, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA

Dara H. Sorkin, Department of Psychological Science, University of California, Irvine, CA, USA

R. L. Spencer, University of Colorado, Boulder, CO, USA

Melissa Spina, Psychology Department, University of Missouri System, Columbia, MO, USA

Bonnie Spring, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA

Annette L. Stanton, Department of Psychology, University of California, Los Angeles, Los Angeles, CA, USA

Ellen Stephenson, Department of Psychology, University of British Columbia, Vancouver, BC, Canada

Robert A. Stern, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Department of Neurosurgery and Anatomy & Neurobiology, Boston University School of Medicine, Boston, MA, USA

Angela K. Stevens, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Courtney J. Stevens, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA

Elizabeth M. Stewart, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia

Danu Anthony Stinson, Psychology Department, University of Victoria, Victoria, BC, Canada

Michelle L. Stock, Department of Psychological and Brain Sciences, George Washington University, Washington, DC, USA

Arthur A. Stone, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA

Department of Psychology, Dana and David Dornsife College of Letters Arts and Sciences, University of Southern California, Los Angeles, CA, USA

Jeff Stone, Psychology Department, University of Arizona, Tucson, AZ, USA

John A. Sturgeon, Center for Pain Relief, University of Washington, Seattle, WA, USA

Kieran T. Sullivan, Department of Psychology, Santa Clara University, Santa Clara, CA, USA

Amy Summerville, Psychology Department, Miami University, Oxford, OH, USA

Jessie Sun, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia

Department of Psychology, University of California, Davis, CA, USA

Steve Sussman, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

- Trevor James Swanson**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Allison M. Sweeney**, Department of Psychology, University of South Carolina, Columbia, SC, USA
- Kate Sweeny**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Sergey Tarima**, Medical College of Wisconsin, Milwaukee, WI, USA
- Carly A. Taylor**, Boston Medical Center, Boston, MA, USA
- George T. Taylor**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Interfakultäre Biomedizinische Forschungseinrichtung (IBF) der Universität Heidelberg, Heidelberg, Germany
- A. Janet Tomiyama**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Victoria A. Torres**, Department of Psychology, College of Liberal Arts, University of Mississippi, Oxford, MS, USA
- David R.M. Trotter**, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
- Bert N. Uchino**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- John A. Updegraff**, Department of Psychological Sciences, Kent State University, Kent, OH, USA
- Jay Urbain**, Milwaukee School of Engineering, Milwaukee, WI, USA
- Amy E. Ustjanauskas**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- UC San Diego Moores Cancer Center, San Diego, CA, USA
- Jason Van Allen**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Erika D. Van Dyke**, Department of Sport Sciences, College of Physical Activity and Sport Sciences, West Virginia University, Morgantown, WV, USA
- Department of Psychology, Springfield College, Springfield, MA, USA
- Judy L. Van Raalte**, Department of Psychology, Springfield College, Springfield, MA, USA
- Maryke Van Zyl-Harrison**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Rachel Vanderkruik**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Elizabeth J. Vella**, Department of Psychology, University of Southern Maine, Portland, ME, USA
- Birte von Haaren-Mack**, German Sport University Cologne, Köln, Nordrhein-Westfalen, Germany
- David Von Nordheim**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Mikhail Votinov**, Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany
- Institute of Neuroscience and Medicine (INM-10), Jülich, Germany
- Rebecca K. Vujnovic**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA
- Amy Wachholtz**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA

- Eric F. Wagner**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Ryan J. Walker**, Psychology Department, Miami University, Oxford, Ohio, USA
- Rachel A. Wamser-Nanney**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Britney M. Wardecker**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa Ward-Peterson**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Lisa Marie Warner**, Health Psychology, Freie Universitat Berlin, Berlin, Germany
- Neil D. Weinstein**, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
- Suzanne E. Welcome**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Kristen J. Wells**, San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- Amy Wenzel**, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, PA, USA
- Private Practice, Rosemont, PA, USA
- Summer L. Williams**, Department of Psychology, Westfield State University, Westfield, MA, USA
- Cynthia J. Willmon**, Reno VA Sierra Nevada Health Care System, Reno, NV, USA
- Sara M. Witcraft**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Stephen A. Wonderlich**, Neuropsychiatric Research Institute, Fargo, ND, USA
- Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA
- E.R. Woodruff**, University of Colorado, Boulder, CO, USA
- Patrick W. Wright**, Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Robert C. Wright**, Department of Psychology, University of California, Riverside, CA, USA
- Shawna Wright**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Tuppett M. Yates**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Julie D. Yeterian**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Lok-Kin Yeung**, Columbia University Medical Center, New York, NY, USA
- Alex J. Zautra**, Psychology Department, Arizona State University, Tempe, AZ, USA
- Colin A. Zestcott**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Peggy M. Zoccola**, Psychology Department, Ohio University, Athens, OH, USA

Foreword

Until the 1970s, there were no books, journals, or university courses on health psychology. Although the field's intellectual roots stretch back to the beginnings of psychology more than a century ago, its formal emergence depended on a convergence of influences (Friedman & Silver, 2007), including psychosomatic medicine, social-psychological and socio-anthropological perspectives on medicine, epidemiology, and medical and clinical psychology. Today, health psychology is a principal area of significant social science research and practice, with vital implications for the health and well-being of individuals and societies. Understanding the explosive trajectory of health psychology is useful to appreciating the strengths of the field and to approaching this new encyclopedia, the *Wiley Encyclopedia of Health Psychology*.

What is the nature of health? That is, what does it mean to be healthy? The way that this question is answered affects the behaviors, treatments, and resource allocations of individuals, families, health practitioners, governments, and societies. For example, if it is thought that you are healthy unless and until you contract a disease or suffer an injury, then attention and resources are primarily allocated toward “fixing” the problem through medications or surgical repairs. This is the traditional biomedical model of disease (sometimes called the “disease model”). Indeed, in the United States, the overwhelming allocation of attention and resources is to physicians (doing treatments) and to pharmaceuticals (prescription drugs and their development). In contrast, health psychology developed around a much broader and more interdisciplinary approach to health, one that is often termed the biopsychosocial model.

The biopsychosocial model (a term first formally proposed by George Engel in 1968) brings together core elements of staying healthy and recovering well from injury or disease (Stone et al., 1987). Each individual—due to a combination of biological influences and psychosocial experiences—is more or less likely to thrive. Some of this variation is due to genetics and early life development; some depends on the availability and appropriate applications of medical treatments; some involves nutrition and physical activity; some depends on preparations for, perceptions of, and reactions to life's challenges; and some involves exposure to or seeking out of healthier or unhealthier environments, both physically and socially. When presented in this way, it might seem obvious that health should certainly be viewed in this broader interdisciplinary way. However, by misdirecting its vast expenditures on health care, the United States gives its residents mediocre health at high cost (Kaplan, 2019). To approach these matters in a thorough manner, this encyclopedia includes four volumes, with Volume 1 focusing on the biological bases of health and health behavior, Volume 2 concentrating on the social bases, Volume 3 centering around the psychological and clinical aspects, and Volume 4 focused more broadly on crosscutting and applied matters.

Key parts of health depend on biological characteristics and how they interact with our experiences and environments. So, for example, in Volume 1, there are articles on injury to the brain, alcohol effects on the brain, nutrition, drug abuse, psychophysiology, and the tools and key findings of neuroscience. Note that even individuals with the same genes (identical twins) can and do have different health and recovery outcomes, and the articles delve into such complexities of health. Of course humans are also social creatures, and people's growth, development, and health behaviors take place in social contexts. So, in Volume 2, there are articles on such fundamental matters as social support, coping, spirituality, emotion, discrimination, communication, psychosocial stress, and bereavement.

Because our approaches to and conceptions of health are heavily influenced by society's institutions and structures revolving around medical care, much of health psychology derives from or intersects with clinical psychology and applied behavioral medicine. Volume 3 covers such clinical topics as psycho-oncology, depression, drug abuse, chronic disease, eating disorders, and the psychosocial aspects of coronary heart disease. Finally and importantly, there are a number of special and cross-cutting matters that are considered in Volume 4, ranging from relevant laws and regulations to telehealth and health disparities. Taken together, the articles triangulate on what it truly means to be healthy.

What are the most promising directions for the future that emerge from a broad and deep approach to health? That is, to where do these encyclopedia articles point? One key clue arises from a unique opportunity to enhance, extend, and analyze the classic Terman study of children who were followed and studied throughout their lives (Friedman & Martin, 2012). These studies revealed that there are lifelong trajectories to health, thriving, and longevity. Although anyone can encounter bad luck, a number of basic patterns emerged that are more likely to lead to good health. That is, for some individuals, certain earlier life characteristics and circumstances help propel them on *pathways* of healthier and healthier behaviors, reactions, relationships, and experiences, while others instead face a series of contingent stumbling blocks. There are multipart but nonrandom pathways across time linking personalities, health behaviors, social groups, education, work environments, and health and longevity. The present encyclopedia necessarily is a compendium of summaries of the relevant elements of health and thriving, but one that would and can profitably be used as a base to synthesize the long-term interdependent aspects of health.

In sum, this encyclopedia is distinctive in its explicit embrace of the biopsychosocial approach to health, not through lip service or hand-waving but rather through highly detailed and extensive consideration of the many dozens of topics crucial to this core interdisciplinary understanding.

References

- Friedman, H. S., & Martin, L. R. (2012). *The longevity project: Surprising discoveries for health and long life from the landmark eight-decade study*. New York: Penguin Plume Press.
- Friedman, H. S., & Silver, R. C. (Eds.) (2007). *Foundations of health psychology*. New York: Oxford University Press.
- Kaplan, R. M. (2019). *More than medicine: The broken promise of American health*. Cambridge, MA: Harvard University Press.
- Stone, G., Weiss, M., Matarazzo, J. D., Miller, N. E., Rodin, J., Belar, C. D., ... Singer, J. E. (Eds.) (1987). *Health psychology: A discipline and a profession*. University of Chicago Press.

Howard S. Friedman
University of California, Riverside
August 15, 2019

Preface Volume 1: Biological Bases of Health

The field of health psychology is a specialty area that draws on how biology, psychology, behavior, and social factors influence health and illness. Despite the fact that the formal recognition of the field is only about a half of a century old, it has established itself as a major scientific and clinical discipline. The primary reason for this is that there have been a number of significant advances in psychological, medical, and physiological research that have changed the way we think about health, wellness, and illness. However, this is only the tip of the iceberg.

We have the field of health psychology to thank for much of the progress seen across our current healthcare system. Let me provide some examples that illustrate the diversity of these important contributions. Starting with a broad public health perspective, health psychologists have been involved in how our communities are planned and how urban development has a significant and direct impact on our health behaviors. That is, if we live close to where we work, play, and shop, we are more likely to walk or bike to our destination rather than drive or take public transportation. Focusing on a smaller, but no less important, source of data, health psychologists are involved in using information obtained from our genes to help counsel individuals to make good, well-informed health decisions that could have an impact on the individual immediately or in the future. Being well-informed can direct a person toward the best possible path toward wellness, whether it be measures aimed at prevention, further monitoring, or intervention.

Of course, there are many other contributions. For instance, data indicates that several of the leading causes of death in our society can be prevented or delayed (e.g., heart disease, respiratory disease, cerebrovascular disease, and diabetes) via active participation in psychological interventions. Knowing that we can improve health status by changing our behaviors seems like an easy “fix,” but we know that behavior change is tough. As such, it is not surprising that health psychologists have been involved in trying to improve upon treatment success by examining patient compliance. When we better understand what motivates and discourages people from engaging in treatment or pro-health behaviors, we can improve upon compliance and help individuals adopt more healthy lifestyles. Health psychologists have also played a role in shaping healthcare policy via identifying evidence-based treatments. This work has direct effects on how individuals receive healthcare as well as what treatments are available/reimbursable by insurance companies. Finally, there are also factors beyond medical care to consider (e.g., economic, educational) that can lead to differential health outcomes. Thus, health psychologists likewise examine ways to reduce health disparities, ensuring that the public and government officials are made aware of the impact of the social determinants of health.

I could go on and on, however, examples like this illustrate the significant impact this broad and exciting field has had (and will continue to have) on our understanding of health and wellness. Given the constantly changing nature of the field, it is not possible to be all inclusive; however, the aim of these four volumes is to provide readers an up-to-date overview of the field. Each entry is written to stand alone for those who wish to learn about a specific topic, and if the reader is left wanting more, suggested readings are provided to expand one's knowledge. Volume I, *Biological Bases of Health Behavior*, includes entries that cover topics in the broad areas of neuroscience and biopsychology relevant to health behavior. General topics include degenerative and developmental conditions, emerging methodologies available in clinical research, functional anatomy and imaging, and gene $\times$ environment interactions. Volume II, *Social Bases of Health Behavior*, addresses topics related to theories and concepts derived from social psychology. Specifically, topics related to the self, social cognition, social perception, attitudes and attitude change, perception, framing, and pro-health behaviors are included. Volume III, *Clinical Health Psychology and Behavioral Medicine*, covers the applied aspects of the field of health psychology including practical topics that clinical health psychologists face in the workplace, behavioral aspects of medical conditions, the impact of unhealthy behaviors, and issues related to the comorbidity of psychiatric disorders and chronic health concerns. Finally, Volume IV, *Special Issues in Health Psychology*, contains a wide array of topics that are worthy of special consideration in the field. Philosophical and conceptual issues are discussed, along with new approaches in delivering treatment and matters to consider when working with diverse and protected populations.

It is my sincere hope that the *Wiley Encyclopedia of Health Psychology* will serve as a comprehensive resource for academic and applied psychologists, other health care professionals interested in the relationship of psychological and physical well-being, and students across the health professions. I would very much like to thank and acknowledge all those who have made this work possible including Michelle McFadden, the Wiley editorial and production teams, the volume editors, and of course all of the authors who contributed their outstanding work.

Lee M. Cohen, PhD

Preface Volume 1: Biological Basis of Behavior

The chapters of this volume focus on the neural and molecular mechanisms of the biopsychosocial model of behavioral health. The work detailed herein is timely, as the scientific pendulum is shifting away from the long-standing research aimed at dismantling complex clinical phenotypes into biological components as the pathway to discover biological mechanisms. The common reductionistic approach has yielded no curative strategies for children and adults affected by the most vexing neuropsychiatric and neurodegenerative conditions. At the same time, a worsening incidence of behavioral health disorders and associated reductions in health-adjusted life expectancies worldwide raises concern that behavioral health conditions represent a looming global health crisis.

This volume summarizes the current state of science on the biological bases of behavioral health, with a focus on advanced conceptual models that prioritize the integration of numerous biological, psychological, and sociocultural interdependencies inherent to developmental conditions (e.g., autism spectrum disorder, Down syndrome, language-based learning disorder), contextual factors that alter neurodevelopmental trajectories (e.g., obesity, illicit drug use, psychosocial stress, traumatic brain injury, infectious disease), and age-related neurodegenerative conditions (e.g., cerebrovascular disease, Alzheimer's disease). Technological advances are reviewed, including neuroimaging methods capable of visualizing the anatomical and physiological basis of behavioral health disorders that have high potential to improve diagnostic classification and monitoring of patient care outcomes (i.e., precision medicine).

Technologies and computational innovations introduced during the past three decades following the "Decade of the Brain" in 1990 and completion of the Human Genome Project in 1993 offer a viable path to create much needed scientific breakthroughs that are difficult to achieve using traditional research methods designed more to confirm hypotheses than to foster new insights and discovery. Data science offers a new opportunity to identify nonlinear and linear patterns and highly dimensional interactions across explanatory variables that more accurately model the intrinsic features of the biopsychosocial determinants of behavior. Combined with new advances in diagnostic technologies (e.g., ligands for positron emission tomography) and intervention strategies (e.g., transcranial magnetic stimulation), the current scientific momentum has potential to transform behavioral health outcomes on a global scale.

Robert Paul
St. Louis, MO, USA

Editor-in-Chief Acknowledgments

I would like to publicly recognize and thank my family, Michelle, Ross, Rachel, and Becca for being supportive of my work life while also providing me with a family life beyond my wildest dreams. I love you all very much. I would also like to note my great appreciation and thanks to the faculty and staff of the Doctoral Training Program in Clinical Psychology at Oklahoma State University for providing me with an opportunity to expand my education and for providing extraordinary training. In particular, I would like to thank my late mentor Dr. Frank L. Collins Jr., Dr. Larry Mullins, Dr. John Chaney, and Patricia Diaz Alexander. Further, I am grateful to the University of Mississippi and Texas Tech University (and the colleagues and students I have had the privilege to work alongside) for affording me excellent working environments and the support to do the work I am honored to be a part of. Finally, I would like to recognize and thank the volume editors for their vision and perseverance to this project as well as to each of the contributors for their excellent entries and their dedication to this very important field.

Lee M. Cohen, Ph.D.
Editor-in-Chief

Brain Development from Conception to Adulthood

Jodi M. Heaps-Woodruff and David Von Nordheim

*Missouri Department of Mental Health, University of Missouri–St. Louis,
St. Louis, MO, USA*

Missouri Institute of Mental Health, St. Louis, MO, USA

Brain Gray Matter and White Matter Development

The development of the central nervous system (CNS) begins approximately 17 days after conception. Arranged in a flat disk with three layers, the cells that comprise the topmost layer, the ectoderm, forms the neural plate and provides the foundation of the developing nervous system. Within 22–25 days after conception, the cells of this plate fold into a groove that forms the neural tube, the precursor to the brain and spinal cord. Subsequent development leverages a pool of undifferentiated cells that lack functional specificity until the cells migrate to predetermined regions. This process is guided by progenitor cells that release attraction molecules to guide the formation of new synapses. Beyond the first year of life, brain cells undergo ongoing refinement of existing connections (Institute of Medicine and National Academy of Sciences, 1992) and programmed cell death to eliminate connections that are not regularly activated.

In a comprehensive review, Sowell et al. (2003) describes the discordant pattern of gray and white matter development across the lifespan. Gray matter volumes increase exponentially during infancy until about 18 months after birth, when the rate continues to increase, albeit more slowly, until the late 20s. Peak gray matter volume is generally achieved in the third decade followed by progressive reductions in volume thereafter. By contrast, the volume of white matter increases into the fourth decade followed by progressive decline. Given the difference in developmental maturation of the gray and white matter, total brain volume is a poor index of brain integrity, especially during the third and fourth decade.

The brain accounts for 2–3% of total body mass while consuming 20% of total glucose needs. These demands are even greater from birth through late adolescence. The metabolic rate of glucose in the brain (CGMR) doubles from birth to 6 months, with a fourfold increase

from birth through the second year of life. Glucose needs remain high until adolescence, with peak levels accounting for almost half of the total energy needs of the body (Goyal & Raichle, 2018). These data underscore the high resource demands of the developing brain in children and adolescents (Kuzawa et al., 2014).

Critical Time Windows of Brain Development

Development does not occur in a vacuum. Genetic, epigenetic, and environmental factors (Bale, 2015; Fox, Levitt, & Nelson, 2010) are potentially powerful moderators of neurodevelopment. During prenatal development, environmental factors can produce permanent damage to brain structure and connections. Preliminary research suggests that targeted interventions applied during the first years of life have potential to mitigate these deleterious abnormalities, but the durability of treatment outcomes remains unclear.

Many studies indicate that the prenatal environment strongly influences development, particularly maternal stress and immunity. A recent review by Lindsay, Buss, Wadhwa, and Entringer (2018) identifies associations between maternal anxiety, depression, elevated stress hormones, and alterations in both brain structure and function. Maternal diet may have a protective effect on prenatal development. Preliminary data indicate a link between certain high fat diets and reduced maternal stress. Additionally, an antioxidant-rich diet may also mitigate the effects of maternal stress on fetal brain development. Similarly, diets high in antioxidants, nutrients, and certain fatty acids (polyunsaturated), along with exercise and engagement in mental activities, can stimulate adult neurogenesis (Poulose, Miller, Scott, & Shukitt-Hale, 2017).

A sensitive developmental window also occurs around ages 5–7, a time when many children residing in resourced countries are exposed to new pathogens and social stressors at school. During puberty, a great deal of physical and chemical change is taking place in the body. Some brain regions are nearing maturity, whereas others are continuing to undergo dynamic change. Early adolescence is considered the end of a critical period for language acquisition. However, the frontal and prefrontal cortices that mediate reasoning and judgment are still developing. These structures are particularly sensitive to alcohol and drugs both in terms of damage and increased propensity for substance use disorders in adulthood. Early adulthood represents yet another sensitive period, as realized by the onset of chronic mental illness during the second to third decade of life (e.g., schizophrenia).

Moderating Factors of Brain Development

The impact of the gut microbial environment on brain health has been a fairly recent area of study. Research with germ-free mice suggests altered neurogenesis in a number of brain structures relative to a control group. In both the hippocampus and the amygdala, the resulting alterations consist of increased volumes as the result of increased neurogenesis; however, in the hippocampus, fewer dendritic spines were observed, whereas there were more spines evident in the amygdala of germ-free mice. Additionally, hypermyelination in the prefrontal cortex has also been reported in germ-free mice. In addition to effects on brain structure, some bacteria have the ability to synthesize neurotransmitters (NTs). As examples, *Lactobacillus* and *Bifidobacterium* are able to synthesize gamma-aminobutyric acid (GABA), and norepinephrine has been produced by species of *Escherichia*, *Bacillus*, and *Saccharomyces*. Other species,

such as *Bifidobacterium infantis*, may indirectly influence serotonin levels by increasing the levels of precursor molecule, tryptophan. A strong empirical base is emerging that implicates the gut–brain axis in numerous brain disorders including anxiety, autism, schizophrenia, Alzheimer’s disease, and Parkinson’s disease (for review see Dinan & Cryan, 2017).

Another recent and active area of research examines the role of microglia in brain development and pathology. Microglia function as the immune cells of the nervous system, activating in the presence of injury or inflammation. Microglia facilitate the clearing of toxins and dying cells and support neurodevelopmental processes including modulation of neuronal apoptosis, cell proliferation, and synaptic activity. In studies of schizophrenia and autism, elevated levels of activated microglia have been observed, along with evidence of perturbations in brain development. As with numerous other factors, it is unclear whether this abnormal microglia activity is responsible for altered brain development or whether the microglia are responding to other insults that have negatively impacted neurodevelopment (Bilimoria & Stevens, 2015).

In this chapter we have reviewed the growth and development of the human brain, critical or sensitive periods in brain development, discordant gray and white matter maturation, and factors that influence neurodevelopment. The research literature in these areas is continuing to evolve to produce more complete explanatory models of brain development. Considerable research is needed on therapeutic targets to mitigate the negative effects of moderating factors on development to improve brain health across the lifespan.

Author Biographies

Jodi M. Heaps-Woodruff is a research neuropsychologist and health services researcher. She earned her PhD from the University of Missouri–St. Louis and is a research assistant professor at the Missouri Institute of Mental Health at the University of Missouri–St. Louis. Her research is focused on the cognitive aspects involved in recovery from mental illness and the impact of chronic disease on the brain.

David Von Nordheim earned his Masters of Arts in Cognitive Psychology from the University of Nebraska–Omaha in May 2018. His research interests include memory, attention, word processing, and the cognitive psychology of addictive behaviors.

References

- Bale, T. L. (2015). Epigenetic and transgenerational reprogramming of brain development. *Nature Reviews Neuroscience*, 16(6), 332.
- Bilimoria, P. M., & Stevens, B. (2015). Microglia function during brain development: New insights from animal models. *Brain Research*, 1617, 7–17.
- Dinan, T. G., & Cryan, J. F. (2017). Gut instincts: Microbiota as a key regulator of brain development, ageing and neurodegeneration. *The Journal of Physiology*, 595(2), 489–503.
- Fox, S. E., Levitt, P., & Nelson, C. A., III (2010). How the timing and quality of early experiences influence the development of brain architecture. *Child Development*, 81(1), 28–40.
- Goyal, M. S., & Raichle, M. E. (2018). Glucose requirements of the developing human brain. *Journal of Pediatric Gastroenterology and Nutrition*, 66, S46–S49.
- Institute of Medicine and National Academy of Sciences. (1992). *Discovering the Brain*. Washington, DC: The National Academies Press. <https://doi.org/10.17226/1785>
- Kuzawa, C. W., Chugani, H. T., Grossman, L. I., Lipovich, L., Muzik, O., Hof, P. R., ... Lange, N. (2014). Metabolic costs and evolutionary implications of human brain development. *Proceedings of the National Academy of Sciences*, 111(36), 13010–13015.

- Lindsay, K. L., Buss, C., Wadhwa, P. D., & Entringer, S. (2018). The interplay between nutrition and stress in pregnancy: Implications for fetal programming of brain development. *Biological Psychiatry*, 85(2), 135–149.
- Poulose, S. M., Miller, M. G., Scott, T., & Shukitt-Hale, B. (2017). Nutritional factors affecting adult neurogenesis and cognitive function. *Advances in Nutrition (Bethesda, Md.)*, 8(6), 804–811. doi:10.3945/an.117.016261
- Sowell, E. R., Peterson, B. S., Thompson, P. M., Welcome, S. E., Henkenius, A. L., & Toga, A. W. (2003). Mapping cortical change across the human life span. *Nature Neuroscience*, 6(3), 309.

Suggested Reading

- Hair, N. L., Hanson, J. L., Wolfe, B. L., & Pollak, S. D. (2015). Association of child poverty, brain development, and academic achievement. *JAMA Pediatrics*, 169(9), 822–829.
- Hanson, J. L., Hair, N., Shen, D. G., Shi, F., Gilmore, J. H., Wolfe, B. L., & Pollak, S. D. (2013). Family poverty affects the rate of human infant brain growth. *PLoS One*, 8(12), e80954.
- Institute of Medicine and National Academy of Sciences. (1992). *Discovering the Brain*. Washington, DC: The National Academies Press. <https://doi.org/10.17226/1785>
- Knickmeyer, R. C., Gouttard, S., Kang, C., Evans, D., Wilber, K., Smith, J. K., ... Gilmore, J. H. (2008). A structural MRI study of human brain development from birth to 2 years. *Journal of Neuroscience*, 28(47), 12176–12182.
- Prado, E. L., & Dewey, K. G. (2014). Nutrition and brain development in early life. *Nutrition Reviews*, 72(4), 267–284.
- Stouffer, M. A., Golden, J. A., & Francis, F. (2016). Neuronal migration disorders: Focus on the cytoskeleton and epilepsy. *Neurobiology of Disease*, 92, 18–45.

Functional Anatomy: Types of Cells/Physiology

Patrick W. Wright

Neurology, Washington University in St. Louis, St. Louis, MO, USA

Neurons

Neurons are the primary fundamental unit of the central nervous system (CNS) and provide the infrastructure for all major communication within the brain. Grossly, typical neuron morphology consists of a cell body (soma), dendrites, and axons. The soma contains the nucleus and other organelles. Arising from the cell body is the arborization of dendrites, which act as the primary synaptic target from other neurons. The axon is a long projection from the soma that communicates integrated information from the dendrites and acts on a downstream synapse. In humans, neurons can be classified by the number of processes that extend from the soma: bipolar, multipolar, and pseudounipolar neurons. Bipolar neurons have one axon and one dendrite that extend from the soma and can be found in the visual pathway (e.g., retinal bipolar cells). Multipolar neurons (e.g., Purkinje cells in the cerebellum) have one axon and multiple dendrites; they are the most common type found in the CNS. Pseudounipolar neurons have one soma process that bifurcates into two branches. Most sensory neurons are pseudounipolar. Neurons can be further classified by their context-dependent structure determined by the neural circuit in which they participate. Interneurons, or local circuit neurons, have relatively short axons, whereas projection neurons have axons that extend to distant targets (Nicholls, Martin, Wallace, & Kuffler, 2001; Purves, 2008).

The Synapse

The interface between neurons where information is transferred is known as the synapse, which consists of the axon terminal of the presynaptic cell, the intermediate extracellular space (the synaptic cleft), and the postsynaptic cell. The axon of presynaptic cell is not limited to forming

synapses with only the dendritic projection of the postsynaptic cell. Instead, axosecretory (axon–bloodstream), axoaxonic (axon–axon), axodendritic (axon–dendrite), and axosomatic (axon–soma) are examples of alternative synaptic interfaces. Formed synapses are primarily classified by the mode of information transfer: chemical synapses and electrical synapses (Nicholls et al., 2001; Purves, 2008).

Synaptic Transmission

Electrical synapse

Electrical transmission at electrical synapses is mediated by gap junctions, which are channels that connect the intracellular space and cytoplasm of adjacent cells. Electrical synapses are inherently bidirectional, enabling the passage of electrical currents (carried by ions) as well as intracellular messengers to and from the postsynaptic cell. Each gap junction channel (connexon) is made up of a pair of hexameric connexin proteins (e.g., connexin 36 [CX36], CX50), with each cell providing one of the pair. These gap junctions allow passive ionic current flow, providing an almost instantaneous route of transmission (Nicholls et al., 2001; Pereda, 2014; Purves, 2008).

Chemical synapse

Chemical synapses are the most common type of synapse. Information transfer at chemical synapses is facilitated by presynaptic synaptic vesicles containing neurotransmitters. Action potentials, most often originating at the “neck” of the axon (known as the axon hillock) where it connects to the neuronal soma, propagate down the length of the axon to the axon terminals. Myelin sheaths provided by neighboring oligodendrocytes are insulating adipose tissue that improve conductance rate by constraining electrochemical gradient-associated depolarization to occur at the gaps between sheaths known as nodes of Ranvier; “white matter” is so named due to the white appearance of these myelin sheaths on axons. When the action potential arrives at the axon terminal, it activates voltage-gated calcium (Ca^{2+}) channels that are present along the presynaptic membrane. The subsequent influx of Ca^{2+} along its concentration gradient ($[\text{Ca}^{2+}]_e \approx 1\text{--}2\text{ mM}$, $[\text{Ca}^{2+}]_i \approx 0.0001$) enables vesicle fusion to the presynaptic membrane. These vesicles contain neurotransmitters that can then be exocytosed into the synaptic cleft. Released neurotransmitters will diffuse across the synaptic cleft via Brownian motion to the postsynaptic membrane containing metabotropic and ionotropic receptors; information transfer via this mechanism is inherently probabilistic, in contrast to electrical transmission via gap junctions as occurs at electrical synapses (Nicholls et al., 2001; Pereda, 2014; Purves, 2008).

Synaptic vesicle exocytosis and recycling

SNARE (soluble *N*-ethylmaleimide-sensitive factor [NSF] attachment protein receptor) protein complexes at the vesicle (v-SNARE) and membrane (target-localized, t-SNARE) enable docking of the vesicle to the inner membrane surface following binding of Ca^{2+} to the Ca^{2+} -sensing vesicular protein synaptotagmin-1, enabling neuroexocytosis. The three-vesicle pool model describes a reserve pool consisting of 80–90% of the total pool of vesicles, a recycling pool with 10–15%, and the readily releasable pool (RRP) at the active zone, the portion of the presynaptic membrane where vesicle exocytosis occurs that faces the postsynaptic density, with only 1–2% of the total vesicle population. The vesicles in the RRP are docked and primed (i.e., SNARE complex has formed) and are ready for fusion and neurotransmitter exocytosis in $\ll 1$ s in the presence of Ca^{2+} . The vesicles of the recycling and reserve pools will release their

neurotransmitter on the order of seconds to minutes and in response to moderate to intense (e.g., 5–10 Hz in frog neuromuscular junction or 30 Hz in *Drosophila* larval neuromuscular junction) stimulation frequencies. The synaptic vesicle cycle consists of the endocytosis and subsequent recycling of vesicles that have bound to the presynaptic membrane and released neurotransmitter. This endocytosis of incorporated synaptic vesicles can be clathrin mediated for fully fused membranes or can consist of direct retrieval and recycling for vesicles that did not fuse completely for exocytosis (“kiss and run” fusion) (Gundelfinger, Kessels, & Qualmann, 2003; Kavalali, 2015; Nicholls et al., 2001; Purves, 2008; Rizzoli & Betz, 2005).

The action potential

The electrical signals that propagate over distances within the CNS and enable neurotransmitter release are called action potentials. Action potentials are an all-or-nothing event that are a consequence of the transmembrane electrochemical gradient of local sodium (Na^+) and potassium (K^+) ions. Prior to the evocation of an action potential, when the cell is at rest, resting membrane potential is ~ -65 mV (range: ~ -40 to -90 mV). At equilibrium, there is higher extracellular Na^+ concentration than intracellular ($[\text{Na}^+]_e \approx 145$ mM, $[\text{Na}^+]_i \approx 5$ – 15 mM) and higher intracellular K^+ concentration than extracellular ($[\text{K}^+]_e \approx 5$ mM, $[\text{K}^+]_i \approx 140$ mM). The resting membrane is most permeable to K^+ compared with other ions, underlying the resting membrane potential being close to the equilibrium potential of K^+ (~ -58 mV).

When a threshold potential is reached that is depolarized relative to rest (~ -50 vs. ~ -65 mV), an action potential is triggered. Voltage-gated Na^+ channels open, allowing an influx of Na^+ into the cell along its electrochemical gradient; this is the primary depolarization phase of the action potential. Spike amplitude will peak at around $\sim +25$ mV before the repolarization phase begins, in which voltage-gated Na^+ channels are beginning to inactivate and voltage-gated K^+ channels are opening. This efflux of K^+ repolarizes the membrane potential to resting potential and overshoots it due to the slow closing of these channels. The shape and timing of action potentials can vary considerably across cell types, but the typical total duration of an action potential is 2–5 ms. Finally, Na^+/K^+ -ATPase, an active transporter pump that relies on energy from the hydrolysis of ATP, actively pumps 3 Na^+ out of the cell for every 2 K^+ brought in to return the cell to steady-state resting potential and establish the electrochemical gradients necessary to facilitate subsequent action potentials (Bean, 2007).

Following an action potential, the neuron enters a refractory period, in which it is either impossible (the absolute refractory period, initial ~ 1 ms following action potential) or more difficult (the relative refractory period, ~ 2 – 4 ms following the absolute refractory period) to attain threshold. This is due to the initial depolarization causing in the delayed action of K^+ channels and inactivation of Na^+ channels. This period of enforced silence limits the highest frequency of action potential occurrence (Nicholls et al., 2001; Purves, 2008).

Excitatory and inhibitory postsynaptic potentials

Small induced potential differences at the individual synapse level are known as postsynaptic potentials and are defined by the manner in which they affect the probability of action potential production. Excitatory postsynaptic potentials (EPSPs) increase the likelihood of action potential occurrence, whereas inhibitory postsynaptic potentials (IPSPs) decrease it. Generally, EPSPs and IPSPs can be differentiated by where their reversal potential, the potential at which current polarity is reversed, is relative to action potential threshold. For example, a net flow of cations causing depolarization (e.g., flow of Na^+/K^+) upon activation of an *N*-methyl-D-aspartate (NMDA) glutamate receptor will have a reversal potential that is depolarized relative to resting membrane potential (e.g., 0 mV compared to -60 mV), inducing a depolarization that

increases the probability of threshold being achieved. Alternatively, a hyperpolarizing inward flow of anions (e.g., chloride $[\text{Cl}^-]$) due to a reversal potential that is more negative than resting potential will drive an IPSP and a reduction in likelihood that threshold will be achieved. It should be noted that a depolarizing flow of ions will still drive an IPSP if its reversal potential is negative relative to threshold potential, despite being positive relative to resting potential. It is the cumulative contribution of incident EPSPs and IPSPs from all synapses received by the postsynaptic cell, integrated spatially and temporally, that determines if membrane potential threshold is achieved and an action potential evoked (Nicholls et al., 2001; Purves, 2008).

Ionotropic and Metabotropic Receptors

Ionotropic receptors across the synaptic cleft at the postsynaptic membrane serve as ligand-gated ion channels. These allow for direct synaptic transmission driven by the binding of released presynaptic neurotransmitters. Metabotropic receptors, on the other hand, typically act through second messenger systems such as GTP-binding proteins (G-proteins) and are thus considered “slow” and indirect. These receptors are not themselves ion channels, as in the case of ionotropic receptors, and instead can modify the activity of ion channels or receptor proteins. G-protein-coupled receptors (GPCRs) can also commonly act at the level of gene expression, modulating transcription through, for example, the cyclic AMP-protein kinase A pathway (Nicholls et al., 2001; Purves, 2008).

Neurotransmitters

The chief low-molecular-weight (non-peptide) CNS neurotransmitters can be delineated in three ways: the biogenic amines, amino acids, and acetylcholine. A single neuron can release multiple neurotransmitters, often at least one low-molecular-weight neurotransmitter as well as neuropeptides. Among the biogenic amines or monoamines include the catecholamines, which are derived from tyrosine and defined by the possession of a benzene ring with two adjacent hydroxyl groups: dopamine, norepinephrine (NE, noradrenaline), and epinephrine (adrenaline). Another class of biogenic amine is the tryptophan-derived 5-hydroxytryptamine (5-HT, serotonin), an indoleamine (containing a benzene ring fused with a pyrrole ring). Relatively few neurons in the CNS release these biogenic amines, and most are localized to nuclei in the brain stem. The amino acid neurotransmitters include glycine, glutamate, and γ -aminobutyric acid (GABA). A key component of synaptic transmission via neurotransmitter is clearance of transmitter from the synaptic cleft. This can be done by diffusion, degradation (e.g., acetylcholinesterase is an enzyme that hydrolyzes ACh to choline and acetate), or reuptake by glia or nerve terminals (Nicholls et al., 2001; Purves, 2008).

Glutamate

Glutamate is an amino acid that serves as the primary excitatory neurotransmitter of the CNS. The four classes of glutamate receptors are NMDA, α -amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA), kainate, and metabotropic glutamate receptors (mGluRs). The ionotropic channels are all nonselective cation channels, which enable consistent production of excitatory postsynaptic responses. The mGluRs are slower and can either increase or

decrease postsynaptic excitability. Increased glutamate concentrations have been associated with depression and bipolar disorder. Elevated glutamate at the synaptic cleft, for example, following neural injury such as status epilepticus, trauma, or ischemic stroke, can be excitotoxic and lead to the destruction of postsynaptic neurons (Nicholls et al., 2001; Purves, 2008; Raiteri, 2006).

GABA

GABA and glycine are amino acids that are the principal inhibitory neurotransmitters in the CNS. The primary target receptors of GABA are GABA_A receptor (GABA_AR) and metabotropic GABA_BR. GABA_AR preferentially permits flow of Cl⁻ ions (anions). Extracellular Cl⁻ concentration is greater than intracellular concentration ($[Cl^-]_e \approx 110 \text{ mM}$, $[Cl^-]_i \approx 4\text{--}30 \text{ mM}$), so Cl⁻ enters and consequently hyperpolarizes the cell, decreasing the likelihood of threshold potential being achieved and an action potential produced. Presynaptic GABA_BR receptors suppress the activity of voltage-gated Ca²⁺ channels, while postsynaptic GABA_BR receptors activate inward rectifier K⁺ channel. GABA_BR is unique in that it is sensitive to allosteric modulation. These receptors have binding sites that act as targets for anti-anxiety benzodiazepines (e.g., diazepam [Valium] and chlordiazepoxide [Librium]) and anticonvulsant barbiturates (e.g., phenobarbital) (Nicholls et al., 2001; Purves, 2008; Raiteri, 2006).

Dopamine

Dopamine is both an excitatory and inhibitory (dependent on target receptor) catecholamine with five identified metabotropic receptors (D₁₋₅). Dopamine-containing nuclei and their projections are involved in regulating selection and execution of motor behaviors, problem solving, regulation of emotion, response to reward, and motivation. It is because of the influence of these pathways that antipsychotics for treating schizophrenia and bipolar disorder target dopamine receptors (e.g., aripiprazole [Abilify], a partial agonist of the D₂ receptor, and olanzapine [Zyprexa], a D₂ antagonist). Furthermore, the degeneration of dopaminergic neurons in the pars compacta of the substantia nigra is the defining feature of Parkinson's disease. A reduction of presynaptic dopaminergic axon terminals drives a loss of dopamine in the basal ganglia, leading to downstream motor disorders such as difficulty in initiating movement and tremor at rest. L-DOPA, the precursor for dopamine, can be used pharmacologically to improve dopamine concentrations and reduce associated motor deficits (Nemeroff & Owens, 2002; Nicholls et al., 2001; Purves, 2008; Raiteri, 2006; Schatzberg & Nemeroff, 2009).

Serotonin

Serotonin is a predominantly excitatory monoamine (indoleamine) neurotransmitter. All serotonin (5-HT) receptors except for the cation-selective ionotropic 5-HT₃ receptor are metabotropic. Serotonin receptors can act broadly and influence activity across the cortex by modulating the release of many neurotransmitters (e.g., glutamate, GABA, ACh) through inhibition, facilitation, or stimulation of specific neurotransmitter exocytosis by a postsynaptic neuron. Serotonin projections originate at the raphe nuclei of the midbrain. Serotonergic neurons are involved with arousal and the sleep-wake cycle, mood, aggression, and the establishment of social dominance. The monoamines, including 5-HT, are the primary neurotransmitters implicated in depression. Pharmacotherapy via antidepressants acts by either inhibiting

monoamine oxidase, which degrades monoamine neurotransmitters and excites or inhibits pre-/postsynaptic receptors that regulate neurotransmitter release and neuronal firing rates, or by blocking presynaptic monoamine transporter proteins (which remove neurotransmitter from the synaptic cleft). Examples of this final class of antidepressants include selective serotonin reuptake inhibitors (SSRIs) (e.g., fluoxetine [Prozac] and citalopram [Celexa]) and serotonin–norepinephrine reuptake inhibitors (SNRIs) (e.g., duloxetine [Cymbalta] and venlafaxine [Effexor]) (Nemeroff & Owens, 2002; Nicholls et al., 2001; Purves, 2008; Schatzberg & Nemeroff, 2009).

Acetylcholine

Acetylcholine (ACh) is an excitatory small-molecule neurotransmitter that is involved with memory, learning, and cognitive performance. Cholinergic neurons project from the basal forebrain nucleus and innervate most of the cortex. Cholinergic receptors are classified based on their responsiveness to nicotine (“nicotinic” acetylcholine receptor [nAChR]) or muscarine (“muscarinic” acetylcholine receptor [mAChR]). nAChRs are cationic ionotropic receptors, whereas mAChRs are metabotropic. nAChRs are preferentially impacted by amyloid-beta plaque deposition in Alzheimer’s disease (AD) (compared with mAChRs), more so than the effects seen due to normal aging, providing a potential therapeutic target for AD-associated cognitive decline. AD patients are currently often prescribed drugs that are acetylcholinesterase inhibitors (e.g., donepezil [Aricept]), thus increasing the concentration of ACh (Nicholls et al., 2001; Purves, 2008; Raiteri, 2006; Sadigh-Eteghad, Majdi, Talebi, Mahmoudi, & Babri, 2015).

Norepinephrine and Epinephrine

Norepinephrine (noradrenaline) and epinephrine (adrenaline) are excitatory catecholamines that share metabotropic noradrenergic receptors (α and β receptors). The primary source of norepinephrine in the brain is the locus coeruleus in the brain stem. Norepinephrine is involved with dysphoria, fatigue, apathy, and cognitive dysfunction. It regulates circadian patterns of melatonin synthesis in mammals. Epinephrine has the lowest concentration in the brain among the catecholamines and is found in the fewest neurons. No plasma membrane transporter specific for epinephrine has been identified. Epinephrine plays an important role in driving a sympathetic response in autonomic nervous system, but it also is involved in memory consolidation within the CNS (Cahill & Alkire, 2003; Nicholls et al., 2001; Purves, 2008; Raiteri, 2006; Schatzberg & Nemeroff, 2009).

Glia

In addition to neurons, a family of non-neuronal support cells, called glia, are present in the CNS. In the combined cortical gray and white matter, glia are more numerous, outnumbering neurons by $>3.5 : 1$ (Azevedo et al., 2009). Additionally, they lack axons and have more negative resting potentials (-90 vs. ~ -65 mV) compared with neurons. Glia are involved with maintaining ion concentrations in the neuropil, regulating the uptake of neurotransmitters at or near the synaptic cleft, and controlling rate of action potential propagation among other support roles. Also, glial cells react to neuronal injury by replication and will propagate and occupy the

injured area after all cellular debris has been removed. They can then secrete the extracellular matrix glycoprotein laminin to encourage neurite outgrowth or neoangiogenesis, for example, after ischemic stroke (Ji & Tsirka, 2012). The primary glial cell types are oligodendrocytes, microglia, ependymal cells, radial glial cells, and astrocytes (Nicholls et al., 2001; Purves, 2008).

Oligodendrocytes, Microglia, Ependymal Cells, and Radial Glial Cells

Oligodendrocytes are primarily found within white matter regions in the brain where they form myelin sheaths around neuron axons. This subsequently aids in increasing speed of electrical transmission. Microglia are phagocytic, specialized macrophages that are involved in scavenging and clearing debris from sites of injury or normal cell turnover. They can release cytokines as part of an inflammatory response as well as prune synapses by monitoring synaptic transmission. Ependymal cells form an epithelial layer in CNS ventricular spaces. Their apical surfaces have cilia that circulate cerebrospinal fluid (CSF) in the ventricles. Modified ependymal cells, joined by gap junctions, surrounding a core of capillaries and connective tissue collectively make up the choroid plexus, which produces CSF. Finally, radial glial cells act to guide neuronal migration during development; their processes act as physical tracks that migrating neurons traverse along toward their target region and synapse (Fields et al., 2014; Nicholls et al., 2001; Purves, 2008).

Astrocytes

Astrocytes interface with neurons and capillaries and can ensheath thousands of synapses. The two chief morphological classifications of astrocytes are fibrous and protoplasmic astrocytes. Fibrous astrocytes contain filaments and are found primarily within white matter. Their end feet envelop nodes of Ranvier. Protoplasmic astrocytes are abundant in gray matter and have end feet that surround synapses. Astrocytes serve a number of different functions in the CNS. They are involved with neurovascular coupling and can control local blood flow through release of arachidonic acid derivatives that can act on smooth muscle surrounding oxygen-supplying arterioles to either constrict or dilate vessel diameter (Attwell et al., 2010). Astrocytes also modify the concentration of extracellular K^+ , neurotransmitters, and neuromodulatory substances. They can also modify the volume and geometry of the immediate extracellular space (Fields et al., 2014).

Author Biography

Patrick W. Wright is currently a postdoctoral research fellow under Alan Koretsky, PhD, in the Laboratory of Functional and Molecular Imaging at the National Institute of Neurological Disorders and Stroke and the National Institutes of Health in Bethesda, Maryland. He received his BS in Biomedical Engineering from the University of Alabama at Birmingham in 2010. He subsequently earned his PhD in Biomedical Engineering from Washington University in St. Louis in 2016 with Joseph Culver, PhD, and Beau Ances, MD, PhD, serving as his thesis advisers. During graduate school, he developed an *in vivo* optical imaging system to study resting-state cortical activity using concurrently measured hemodynamics and fluorescent calcium transients in mice. He also used clinical MRI data to examine the effects of early HIV infection on the brain.

References

- Attwell, D., Buchan, A. M., Charpak, S., Lauritzen, M., Macvicar, B. A., & Newman, E. A. (2010). Glial and neuronal control of brain blood flow. *Nature*, 468(7321), 232–243. doi:10.1038/nature09613. PubMed PMID: 21068832; PubMed Central PMCID: PMCPMC3206737.
- Azevedo, F. A., Carvalho, L. R., Grinberg, L. T., Farfel, J. M., Ferretti, R. E., Leite, R. E., ... Herculano-Houzel, S. (2009). Equal numbers of neuronal and nonneuronal cells make the human brain an isometrically scaled-up primate brain. *Journal of Comparative Neurology*, 513(5), 532–541. doi:10.1002/cne.21974. PubMed PMID: 19226510.
- Bean, B. P. (2007). The action potential in mammalian central neurons. *Nature Reviews. Neuroscience*, 8(6), 451–465. doi:10.1038/nrn2148. PubMed PMID: 17514198.
- Cahill, L., & Alkire, M. T. (2003). Epinephrine enhancement of human memory consolidation: Interaction with arousal at encoding. *Neurobiology of Learning and Memory*, 79(2), 194–198. PubMed PMID: 12591227.
- Fields, R. D., Araque, A., Johansen-Berg, H., Lim, S. S., Lynch, G., Nave, K. A., ... Wake, H. (2014). Glial biology in learning and cognition. *The Neuroscientist*, 20(5), 426–431. doi:10.1177/1073858413504465. PubMed PMID: 24122821; PubMed Central PMCID: PMCPMC4161624.
- Gundelfinger, E. D., Kessels, M. M., & Qualmann, B. (2003). Temporal and spatial coordination of exocytosis and endocytosis. *Nature Reviews. Molecular Cell Biology*, 4(2), 127–139. doi:10.1038/nrm1016. PubMed PMID: 12563290.
- Ji, K., & Tsirka, S. E. (2012). Inflammation modulates expression of laminin in the central nervous system following ischemic injury. *Journal of Neuroinflammation*, 9, 159. doi:10.1186/1742-2094-9-159. PubMed PMID: 22759265; PubMed Central PMCID: PMCPMC3414761.
- Kavalali, E. T. (2015). The mechanisms and functions of spontaneous neurotransmitter release. *Nature Reviews. Neuroscience*, 16(1), 5–16. doi:10.1038/nrn3875. PubMed PMID: 25524119.
- Nemeroff, C. B., & Owens, M. J. (2002). Treatment of mood disorders. *Nature Neuroscience*, 5(Suppl), 1068–1070. doi:10.1038/nn943. PubMed PMID: 12403988.
- Nicholls, J. G., Martin, A. R., Wallace, B. G., & Kuffler, S. W. (2001). *From neuron to brain: A cellular and molecular approach to the function of the nervous system* (4th ed., pp. xx). Sunderland, MA: Sinauer Associates, 807 p.
- Pereda, A. E. (2014). Electrical synapses and their functional interactions with chemical synapses. *Nature Reviews. Neuroscience*, 15(4), 250–263. doi:10.1038/nrn3708. PubMed PMID: 24619342; PubMed Central PMCID: PMCPMC4091911.
- Purves, D. (2008). *Neuroscience* (4th ed.). Sunderland, MA: Sinauer.
- Raiteri, M. (2006). Functional pharmacology in human brain. *Pharmacological Reviews*, 58(2), 162–193. doi:10.1124/pr.58.2.5. PubMed PMID: 16714485.
- Rizzoli, S. O., & Betz, W. J. (2005). Synaptic vesicle pools. *Nature Reviews. Neuroscience*, 6(1), 57–69. doi:10.1038/nrn1583. PubMed PMID: 15611727.
- Sadigh-Eteghad, S., Majdi, A., Talebi, M., Mahmoudi, J., & Babri, S. (2015). Regulation of nicotinic acetylcholine receptors in Alzheimers disease: A possible role of chaperones. *European Journal of Pharmacology*, 755, 34–41. doi:10.1016/j.ejphar.2015.02.047. PubMed PMID: 25771456.
- Schatzberg, A. F., & Nemeroff, C. B. (2009). *The American Psychiatric Publishing textbook of psychopharmacology* (4th ed., pp. xxxii). Washington, DC: American Psychiatric Publishing, 1616 p.

Functional Neuroanatomy: Cortical–Subcortical Distinctions and Pathways

David Von Nordheim and Jodi M. Heaps-Woodruff

*Missouri Department of Mental Health, University of Missouri–St. Louis,
St. Louis, MO, USA*

Missouri Institute of Mental Health, St. Louis, MO, USA

The cerebrum, also known as the forebrain, is the largest area of the mammalian brain and the most recently developed in evolutionary history. Consisting of all brain structures above the cerebellum, the cerebrum is further divided into the cerebral cortex, the brain's convoluted outer surface, and the subcortical structures beneath. The cerebrum is responsible for the higher order processes that give rise to thought, emotion, and sensory experiences.

The structures of the cerebral cortex are central to processing sensory, motor, and perceptual information. These structures are intricately connected, allowing information from disparate processing streams to combine, producing a harmonized response within a dynamic environment. The coordination between cortical and subcortical structures occurs via networks of feedback loops designed to process continuously updated sensory information. To understand the coordination between subcortical structures and different regions of the cortex, we will describe the connections between the subcortical nuclei and the cortical–subcortical feedback loops.

The Thalamus

The thalamus is a pivotal structure involved in coordinating communication between the cortex and subcortex. With the notable exception of olfaction, the thalamus is the first destination for all sensory information, earning its title as the “relay station” of the brain. The thalamus is divided into nuclei dedicated to processing sensory information and relaying information back to the relevant cortical areas. As an example, visual information initially travels from

the retina to the lateral geniculate nucleus (LGN) of the thalamus, where it is subsequently transmitted to the primary visual cortex of the occipital lobe. As described further in the chapter, the thalamus also processes incoming information from other subcortical structures.

Basal Ganglia

The basal ganglia consist of a cluster of subcortical nuclei, including the striatum, the globus pallidus, ventral pallidum, substantia nigra, and subthalamic nucleus (STN). The striatum is divided into four nuclei: the caudate and putamen (dorsal striatum) and the nucleus accumbens (NAcc) and olfactory tubercle (ventral striatum). The dorsal and ventral striatal regions are innervated by both cortical and subcortical afferents. The caudate has been described as having a critical role in planning and execution of goal-directed behavior (Grahn, Parkinson, & Owen, 2008). A recent functional connectivity study described an age-related reduction in differentiation of connections between the caudate and cortical networks. Specifically, caudate areas in younger adults were connected to the frontal parietal control network (FPN), whereas these same areas were connected to the default network (DN) in older adults. This difference in connectivity is related to memory decline in adults over the age of 75. This same study also reported that reduction dopamine transporter density in the striatum predicted memory decline in older adults (Rieckmann, Johnson, Sperling, Buckner, & Hedden, 2018).

The putamen is interconnected with the primary motor, primary somatosensory area, and premotor cortex. As such, damage observed in the putamen results in movement disorders. A recent study determined that children with complex motor stereotypies (repetitive and rhythmic movements) displayed diminished size of the putamen compared with controls (Mahone et al., 2016). Additionally, connections between the cerebellum and the putamen are active in parallel with the putamen to primary motor cortex, likely coordinating the sequence and learning of motor movements (Penhune & Steele, 2012). The internal capsule, a dense white matter tract that carries sensory and motor information from the spinal cord to the frontal cortex, separates the caudate and putamen. Lesions in the internal capsule commonly result in motor impairments, though the location of the lesion may produce variations in symptomatology. Lesions in the internal capsule resulting from stroke are associated with poorer recovery (Kobayashi et al., 2015).

The nuclei of the ventral striatum—the NAcc and the olfactory tubercle—are involved in components of behavioral reinforcement. The olfactory tubercle is located in both the ventral striatum and the olfactory cortex and, despite its name, does not appear to have a role in olfaction. The NAcc is often referred to as the “pleasure center” of the brain; however, recent research has characterized the function of the NAcc a bit differently. NAcc function is related to motivated actions toward relevant stimuli and suppression of actions that would deter unsuitable actions (Floresco, 2015). The structures of the ventral striatum are strongly coactive with the limbic system, a network of cortical and subcortical circuits that process emotions and episodic memories, implicating the role of emotion and memory in the development and maintenance of motivational behaviors.

Globus Pallidus

The globus pallidus is composed of the internus (GPi) and the externus (GPe) regions. The GPi receives inputs from both the excitatory direct path via the striatum and inhibitory, indirect

path via the STN. Inputs from the striatum inhibit the activity of the GPi, preventing the release of gamma-aminobutyric acid (GABA) from GPi to the thalamus. The GPi is a target for deep brain stimulation of dystonias or movement disorders that produce uncontrolled contractions of muscles that result in irregular twisting and abnormal postures. The GPe once thought to have a singular function of suppressing motor activity via inputs from the STN recently has been described as having diversified projections through distinct subconnections (Gittis et al., 2014). The GPe is a fairly active area of research, as effective treatment of Parkinson's disease can be achieved through deep brain stimulation of the GPe (Carvalho, SuKul, Bookland, Koch, & Connolly, 2014).

Subthalamic Nucleus

Described in some detail above, the STN not only receives input via the indirect pathway but also receives direct input from cortical areas. A small structure subnuclei within the STN are associated with connectivity between motor, association networks, and the limbic system. Like the GPe, the STN is a potential target for deep brain stimulation. A recent functional connectivity study revealed that in Parkinson's patients, the STN had developed increased connectivity in two particular cortical areas (motor and precuneus), whereas controls had a more dispersed pattern of connectivity (Fernández-Seara et al., 2015).

The final major structure of the basal ganglia, the substantia nigra, is a small nucleus located in the most ventral section of the subcortex. It has two distinct regions, the substantia nigra pars compacta (SNpc) and the substantia nigra pars reticulata (SNpr). The SNpc produces and releases the majority of dopamine in the brain. A decrease in SNpc dopamine is characteristic of Parkinson's disease; however, the SNpc also plays a role in circuits between the striatum and prefrontal cortex and is implicated in the cognitive dysfunctions associated with schizophrenia (Yoon, Minzenberg, Raouf, D'Esposito, & Carter, 2013). The SNpr receives input from both the striatum (direct pathway) and the STN (indirect pathway). Inhibitory input from the striatum can prevent the SNpr from sending inhibitory GABA signals to the thalamus, increasing the potential for motor activity. Likewise, the SNpr can also receive excitatory glutamatergic signals from the STN, ultimately resulting in decreased potential for motor activity.

As described above, cortical–subcortical communication originates within the sensory regions of the cortex. The subcortical structures organize and coordinate a response through either the direct excitatory stream or indirect inhibitory stream (see Figure 1). Specific feedback loops have been defined that characterize the patterns or circuits of communication between cortical and subcortical structures. The loops are detailed below, with brief examples of different disease processes that result from alterations in the communication patterns of the feedback loops.

Skeletomotor Loop

The skeletomotor loop facilitates communication between the motor cortices and subcortical structures to produce movement. A consequence of dysfunction in the skeletomotor loop is impaired muscle movement, resulting in range of symptoms referred to as dyskinesias. Dyskinesias can be characterized as ranging from slow or hypokinetic movements resulting from excitatory pathway dysfunction to fast or hyperkinetic movements resulting from damage within the inhibitory pathway. Examples include bradykinesia, dystonia, athetosis, chorea, ballisms, and tics. Bradykinesias are hypokinesias that appear as slowed, clumsy, or inaccurate

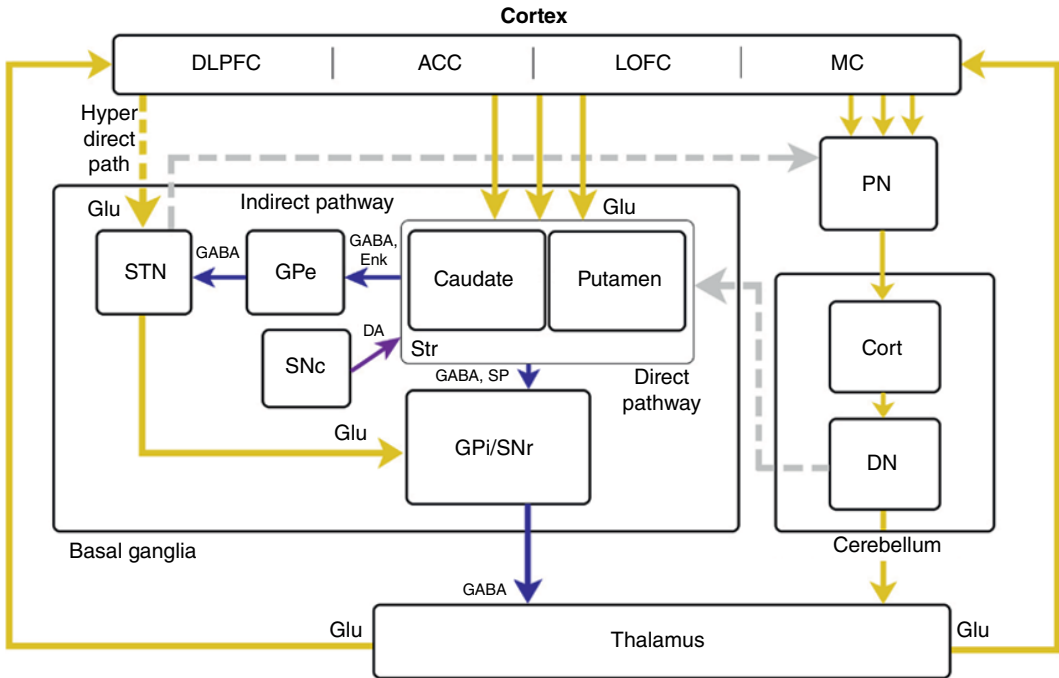


Figure 1 This diagram depicts a portion of the neural circuits and their interactions. Arrows represent signals from GABA, glutamergic and dopaminergic signals. The dashed grey lines represent the interconnected circuits between the basal ganglia and the cerebellum. The abbreviations go as follows: The Dorsolateral Prefrontal Cortex (DLPFC), Anterior Cingulate Cortex (ACC), Lateral Orbitofrontal Cortex (LOFC), Motor Cortex (MC), Globus Pallidus externus (GPe), Subthalamic Nucleus (STN), Substantia Nigra pars compacta (SNc), Globus Pallidus internus (GPi), Substantia Nigra pars reticulata (SNr), Pontine Nuclei (PN), Cerebellar Cortex (Cort), Dentate Nucleus (DN). *Source:* Original framework for this diagram can be found at Caligiore et al. (2017), courtesy of Daniele Caligiore. Reproduced with permission of Elsevier.

movements as well as poverty or lack of movement, resulting from impairments with initiated movements. Symptoms of dytonias include involuntary contractions of muscles, creating repetitive or twisting movements of various body parts, including the entire torso. Athetosis, a condition observed in athetoid cerebral palsy, consists of involuntary, writhing movements of the hands and feet. In some patients athetosis may affect arms, legs, neck, and even the tongue. Choreas or chorionic movements are characteristic of Huntington's disease and include erratic movements that lack purpose and may include awkward movement of the facial muscles. Ballism, typically hemiballismus, is a severe form of movement disorder that involves involuntary, violent, or extreme motions that occur on one side of the body. Hemiballismus is the result of damage to the STN located on the side contralateral to the movements. Finally, tics are hyperkinesias that appear as rapid, repetitive involuntary movements often associated with conditions such as autism and Tourette's syndrome.

Oculomotor Loop

The oculomotor loop is a circuit including the frontal eye fields, prefrontal cortex, posterior parietal cortex, the caudate nucleus, GPi, SNpr, superior colliculus, and visual regions of the

thalamus. One function of the oculomotor loop includes suppression of unnecessary eye movements and enables the eyes to fixate on a target. Signals from this loop originate in the posterior parietal lobe and dorsolateral prefrontal cortex (DLPFC), structures associated with spatial mapping and attentional processing. The thalamus ultimately initiates or inhibits eye movement through projections to the frontal and supplementary receptive fields of the eyes. Dysfunctions in some psychiatric disorders include inaccuracies in visual fixation. A current report indicates that schizophrenia and obsessive–compulsive disorder share both overlapping and unique patterns of dysfunction. Shared deficits appear to involve the DLPFC, anterior cingulate, and parietal circuits, whereas additional deficits in schizophrenia pointed to motor control and frontal eye field dysfunction (Damilou, Apostolakis, Thrapsanioti, Theleritis, & Smyrnis, 2016).

Executive–Associative Loops

Numerous circuits between the prefrontal cortex and subcortical structures have been defined and are continually being redefined through the use of neuroimaging tools that provide new insights into functional connections. One loop, the DLPFC, is associated with cognitive processes, including outcome prediction, working memory, reasoning, inhibition, and conscious decision making. The DLPFC loop has received a great deal of attention as it is implicated in both positive and negative behaviors associated with schizophrenia. Specifically, damage to this circuit is associated with substantial deficits in social cognition, including difficulties in reasoning and failure to follow rules (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). The more ventrally positioned orbitofrontal cortex has both medial (mOFC) and lateral (lOFC) circuits that display distinct connectivity. These circuits are implicated in goal-directed behaviors including reward learning, mood regulation, emotional reappraisal, and decision making. The mOFC is implicated in assigning value to stimuli, whereas the lOFC is implicated in appraising or calculating the value between stimuli (Fettes, Schulze, & Downar, 2017).

Limbic Loop

The limbic loop is a circuit of nuclei including the anterior cingulate cortex (ACC), medial prefrontal cortices, NAcc, GPi, and thalamus. A variety of psychopathologies are associated with dysfunction in this circuit, including Tourette's syndrome and obsessive–compulsive disorder (Devinsky, Morrell, & Vogt, 1995). Dysregulation of the thalamus is hypothesized to result in overstimulation of the ACC, resulting in repetitive, involuntary behaviors. On the other hand, thalamic dysregulation may also result in inadequate stimulation of the ACC. Hypostimulation of the ACC may contribute to the profound apathy observed in akinetic mutism, a disorder characterized by an indifference to hunger, thirst, or pain, a lack of spontaneous movement, and an inability to respond to questions and commands (Holroyd & Yeung, 2012).

Conclusion

Because of the intricate connectivity between cortical and subcortical structures, the damage caused by neurological diseases or lesions is unlikely to be isolated entirely within the cortex

or subcortex. Additionally, the loops as presented, while distinct circuits, have further interconnections not described here. Certain pathologies, as described in this chapter, might result from lesions or lack a diffuse pattern of connectivity (Fernández-Seara et al., 2015; Rieckmann et al., 2018). As has been demonstrated in this chapter, damage to the cortex can result in dysfunction in subcortical communications; likewise, damage to subcortical structures will eventually lead to reciprocal impairment and possible atrophy in cortical structures. Despite our relatively thorough understanding of cortical–subcortical feedback loops, there is much that is not well understood, including the role of other neuromodulators in producing behavior or disease processes. Ongoing research using multimodal imaging and examination of the role of other body systems (e.g., the gut–brain axis, immune system) in disease states will provide greater understanding of the complexity of cortical–subcortical communication systems.

Author Biographies

David Von Nordheim earned his Masters of Arts in Cognitive Psychology from the University of Nebraska–Omaha in May 2018. His research interests include memory, attention, word processing, and the cognitive psychology of addictive behaviors. His long-term goal is to earn a PhD in cognitive neuroscience and conduct neuropsychological research with clinical populations.

Jodi M. Heaps-Woodruff is a research neuropsychologist and health services researcher. She earned her PhD from the University of Missouri–St. Louis and is a research assistant professor at the Missouri Institute of Mental Health at the University of Missouri–St. Louis. Her research is focused on the cognitive aspects involved in recovery from mental illness and the impact of chronic disease on the brain.

References

- Caligiore, D., Pezzulo, G., Baldassarre, G., Bostan, A. C., Strick, P. L., Doya, K., ... Herreros, I. (2017). Consensus paper: Towards a systems-level view of cerebellar function: the interplay between cerebellum, basal ganglia, and cortex. *Cerebellum*, 16, 203–229. <https://doi.org/10.1007/s12311-016-0763-3>
- Carvalho, K. S., Sukul, V. V., Bookland, M. J., Koch, S. A., & Connolly, P. J. (2014). Deep brain stimulation of the globus pallidus suppresses post-traumatic dystonic tremor. *Journal of Clinical Neuroscience*, 21(1), 153–155.
- Damilou, A., Apostolakis, S., Thrapsanioti, E., Theleritis, C., & Smyrnis, N. (2016). Shared and distinct oculomotor function deficits in schizophrenia and obsessive compulsive disorder. *Psychophysiology*, 53(6), 796–805.
- Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain*, 118(1), 279–306.
- Fernández-Seara, M. A., Mengual, E., Vidorreta, M., Castellanos, G., Irigoyen, J., Erro, E., & Pastor, M. A. (2015). Resting state functional connectivity of the subthalamic nucleus in Parkinson's disease assessed using arterial spin-labeled perfusion f MRI. *Human Brain Mapping*, 36(5), 1937–1950.
- Fettes, P., Schulze, L., & Downar, J. (2017). Cortico-striatal-thalamic loop circuits of the orbitofrontal cortex: Promising therapeutic targets in psychiatric illness. *Frontiers in Systems Neuroscience*, 11, 25.
- Floresco, S. B. (2015). The nucleus accumbens: An interface between cognition, emotion, and action. *Annual Review of Psychology*, 66, 25–52.

- Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., & Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. *Psychological Medicine*, 31(2), 189–195.
- Gittis, A. H., Berke, J. D., Bevan, M. D., Chan, C. S., Mallet, N., Morrow, M. M., & Schmidt, R. (2014). New roles for the external globus pallidus in basal ganglia circuits and behavior. *Journal of Neuroscience*, 34(46), 15178–15183.
- Grahn, J. A., Parkinson, J. A., & Owen, A. M. (2008). The cognitive functions of the caudate nucleus. *Progress in Neurobiology*, 86(3), 141–155.
- Holroyd, C. B., & Yeung, N. (2012). Motivation of extended behaviors by anterior cingulate cortex. *Trends in Cognitive Sciences*, 16(2), 122–128.
- Kobayashi, Z., Akaza, M., Endo, H., Numasawa, Y., Tomimitsu, H., & Shintani, S. (2015). Deterioration of pre-existing hemiparesis due to an ipsilateral internal capsule infarction after a contralateral stroke. *Journal of the Neurological Sciences*, 354(1–2), 140–141.
- Mahone, E. M., Crocetti, D., Tochen, L., Kline, T., Mostofsky, S. H., & Singer, H. S. (2016). Anomalous putamen volume in children with complex motor stereotypies. *Pediatric Neurology*, 65, 59–63.
- Penhune, V. B., & Steele, C. J. (2012). Parallel contributions of cerebellar, striatal and M1 mechanisms to motor sequence learning. *Behavioural Brain Research*, 226(2), 579–591.
- Rieckmann, A., Johnson, K. A., Sperling, R. A., Buckner, R. L., & Hedden, T. (2018). Dedifferentiation of caudate functional connectivity and striatal dopamine transporter density predict memory change in normal aging. *Proceedings of the National Academy of Sciences*, 115(40), 10160–10165.
- Yoon, J. H., Minzenberg, M. J., Raouf, S., D’Esposito, M., & Carter, C. S. (2013). Impaired prefrontal-basal ganglia functional connectivity and substantia nigra hyperactivity in schizophrenia. *Biological Psychiatry*, 74(2), 122–129.

Suggested Reading

- Corbetta, M., Ramsey, L., Callejas, A., Baldassarre, A., Hacker, C. D., Siegel, J. S., ... Connor, L. T. (2015). Common behavioral clusters and subcortical anatomy in stroke. *Neuron*, 85(5), 927–941.
- Coyle, J. T., Price, D. L., & Delong, M. R. (1983). Alzheimer’s disease: A disorder of cortical cholinergic innervation. *Science*, 219(4589), 1184–1190.
- Gutman, B., Bright, J., Rummel, C., Rocha, C. S., Debove, I., Yasuda, C., ... Wiest, R. (2018). Widespread cortical thinning in Parkinson’s disease: Findings from the ENIGMA-Parkinson’s Disease Working Group (S45.008). *Neurology*, 90(15 Supplement), S45.008.
- Heller, A. S. (2016). Cortical-subcortical interactions in depression: From animal models to human psychopathology. *Frontiers in Systems Neuroscience*, 10, 20.
- Kiverstein, J., & Miller, M. (2015). The embodied brain: Towards a radical embodied cognitive neuroscience. *Frontiers in Human Neuroscience*, 9, 237.
- Leunissen, I., Coxon, J. P., Caeyenberghs, K., Michiels, K., Sunaert, S., & Swinnen, S. P. (2014). Subcortical volume analysis in traumatic brain injury: The importance of the fronto-striato-thalamic circuit in task switching. *Cortex*, 51, 67–81.

Neurobiology of Stress–Health Relationships

R.L. Spencer, L.E. Chun, M.J. Hartsock, and
E.R. Woodruff

*Department of Psychology and Department of Neuroscience, University of Colorado,
Boulder, CO, USA*

Stress and Health Overview

The adverse effects that stress can have on physical and mental health are well established. Stress has a causal role in the development of some psychological disorders, such as major depressive disorder or posttraumatic stress disorder, and it can negatively impact all phases of physiological and psychological illness progression including precipitation of episode onset, symptom exacerbation, increased treatment resistance, delayed recovery, and increased relapse (Dimsdale, 2008; Reagan & Grillo, 2008). Recent life stress is reported to be a factor contributing to 60–80% of healthcare provider visits in the United States. Because most major stressors identified by members of our society are psychological rather than physiological in nature, understanding and mitigating the negative relationship between stress and health is a central issue for health psychology.

The Stress Concept

The foundation for the scientific study of stress was firmly established by the physiologist Walter Cannon and the endocrinologist Hans Selye. Cannon, as a physiologist at Harvard Medical School (1900–1945), coined the term homeostasis to represent the elaborate physiological processes that operate in a coordinated fashion to ensure the stability of cellular and physiological functions in the body. Cannon was instrumental in determining the critical role that the sympathetic nervous system (SNS) plays in restoring homeostasis in the face of a wide range of environmental or internal disturbances. He described the pluripotent effects of SNS activation

as mediating key physiological changes in the body, such as energy mobilization and increased cardiovascular function, that support the “fight-or-flight” response to physical threat.

Hans Selye, while working (1936–1982) at McGill University and Université de Montréal, extensively characterized in controlled laboratory experiments the physiological effects of stress. Whereas Cannon advanced the importance of the SNS, Selye determined that another key physiological mediator of the body’s stress response was increased secretion of glucocorticoid hormones as a result of hypothalamic–pituitary–adrenal (HPA) axis activation. Selye referred to stressors as the factors that cause stress and the stress response as the body’s adaptive response to stressors. He proposed the theory that a wide range of experiences (stressors) have the ability to exert a similar influence on the body (stress), which can lead to detrimental effects when sustained. However, the body can detect this stress state and effectively respond by mounting a coordinated stress response. Selye described this stress state as a nonspecific demand placed upon the body. Despite the various theoretical conundrums surrounding the nature of this stress state, researchers have made a number of important discoveries concerning the neurobiology of stress.

Types of Stressors

The categorization of stressors into two general types, physiological and psychological, has proven useful for studying the specific underlying neurocircuitry that participates in the processing and response coordination to these stressors. Physiological stressors are those altered physiological states that elicit well-characterized autonomic homeostatic adjustments, such as hypothermia, hypoxia, dehydration, tissue damage, hypotension, and hemorrhage. These stressors are also referred to as interoceptive or systemic stressors. An important finding of stress research has been that the two principal physiological stress response systems (SNS and HPA) are not only activated after a disruption of homeostasis has been detected but also by a variety of environmental or psychosocial situations that do not themselves disrupt physiological function. Some of these so-called psychological (or processive) stressors are environmental stimuli that appear to be innately recognized as an immediate threat to physiological well-being, such as physical attack, sudden loud noise, impending collision with a fast moving object, or teetering on the edge of a high precipice. However, physiological stress responses are also effectively triggered by many other experiential situations that do not reflect impending bodily harm, such as public speaking, novel social circumstances, traffic jams, relationship difficulties, financial insecurity, and job performance concerns. Moreover, psychological stressors may be self-generated (e.g., traumatic experience recall and anticipation of future negative outcomes). Research has identified several underlying psychological factors that are shared by various psychosocial situations that are considered stressful, and these factors include lack of controllability, lack of predictability, novelty, and social-evaluative threat.

The ability to respond to psychological stressors with a physiological stress response confers considerable adaptive advantage to the organism. If the organism is able to initiate a physiological stress response not only to a direct physical insult (e.g., attack from a predator) but also to cues associated with the stressor (e.g., odors, sounds, or sight of a nearby predator), then the physiological stress response may be vital for the organism’s ability to avoid the stressor or may optimally prepare the organism for a subsequent encounter with the stressor. Moreover, this capability can draw upon prior experience (i.e., learning and memory), such that previously neutral environmental cues, when paired with stressors, will subsequently be sufficient to elicit physiological stress responses. The ability not only to respond to immediate physical stressors but also to anticipate and prepare for future stressors is a key aspect of evolutionary stress neurobiology. However, in modern society, many complex psychosocial stressors are not

associated with impending physical threat to well-being, and consequently the activation of physiological stress responses in these situations is not adaptive and contributes to the adverse effects of chronic stress on health.

Types of Stress Responses

Physiological and psychological stressors elicit not only a physiological response but also psychological responses. The psychological responses often include generation of emotional states, such as fear, anxiety, or frustration, as well as shifts in attention (e.g., hypervigilance), decision-making (e.g., bias), and memory processes. This psychological aspect of the stress response is essential for motivation and coordination of adaptive behavioral responses. The importance of behavioral responses to stress was featured early on in stress response physiology by Cannon as he framed the significance of the SNS physiological response in terms of behavior—the “fight-or-flight response.” Other basic behavioral responses elicited by stressors include freezing and withdrawal, but the psychological aspect of the response to stress may also motivate elaborate behavioral strategies to avoid or reduce stress. No physiological or psychological response alone, or in combination, is a dedicated response exclusively reserved for combatting stress. Each can occur in other contexts, and therefore the presence of these responses is not a definitive measure of stress, although they may be useful biomarkers associated with stress.

Neurobiology of the Body’s Response to Stress

The ability of the body to detect stressors and then mount a coordinated stress response that effectively copes with the stressful situation is fundamentally a neurobiological process. It requires neural communication between sensory afferents and motor efferents. However, because there is not a physical dimension of stressfulness inherent in stimuli, the determination of stressfulness or threat requires integrative neural processing. The perception of and the coordinated response to stress depend on hierarchically organized neural systems in the brain. In the case of an appropriate response to physiological stressors, the underlying neural circuitry tends to be relatively simple. For example, a sudden drop in blood pressure results in reduced activity of baroreceptors located in the aortic arch and carotid artery sinus. These specialized sensory neurons project via the vagus and glossopharyngeal cranial nerves to the vasomotor control centers in the brain stem. The vasomotor control centers then direct an increase in activity of descending spinal cord premotor efferent neurons that innervate components of the SNS. Increased SNS activity then mediates a rapid increase in cardiac output and arteriole vasoconstriction. Superimposed on these evolutionarily primitive neurocircuits are more complex neural circuits and networks (e.g., components of the limbic system) that subserve detection of the wide range of psychological stressors and orchestration of the physiological and psychological responses directed toward reducing stress.

The two primary physiological stress response systems are the SNS and the HPA axis. Each system has both a neuronal and a hormonal component. The two systems mediate complementary adaptive changes in virtually every cell and system of the body, and both systems operate on a complementary time scale. The SNS typically produces rapid (within seconds) but short-lasting (minutes) actions, whereas the HPA axis typically produces delayed (45–60 min) but long-lasting (hours to days) actions. Individuals do not have voluntary control over the initiation or termination of activity within these two stress response systems, but they may learn strategies, such as relaxation techniques to modulate their activity.

SNS

The SNS (Figure 1) is one of two subdivisions of the autonomic nervous system (ANS) (Powley, 2013). The parasympathetic nervous system (PSNS) forms the other subdivision. The ANS is primarily a motor system that innervates and controls the activity of internal organs and glands throughout the body including the heart, lungs, liver, kidney, gastrointestinal

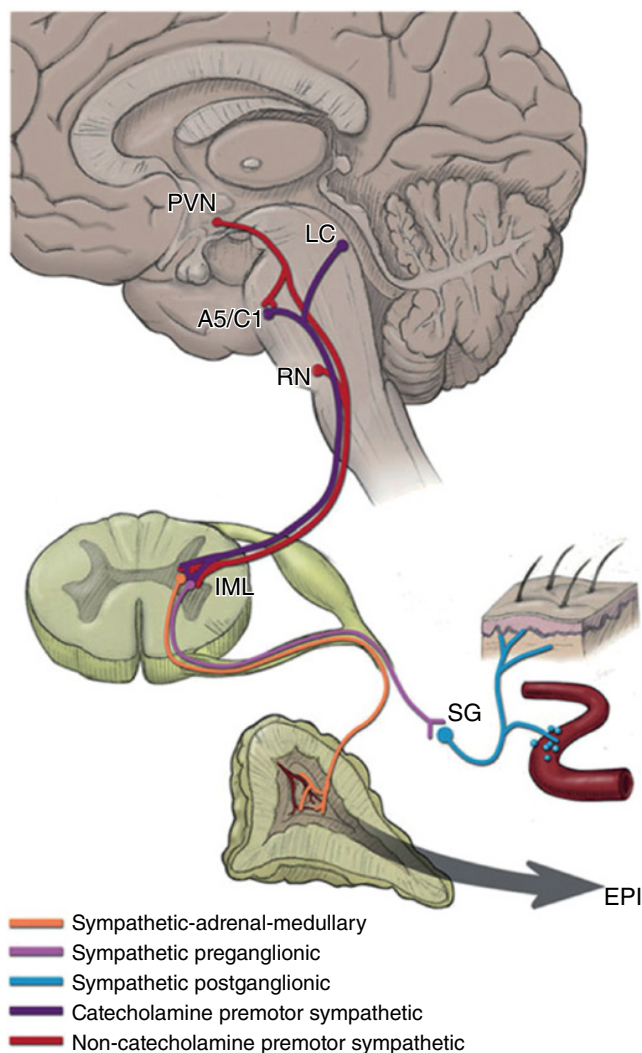


Figure 1 Neural pathways of the stress response. Depicted are examples of sympathetic preganglionic neuron projections from the intermediolateral cell column of the spinal cord (IML) to sympathetic postganglionic neurons in the sympathetic ganglion (SG) and to the adrenal medulla, where epinephrine (EPI) is released into the circulation from chromaffin cells. Also depicted is an example of sympathetic postganglionic neuron projections to skin and a section of a blood vessel, where norepinephrine is released from nerve terminals. Descending neural control originates in paraventricular nucleus of the hypothalamus (PVN), locus ceruleus (LC), A5 noradrenergic and C1 adrenergic cell groups (A5/C1), and raphe nuclei (RN). *Source:* Reprinted with permission from McCarty (2016). Reproduced with permission of Taylor & Francis.

system, bladder, reproductive organs, salivary glands, and lacrimal glands. The ANS also regulates blood pressure and blood flow to various parts of the body by adjusting the diameter of arterioles, and it regulates the amount of light that enters the eye by controlling the diameter of the iris. The ANS comprises a series of two neuron units (preganglionic neuron and postganglionic neuron) that connect the spinal cord and brain stem with the targeted organs and glands. In general, the SNS and PSNS have opposing actions on the activity and function of the target organs, for example, increased SNS activity leads to an increase in heart rate, and increased PSNS activity leads to a decrease in heart rate. Combined the SNS and PSNS provide the critical balance that largely underlies physiological homeostasis.

The SNS preganglionic neurons receive their primary inputs from descending brain stem neural pathways, which synapse onto the cell bodies of preganglionic sympathetic neurons located in the intermediolateral cell column of the thoracolumbar spinal cord. Axons of the preganglionic sympathetic neurons extend outside the spinal cord to synapse onto postganglionic cell bodies, many of which are located in sympathetic chain ganglia adjacent to the spinal column. The axons of postganglionic neurons terminate on target organs throughout the body. Preganglionic neurons release acetylcholine onto nicotinic cholinergic receptors present on postganglionic neurons, while postganglionic neurons release norepinephrine onto adrenergic receptors present on target organs. Norepinephrine release onto target organs triggers physiological changes associated with the fight-or-flight response, including pupil dilation, shunting of blood flow from the viscera to the skeletal muscles of the limbs, increased respiration, and heart rate acceleration.

In addition to this direct neural control of SNS target organs, an endocrine component of the SNS provides hormonal control over target organs. The chromaffin cells of the adrenal medulla comprise this endocrine component. The axons of sympathetic preganglionic neurons in the thoracic spinal cord extend as part of the splanchnic nerve to terminate on chromaffin cells. Chromaffin cells, in response, secrete the catecholamine molecules epinephrine (also known as adrenaline) and norepinephrine into the systemic circulation. Catecholamine hormones cannot cross the blood–brain barrier and therefore act only in the periphery, with epinephrine binding all classes of adrenergic receptors (α_1 , α_2 , β_1 , β_2 , and β_3 adrenergic receptors) and norepinephrine binding preferentially α_1 , α_2 , and β_1 adrenergic receptors. Given the different receptor binding affinities of epinephrine and norepinephrine and the differential distribution of adrenergic receptor subtypes on target organs throughout the periphery, epinephrine and norepinephrine generate different effects. Epinephrine, for instance, is particularly effective in increasing heart rate, cardiac output, and blood glucose, while norepinephrine is most effective in increasing blood pressure and energy mobilization by breaking down fats (lipolysis). Although homeostasis depends on the selective activation of various SNS pre- and postganglionic neuron units for specific reflexive homeostatic adjustments, a more global SNS activation, including the hormonal component, is observed in response to psychological stressors.

HPA Axis

The HPA axis (Figure 2) is a neuroendocrine system that controls the secretion of glucocorticoid hormones (Spencer & Deak, 2017). The principal glucocorticoid hormones (CORT) produced in vertebrates are cortisol (e.g., in humans, fish, dogs) and the closely related hormone corticosterone (e.g., in rats, mice, birds, and many reptiles). The HPA axis consists of three populations of cells and the hormonal signals that they use to communicate with each other and the rest of the body.

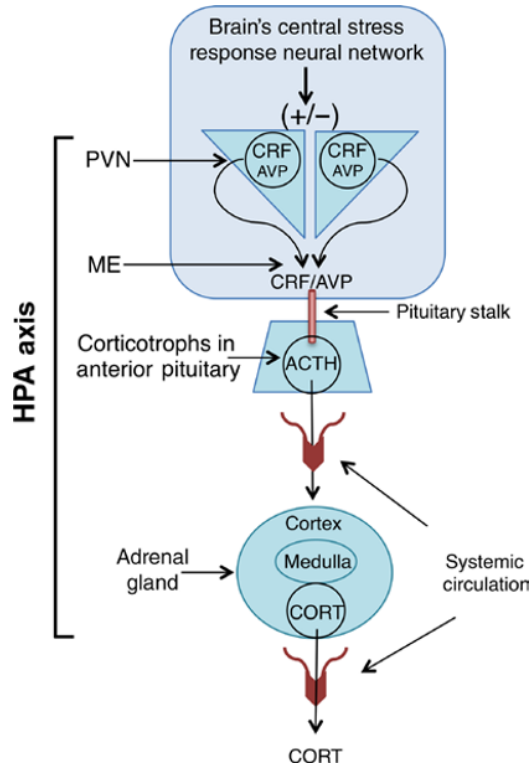


Figure 2 The hypothalamic-pituitary-adrenal axis. Neuron cell bodies located in the paraventricular nucleus (PVN) of the hypothalamus release corticotropin releasing factor (CRF) and arginine vasopressin (AVP) from axon terminals located in the median eminence (ME) at the base of the hypothalamus. CRF and AVP are carried down the portal vein within the pituitary stalk to corticotroph cells located in the anterior pituitary. In response to CRF and AVP, corticotrophs secrete adrenocorticotrophic hormone (ACTH) into the systemic circulation. ACTH stimulates the production of the endogenous glucocorticoid hormones cortisol and corticosterone (CORT) in the adrenal cortex. CORT is released into the systemic circulation and acts as the effector hormone of the HPA axis throughout the body. The brain's central stress response neural network provides excitatory and inhibitory (+/-) neural input to the HPA axis.

The neuronal component of the HPA axis is composed of neurons whose cell bodies are located in the medial parvocellular portion of the paraventricular nucleus (PVN) of the hypothalamus. These neurons project to a region at the base of the hypothalamus (median eminence) that forms the junction between the brain and the pituitary stalk. These neurons are called hypophysiotropic neurons because they project to the interface region between the brain and pituitary (also known as hypophysis). These neurons secrete the neurohormone corticotropin-releasing factor (CRF) (also known as corticotropin-releasing hormone). CRF is carried by the portal vein within the pituitary stalk the short distance (~1 mm) down to the anterior pituitary. The second population of cells consists of endocrine cells, called corticotrophs, located in the anterior pituitary. When these cells are stimulated by CRF, they release adrenocorticotrophic hormone (ACTH) into the general circulation. Some CRF neurons of the PVN also secrete vasopressin, which is co-released with CRF and acts on corticotrophs to enhance ACTH release. The third population of cells consists of endocrine cells located in the outer cortex (fasciculata layer) of the adrenal gland. ACTH causes these adrenal cortical cells

to produce CORT. CORT is the effector hormone of the HPA axis; it mediates the effects of the HPA axis throughout the body. CORT is a steroid molecule, which is lipid soluble, and therefore CORT readily crosses the blood–brain barrier. Consequently, CORT can influence all tissues throughout the body, including the brain. The HPA axis endocrine cells in the anterior pituitary and the adrenal cortex have very little intrinsic activity. As a result, CORT secretion ultimately depends on the activity of the hypothalamic CRF neurons and the net excitatory neural input that these neurons receive from other brain regions. In the absence of stress, CORT exhibits a large daily fluctuation (circadian rhythm) in secretion, with maximal circulating levels present around the habitual time of awakening. Superimposed on this circadian rhythm of CORT secretion is a dynamic increase in response to stress, with peak stress levels of CORT attained about 30 min after stressor onset.

CORT exerts its effects throughout the body by activating two structurally similar receptor proteins, the mineralocorticoid receptor (MR) (which is also the primary receptor for the hormone aldosterone) and the glucocorticoid receptor (GR). These receptors are located inside cells rather than on the outer cell surface. These receptors function as transcription factors in the cell nucleus, and they act either to enhance or repress gene expression by directly binding specific sequences of DNA (glucocorticoid response elements) or indirectly by interacting with other transcription factor proteins (Granner, Wang, & Yamamoto, 2015). There are also some rapid non-genomic effects of CORT, although the molecular mechanisms of these rapid effects have not yet been fully determined. Because the majority of CORT effects on physiological function depend on alteration of gene expression, most CORT effects are slow to develop, with the earliest actions evident about 45 min after an elevation in CORT. However, once initiated, these actions are relatively long lasting (for at least several hours) and therefore can persist, even after CORT levels have returned to low basal levels.

GR is widely expressed in most cell types throughout the body. MR is more restricted in its expression but has high levels in the kidney, cardiac tissue, and hippocampus of the brain. CORT has a 10-fold higher affinity for MR than GR, and therefore low circulating levels of CORT act primarily through MR, whereas higher levels, such as those present during stress, also activate GR. For this reason, GR is believed to be the primary mediator of the stress-related actions of CORT, whereas MR is important for mediating more “permissive” or system maintenance effects of CORT. These permissive actions are important, such that glucocorticoid insufficiency (e.g., Addison’s disease), as well as glucocorticoid excess, has pathological consequences. It is important to note that most synthetic glucocorticoids, which are widely prescribed for various medical conditions, primarily activate only GR.

Stress-induced CORT produces a number of effects that can be adaptive during times of stress. In the periphery this includes increased blood glucose levels, shift in fat depots, modulation of immune system function, and increased cardiovascular tone. In the brain CORT modulates the biochemical aspect of intercellular (neurotransmitter systems) and intracellular signaling function, thereby regulating neuronal activity, plasticity, and morphology. Consequently CORT regulates various aspects of brain function that may be adaptive during times of stress, including learning, memory, reward processes, emotions, sleep, and appetite.

Central Stress Response Neural Network

The neural circuitry that underlies the rapid response to physiological stressors, such as a drop in blood pressure, as described above, has been well characterized. Determining the neural circuitry that underlies the physiological and psychological response to complex psychosocial stressors such as public speaking is not nearly as straightforward. However, considerable

progress has been made in recent years at identifying relevant brain regions and their connections (Ulrich-Lai & Herman, 2009) (Figure 3). The direct neural control of the output neurons of the SNS (thoracolumbar spinal cord preganglionic neurons) and the HPA axis (CRF neurons in the PVN) is limited. The SNS receives direct input (premotor neurons) from descending spinal cord projection neurons whose cell bodies are located primarily in the medulla (e.g., nucleus of the solitary tract, ventral medullary reticular formation, and A5 noradrenergic group) and hypothalamus (non-CRF neurons in the PVN and lateral hypothalamic area). There is, however, also an indirect cortical component provided by some neurons located in the ventromedial prefrontal cortex.

The CRF neurons in the PVN receive direct neural input primarily from the brain stem and a few subcortical forebrain regions. Excitatory brain stem input is provided from adrenergic and noradrenergic afferents originating in the medulla (e.g., the nucleus of the solitary tract and the ventral medulla) and from some serotonergic afferents originating in the raphe nuclei. Both excitatory and inhibitory inputs are provided by other hypothalamic brain regions, the

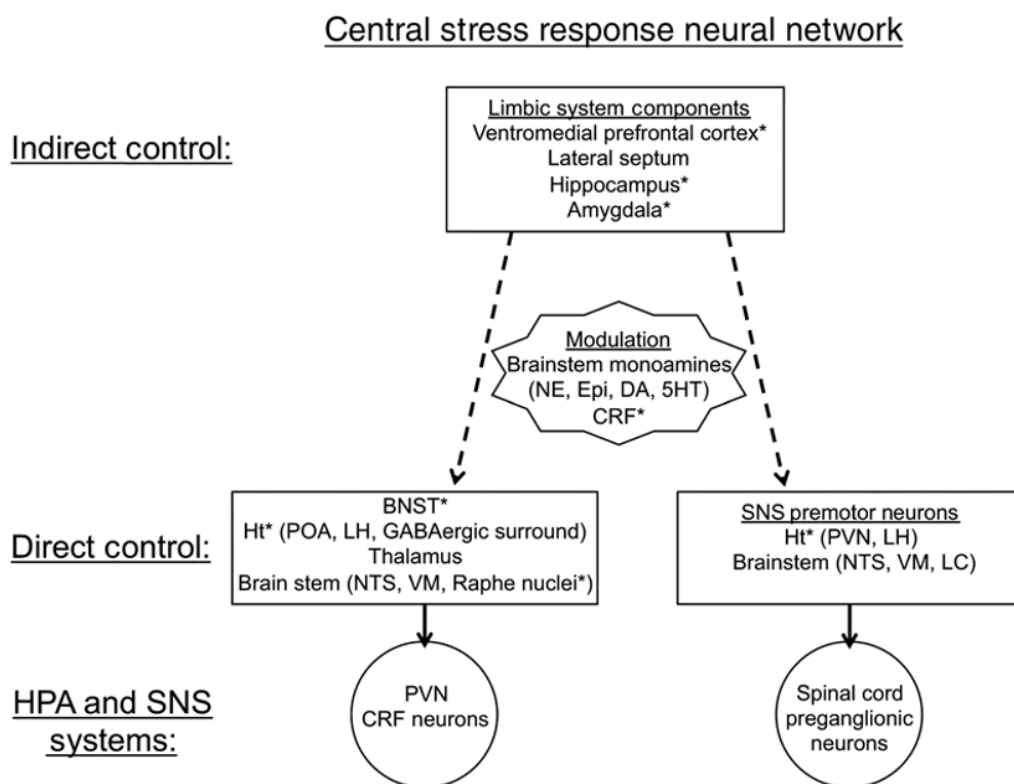


Figure 3 Key neural elements of the central stress response neural network. HPA axis elements (PVN CRFneurons) and SNS elements (spinal cord preganglionic neurons) receive limited direct neural control (primarily from the BNST, hypothalamic subnuclei and brain stem nuclei) and more extensive indirect neural control from limbic system components. Many aspects of the network are modulated by brainstem monoaminergic neurons (NE, norepinephrine; Epi, epinephrine; DA, dopamine; 5HT, serotonin), and by CRF (corticotropin releasing factor) neurons (* indicates brain regions that contain CRF neurons). BNST, bednucleus of the stria terminalis; Ht, hypothalamus; POA, preoptic area; LH, lateral hypothalamus; GABAergic, gamma amino butyric acid releasing neurons; NTS, nucleus of the solitary tract; VM, ventral medullary reticular formation; LC, locus ceruleus.

bed nucleus of the stria terminalis, and the thalamus. CRF neurons in the PVN are also strongly innervated by surrounding GABAergic neurons, and much of the activation and inhibition of CRF neurons may be a result of neural modulation of tonic GABAergic inhibitory tone.

The PVN serves as a unique forebrain integrator of the physiological stress response as it contains both the motor neurons of the HPA axis (CRF hypophysiotropic neurons) and SNS premotor neurons. Other brain regions believed to engage in higher-level stressor processing and stress response coordination are components of the classically described limbic center. These brain regions include the medial prefrontal cortex, lateral septum, hippocampal formation, and amygdala. The bed nucleus of the stria terminalis and subregions of the hypothalamus (e.g., preoptic, dorsomedial, and lateral) are an important interface between these limbic structures and the SNS and HPA axis.

In the context of stress neurobiology, the medial prefrontal cortex is important for situation-specific decision making and control of emotionally motivated behaviors, including the acquisition of conditioned fear extinction learning. The medial prefrontal cortex is also necessary for the detection of stressor controllability and coordination of its protective effects. The hippocampus is especially important for formation and retrieval of contextual memories associated with stressful experiences, whereas the amygdala is critical for forming learned associations between specific environmental cues (e.g., sounds and light patterns) and stressful experiences. The amygdala is also important for generation of negative emotions and coordination of stereotypic defensive behaviors, such as freezing. The lateral septum is important for modulating affective tone, mood, and associated behaviors. The medial prefrontal cortex and the ventral hippocampus also contribute to the timely shutoff of the HPA axis response to stressors.

Strong modulatory regulation of the central stress response neural network is provided by the ascending brain stem monoamine neurotransmitter systems and by the central CRF neuropeptide system (Feldman, Meyer, & Quenzer, 1997). Many noradrenergic neurons (which secrete norepinephrine) are clustered in the brain stem locus coeruleus and project widely throughout the brain. Increased activity in the noradrenergic system contributes to general arousal, and many of these neurons are stress reactive. Adrenergic neurons (which secrete epinephrine) have a more restricted projection to the forebrain but innervate the PVN. Dopaminergic neurons (which secrete dopamine) originating in the ventral tegmental area of the midbrain provide extensive projections to the striatum and medial prefrontal cortex. This mesolimbic dopaminergic system is fundamental for reward processes, but the system is also stress reactive and contributes to emotion modulation. Serotonergic neurons (which secrete serotonin) originating in the raphe nuclei also project widely throughout the forebrain. Subsets of these serotonergic neurons in the dorsal raphe nuclei are also very stress reactive.

In addition to the CRF hypophysiotropic neurons located in the PVN, there are CRF neurons located in strategic portions of the stress response network, such as the hypothalamus, bed nucleus of the stria terminalis, and the amygdala, as well as CRF interneurons located throughout the neocortex and hippocampus. CRF central neurotransmission contributes to SNS and HPA axis activation and regulation of anxiety-related behaviors.

The central stress response network is responsible not just for activation of the SNS and HPA axis but also for generating the psychological stress response. Although there is not a specific stress emotion, there are a number of negative emotional states associated with many stressors, such as fear, anxiety, frustration, and anger. As noted above, the psychological response to stress also includes shifts in attention and decision-making. These shifts are especially dependent on altered operation of the prefrontal cortex. For example, uncontrollable stress increases norepinephrine and dopamine release in the medial prefrontal cortex, leading to an acute reduction in medial prefrontal cortex function. This reduced function contributes

to a shift in behavioral control away from top-down reflective behaviors to more bottom-up reactive behaviors (Arnsten, 2015). Conversely, many of the protective effects of controllable stress depend on medial prefrontal cortex-mediated suppression of stress-induced dorsal raphe nuclei serotonergic activity (Maier, Jose, Baratta, Evan, & Watkins, 2006). Stress also produces robust effects on learning and memory, but the stress–memory relationship is not unidirectional, as stress can interfere with some memory processes but facilitate others. For example, stress can enhance formation of emotional memories while attenuating formation and retrieval of declarative or spatial memories. Stress also shifts reward incentives from a long-term to short-term perspective. In general, all of the orchestrated neural network events associated with stress are directed toward shifting physiological and psychological resources in a manner adaptive for coping with stress.

Regulation of the Stress Response

Stress Response Adaptation

In human experience, all stress responses are superimposed on a background of previous stressor exposure and adaptation. The effects of that adaptation on the expression of subsequent stress responses may be a critical factor in the overall impact of stress on the individual. The deleterious effects of chronic stress on organismal well-being (e.g., disruptions in metabolism, cardiovascular function, inflammation, and depression-like behaviors) largely stem from the cumulative effects of the stress response over time.

A key principle of stress neurobiology that has emerged from studies of stress response adaptation is that repeated exposure of an experimental subject to a particular stressor typically leads to the expression of a diminished hormonal and neural response (habituation) when the subject is then challenged again with the same stressor (homotypic stressor), but often expression of an augmented response (sensitization, also referred to as facilitation) when the subject is subsequently challenged with a different novel stressor (heterotypic stressor) (McCarty, 2016). The rate and degree of stress response habituation generally depends on the type, severity, and duration of the stressor, but the end result is a progressively diminished physiological response to the homotypic stressor. Importantly, this habituation is quite stressor selective, such that even relatively small changes in stressor properties, context, or duration can interfere with the expression of habituation.

A sensitized response to challenge with a novel stressor is evident not only after repeated exposure to a homotypic stressor but also after exposure to chronic variable stress, a procedure in which a subject is exposed to a variety of stressors on an unpredictable schedule. Single-session exposure to some relatively intense acute stressors is also sufficient to produce an exaggerated response to subsequent challenge with a novel stressor. Sensitization of the response to a novel stressor is manifest not only by an exaggerated HPA axis or SNS response but also by increased activation of the dorsal raphe nuclei serotonergic system, potentiated activity in the locus coeruleus, and increased norepinephrine release in the PVN and cortex.

Both impaired stress response habituation and increased stress response sensitization pose increased health risk. Several features of stress response habituation and sensitization described above indicate that the underlying adaptive processes critically occur within central neurocircuits that control physiological stress responses rather than within the anatomical elements of the SNS and HPA axis themselves. First, the stressor selectivity of stress response habituation rules out a generalized decreased responsiveness of the SNS and HPA axis to all stressor input.

Second, the preganglionic neurons of the SNS or the PVN CRH neuron of the HPA axis lack the direct multimodal neural input necessary to discriminate between many stressors. Third, neuronal activity in stress-responsive brain areas shows parallel adaptive changes with the SNS and HPA axis response.

The specific mechanisms that underlie this central stress response neurocircuit adaptation remain to be determined, but recent progress indicates that it is a multidimensional process. Neurocircuit components that may be especially important for the expression of this adaptation are the thalamic paraventricular nucleus, medial prefrontal cortex, and basolateral amygdala. Sensitization may depend on upregulation of generalized stressor responsiveness of serotonergic, noradrenergic, and dopaminergic systems. Although stress response adaptation fundamentally depends on neuroplastic changes within the central stress response neural network, additional adaptation within the HPA axis may fine-tune the expression of HPA axis stress response adaptation.

Sex Differences

Women have a greater prevalence of stress-related mood disorders (e.g., depression and some anxiety-related disorders such as posttraumatic stress disorder) compared with men. This prevalence difference may be due in part to sex differences in the physiological and psychological response to stress. Female rodents display greater stress-induced neuroactivation than males in some brain regions (e.g., bed nucleus of the stria terminalis, medial preoptic hypothalamic area, and the hippocampus) and lesser neuroactivation in others (e.g., amygdala, raphe nuclei, and thalamic nuclei). In the medial prefrontal cortex, female rats exhibit a smaller initial but longer-lasting pattern of neuroactivation than males. In an fMRI study of men and women exposed to negative stimuli, sex differences in blood oxygenation level-dependent signal were evident in a variety of limbic brain regions, and these sex differences varied with female menstrual cycle phase (Goldstein, Jerram, Abbs, & Whitfield-Gabrieli, 2010). There are also some sex differences in neuronal intracellular signal processing in response to acute stress (e.g., in the prefrontal cortex and locus coeruleus) that may contribute to sex differences not only in immediate neural activity but also in neural adaptation to stress.

Female rodents typically exhibit greater stress-induced HPA axis activation compared with males. These HPA axis sex differences depend on both organizational and activational effects of gonadal steroids. Sex differences in HPA axis activity are much less consistently observed in human research. Some studies have found greater stress-induced ACTH and CORT in men compared with women, but other studies have found that while men have greater basal salivary CORT levels, women have equal if not greater stress-induced salivary CORT, suggesting that women overall have a greater increase in stress-induced CORT.

Developmental and Lifespan Factors

Stress occurs throughout the lifespan, and its effects vary depending on the organism's age (Lupien, McEwen, Gunnar, & Heim, 2009). There are important prenatal, childhood, and adolescent critical periods of stress response system development. The extent to which the individual is subject to a stressful environment during these critical periods may program development to be adaptive for a lifetime of excessive stress. For example, in rodent studies, maternal stress results in offspring that as adults exhibit HPA axis hyperreactivity, social withdrawal, and deficits in prefrontal cortex-dependent and hippocampus-dependent learning and memory tasks.

Neonatal stress exposure of rats and mice can also result in increased stress reactivity as an adult. However, the detrimental effects of neonatal stress can be mitigated by good maternal care (e.g., appropriate amount of grooming and licking). Human studies also find long-lasting effects of early life stress on HPA axis function.

During adolescence, rodents and humans show a prolonged HPA axis response to acute stress compared with adults. In the rat the prolonged CORT response is due to increased PVN CRF activity and increased adrenal sensitivity to ACTH. In response to chronic stress, adolescent rats also have higher CORT levels and impaired habituation to a repeated stressor, resulting in a greater likelihood of developing depression-like behavior.

Aged animals also exhibit prolonged CORT release in response to some acute stressors, and this may be due to decreased hippocampal functioning, resulting in decreased inhibitory control of the HPA axis. In regard to chronic stress, it has been found that aged animals do not adapt to a repeated stressor as well as young rats. Similar findings have also been reported in human studies of the elderly.

Mechanisms by Which Stress Impacts Health

Research indicates that the greatest consequence of stress on physical and mental health is not a result of the immediate physiological and psychological impact of an acute stress response, but rather a result of the cumulative and emergent consequence of chronic or repeated stress. In animal studies, some of these effects of chronic stress have been shown to persist for months after chronic stress has ended.

Physiological Health

The long-term alteration of SNS and HPA axis basal and reactive function is a major mediator of the adverse effects of chronic stress on physiological health. Every physiological system has cellular and molecular elements that are directly modulated by catecholamines and glucocorticoids. A shift in the appropriate balance of these modulatory effects directly contributes to systems level pathology. Biomedical research has made tremendous progress in determining many of these specific mechanisms. In addition to the specific regulatory effects that the SNS and HPA axis exert on each physiological system, abnormal catecholamine and glucocorticoid profiles contribute to three processes that can negatively affect all physiological systems. These three processes are increased inflammation, metabolic imbalance, and circadian dysregulation.

Catecholamines lead to increased production of plasma and tissue-specific proinflammatory cytokines (e.g., interleukin-1 β and interleukin-6). Although pharmacological levels of glucocorticoids have potent anti-inflammatory and immunosuppressive effects that are beneficial in treating individuals with autoimmune disorders and tissue transplant recipients, physiological levels of CORT exert a more complex regulatory influence on immune system function (Spencer, Dhabhar, & Kalman, 2001). For example, the permissive effects of CORT prior to stress exposure are necessary for mounting an appropriate proinflammatory response to pathogens, but post-stress glucocorticoids have an important anti-inflammatory influence while promoting certain aspects of acquired immunity. The dysregulation of these immune processes results not only in elevated risk for infection and increased autoimmune expression, but a shift toward chronic tissue and systemic inflammation that negatively impact all physiological systems. This includes the exacerbation of certain inflammatory-related disorders such as rheumatoid arthritis, celiac disease, inflammatory bowel diseases, and asthma.

The chronic activation of the stress response systems is associated with a host of metabolic perturbations including insulin resistance, elevated blood sugar, and adipose tissue redistribution (Bose, Oliván, & Laferrère, 2009). SNS activation acutely promotes negative energy balance, characterized by lipolysis in adipocytes via β -adrenergic receptors, increased triglyceride hydrolysis, free fatty acid mobilization, and brown adipose tissue thermogenesis. Conversely, increased CORT tends to promote positive energy balance in part by antagonizing catecholamine action (e.g., downregulation of β -adrenergic receptor expression), adipogenesis, lipogenesis, and inhibition of brown adipose tissue uncoupling protein-1. Chronic CORT exposure also increases the salience of palatable foods, thereby increasing their consumption that is beneficial for survival in an environment with limited food resources, but contributes to unhealthy diets and metabolic disruption in modern society.

The various depots of adipose tissue are distinguished by their anatomical location and the circulatory system into which they drain. A decreased ratio of subcutaneous white adipose tissue relative to visceral white adipose tissue is a predictor of negative health outcomes associated with obesity. Increased visceral adiposity is associated with chronic stress in many different species including humans. Increased glucocorticoids can promote visceral obesity even when circulating CORT levels appear normal as a result of a stress-induced upregulation within visceral fat of an enzyme (11 β -hydroxysteroid dehydrogenase type 1) that converts inactive glucocorticoids into locally active glucocorticoids (Bose et al., 2009). 11 β HSD-1 knockout mice are protected against high fat diet-induced metabolic disruptions and obesity. Stress-induced disruption of metabolic balance directly contributes to the metabolic syndrome, which is defined as a cluster of conditions that increases risk of cardiovascular disease, stroke, and type 2 diabetes. Metabolic syndrome symptoms include elevated blood sugar, abnormal levels of cholesterol and triglycerides, excess abdominal fat, and high blood pressure.

The optimal function of every physiological system depends on appropriate daily timing of various aspects of its operation (Dickmeis, Weger, & Weger, 2013). These daily fluctuations in operation (circadian rhythms) are maintained and coordinated by a so-called master biological clock located in the suprachiasmatic nucleus of the hypothalamus. CORT is one of the primary intercellular signals used by the suprachiasmatic nucleus to regulate each tissue's unique circadian rhythms. CORT circadian secretion can act as an entrainment factor that contributes to the rhythmic expression of clock genes within many peripheral tissues, and rhythmic clock gene expression then gives rise to tissue-specific circadian operation. Chronic stress can disrupt the body's circadian rhythms not only as a result of disturbed sleep patterns but also by leading to a loss of normal circadian CORT secretion and the resultant misalignment of circadian clocks in tissues throughout the body.

Mental Health

The SNS and HPA axis stress response systems also contribute to the negative effects of chronic stress on mental health. Although the SNS does not have direct effects on brain function, it can indirectly affect the brain through alteration of the activity of visceral sensory afferent nerves, many of which travel with the vagus nerve and project to the brain stem. Chronic elevation of glucocorticoids produces a number of molecular and cellular changes in the brain that are associated with cognitive and emotional disruptions observed in cases of impaired mental health. For example, elevated glucocorticoids lead to dendritic atrophy in the hippocampus and prefrontal cortex while increasing dendritic arborization in the basolateral amygdala. These neuronal morphological changes may enhance amygdala-dependent emotional learning while reducing the appropriate contextualization and situation-specific

modulation of that emotional learning. Chronic elevation of glucocorticoids also changes the functional activity of most neurotransmitter systems by altering levels of neurotransmitter synthetic enzymes (e.g., serotonin tryptophan hydroxylase II and tyrosine hydroxylase), neurotransmitter receptors (e.g., serotonin 2A and NMDA receptors), and degradative enzymes (e.g., monoamine oxidase). However, some of the changes in brain function that emerge with chronic stress are independent of HPA axis and SNS activity. These changes may be directly related to altered activity of components of the central stress response network, as well as additional factors such as altered epigenetic factors and neuroinflammation.

Author Biographies

Prof. R.L. Spencer received a PhD in biopsychology from University of Arizona in 1986 and is currently a professor of neuroscience at the University of Colorado Boulder. His research focuses on the neurobiology of stress response adaptation and circadian regulation of brain function.

Dr. L.E. Chun received a PhD in neuroscience from University of Colorado Boulder in 2017 and is currently a postdoctoral researcher at the University of Colorado Boulder. Her research focuses on sex differences in the influence of stress on circadian neurobiology.

M.J. Hartsock received a MA in neuroscience from University of Colorado Boulder in 2017 and is currently a doctoral student at the University of Colorado Boulder. His research focuses on neural and hormonal mechanisms of circadian regulation of brain function.

Dr. E.R. Woodruff received a PhD in neuroscience from University of Colorado Boulder in 2017 and is currently a postdoctoral researcher at the University of Colorado Boulder. Her research focuses on neurohormonal and circadian regulation of prefrontal cortex-dependent emotional learning.

References

- Arnsten, A. F. T. (2015). Stress weakens prefrontal networks: Molecular insults to higher cognition. *Nature Neuroscience*, 18(10), 1376–1385. doi:10.1038/nn.4087
- Bose, M., Oliván, B., & Laferrère, B. (2009). Stress and obesity: The role of the hypothalamic-pituitary-adrenal axis in metabolic disease. *Current Opinion in Endocrinology, Diabetes, and Obesity*, 16(5), 340–346. doi:10.1097/MED.0b013e32832fa137
- Dickmeis, T., Weger, B. D., & Weger, M. (2013). The circadian clock and glucocorticoids—Interactions across many time scales. *Molecular and Cellular Endocrinology*, 380(1-2), 2–15. doi:10.1016/j.mce.2013.05.012
- Dimsdale, J. E. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology*, 51(13), 1237–1246. doi:10.1016/j.jacc.2007.12.024
- Feldman, R. S., Meyer, J. S., & Quenzer, L. F. (1997). *Principles of neuropsychopharmacology*. Sunderland, MA: Sinauer Associates, Inc.
- Goldstein, J. M., Jerram, M., Abbs, B., Whitfield-Gabrieli, S., & Makris, N. (2010). Sex differences in stress response circuitry activation dependent on female hormonal cycle. *Journal of Neuroscience*, 30(2), 431–438. doi:10.1523/JNEUROSCI.3021-09.2010
- Granner, D. K., Wang, J.-C., & Yamamoto, K. R. (2015). Regulatory actions of glucocorticoid hormones: From organisms to mechanisms. *Advances in Experimental Medicine and Biology*, 872, 3–31. doi:10.1007/978-1-4939-2895-8_1
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews. Neuroscience*, 10(6), 434–445. doi:10.1038/nrn2639

- Maier, S. F., Jose, A., Baratta, M. V., Evan, P., & Watkins, L. R. (2006). Behavioral control, the medial prefrontal cortex, and resilience. *Dialogues in Clinical Neuroscience*, 8(4), 397–406.
- McCarty, R. (2016). Learning about stress: Neural, endocrine and behavioral adaptations. *Stress (Amsterdam, Netherlands)*, 19(5), 1–30. doi:10.1080/10253890.2016.1192120
- Powley, T. L. (2013). Central control of autonomic function. The organization of the autonomic nervous system. In L. R. Squire, D. Berg, F. E. Bloom, S. du Lac, A. Ghosh, & N. C. Spitzer (Eds.), *Fundamental neuroscience* (4th ed.). Waltham, MA: Academic Press.
- Reagan, L. P., Grillo, C. A., & Piroli, G. G. (2008). The As and Ds of stress: Metabolic, morphological and behavioral consequences. *European Journal of Pharmacology*, 585(1), 64–75. doi:10.1016/j.ejphar.2008.02.050
- Spencer, R. L., & Deak, T. (2017). A users guide to HPA axis research. *Physiology and Behavior*, 178, 43–65. doi:10.1016/j.physbeh.2016.11.014
- Spencer, R. L., Dhabhar, F. S., & Kalman, B. A. (2001). Role of endogenous glucocorticoids in immune system function: Regulation and counterregulation. In B. S. McEwen & H. Maurice Goodman (Eds.), *Handbook of physiology*. Section 7 (Vol. 4, pp. 381–423). New York, NY: Oxford University Press.
- Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews. Neuroscience*, 10(6), 397–409.

Stress, Neurogenesis, and Mood: An Introduction to the Neurogenic Hypothesis of Depression

Jacob Huffman^{1,2} and George T. Taylor^{1,3}

¹*Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA*

²*Department of Psychiatry, Washington University in St. Louis School of Medicine, St. Louis, MO, USA*

³*Interfakultäre Biomedizinische Forschungseinrichtung (IBF) der Universität Heidelberg, Heidelberg, Germany*

Introduction

The prevalence of depression in the United States has steadily increased over the years with 6.7% of adults and 11.4% of adolescents diagnosed with major depressive disorder (MDD) in 2014 (NIMH, 2015). MDD is a complex clinical condition resulting from diverse neurobiological anomalies (Fried, Nesse, Zivin, Guille, & Sen, 2014; Milaneschi et al., 2016). A recent neuropathogenic model of MDD highlights the stress response and reduced neurogenesis in the dentate gyrus (DG) as essential underpinning components to the onset and progression of depressive symptoms. This model is referred to as the neurogenic hypothesis of depression (NHD; Jacobs, Van Praag, & Gage, 2000; Snyder, Soumier, Brewer, Pickel, & Cameron, 2011).

Functional Significance of the Dentate Gyrus

The DG is composed of molecular, granule, and polymorphic layers. Of primary interest to the NHD models is the granule layer, which is composed of a large number of tightly grouped cells. Granule cells have unique properties contributing to neurogenesis and exhibit generative

capacity as a function of progenitor cells that emerge from the subgranular zone. Interestingly, granule cell counts are static in healthy adult animals (Rapp & Gallagher, 1996), suggesting that ongoing migration of these cells from the subgranular zone is required to maintain a homeostatic level (Lyons et al., 2010). Newly derived granule cells mature and establish synaptic connectivity with adjacent cells (Amaral, Scharfman, & Lavenex, 2007). These cells are excitatory in nature and are densely populated in efferent/afferent regions of the entorhinal cortex and hippocampus. Not surprisingly, altered hippocampal neurogenesis is correlated with impaired performance on memory tasks in animal models of depression (Jessberger et al., 2009; Parihar, Hattiangady, Kuruba, Shuai, & Shetty, 2011).

Stress Response and Neurogenesis

Elevated cortisol (CORT) suppresses neurogenesis in the DG, likely due to the stimulation of NMDA receptors (Anacker et al., 2013). Granule cells and progenitor cells are sensitive to glucocorticoid levels (Alahmed & Herbert, 2008). Elevated levels of CORT inhibit the differentiation of progenitor cells into mature granule cells (Brummelte & Galea, 2010; Wong & Herbert, 2006). Chronic stress reduces neurogenesis and the total number of mature granule cells. Behaviorally, individuals with chronic stress exhibit high levels of CORT, reduced neurogenesis, and impaired memory performance (Montaron et al., 2006). Interestingly, neurogenic processes decline with normal aging (Cowen, Takase, Fornal, & Jacobs, 2008), which may contribute to memory impairments and sensitization to stressful stimuli in older individuals.

BDNF Versus Glucocorticoids

BDNF is a one of four growth factors in the neurotrophin family, including nerve growth factor, neurotrophin-3, and neurotrophin-4. Growth factors facilitate cellular maturation and differentiation. BDNF expression increases long-term potentiation, a key component to sustained cell signaling and adequate memory function (Gartner, 2006). BDNF also assists cellular activation presynaptically by modifying incoming signals from upstream neurons (Kuczewski et al., 2008). Of particular importance to the current chapter, BDNF regulates NMDA receptor activation postsynaptically (Caldeira et al., 2007) and is essential for preventing excess signaling from driving excitotoxic degradation of mature granule cells.

Reductions in BDNF disrupt NMDA activation (Xu et al., 2000). The level of signal modulation may be an important facet of neurogenic processes and helps explain why granule cells are particularly vulnerable to excitotoxicity. Indeed, injections of BDNF directly into the hippocampus enhances neurogenesis and dendritic branching (Kramár et al., 2012; Scharfman et al., 2005). Snyder et al. (2011) showed that neurogenesis helps to buffer the effects of stress by controlling CORT production through mature granule cells in the DG. The implications are the hippocampus and HPA axis are dependent on neurogenesis to adapt effectively in the context of chronic stress. Observable reductions in BDNF may be the first sign of HPA dysregulation typical of stress-induced depressive behaviors.

Acute and Chronic BDNF Activation

The mechanisms driving different changes in BDNF expression under acute and chronic stress are not well understood. In a study by Murakami, Imbe, Morikawa, Kubo, and Senba (2005),

BDNF expression was reduced considerably more with acute compared with chronic stress. CORT levels continued to rise during sustained stress, whereas BDNF levels remained steady. These data, coupled with findings from Snyder et al. (2011), indicate that stress-induced reductions in neurogenesis follow initial downregulation of BDNF.

BDNF and Antidepressant Efficacy

Fluoxetine enhances the differentiation of progenitor cells and prevents cell death in the DG (Lee et al., 2001). Another antidepressant, tianeptine, reduces apoptosis in the DG (Lucassen, Fuchs, & Czéh, 2004). Chronic administration of venlafaxine, mirtazapine, and fluoxetine reduces depressive-like behaviors and increases BDNF levels (Zhang, Gu, Chen, & Dong, 2010). Of importance, only venlafaxine and mirtazapine reduced serum levels of CORT. The observation that all drugs reduced depressive-like behaviors and increased BDNF levels, but did not decrease CORT, suggests BDNF plays a primary role in the success of antidepressant treatments.

Fluoxetine displays unique properties relevant to the NHD model. Specifically, fluoxetine enhances neurogenesis, improves depressive-like behaviors, and helps to maintain drug efficacy without extensive influence on CORT secretion (Lee et al., 2001; Zhang et al., 2010). Evidence also suggests that chronic CORT administration induces hyperactivity, anxiety, and depressive-like behaviors similar to that seen with chronic stress, which can be reversed following fluoxetine administration (David et al., 2009). These findings were reduced to fluoxetine's ability to reverse CORT-induced disruptions in cellular proliferation, rather than progenitor cell differentiation or maturation. The implication is that fluoxetine modulates depressive-like behaviors through the indirect downregulation of CORT secretion via modified cellular processes derived from BDNF.

Indeed, the well-substantiated link between stress and CORT, per the NHD model, has led to the contention that the effects of BDNF on depressive-like behavior acts to indirectly aid the mechanisms of antidepressant medication, rather directly superseding the effects of CORT. Evidence suggests that lower levels of BDNF in the DG are not sufficient to produce depressive-like behavior (Adachi, Barrot, Autry, Theobald, & Monteggia, 2008; Larsen, Mikkelsen, Hay-Schmidt, & Sandi, 2010). However, genetic depletion of BDNF expression hinders the efficacy of antidepressant medication (Ibarguen-Vargas et al., 2009). Postmortem analysis has shown that BDNF is higher in the DG of patients who were using antidepressant medication at the time of death (Chen, Dowlathahi, Macqueen, Wang, & Young, 2001).

Summary

According to the NHD, reduced neurogenesis in the DG disrupts the HPA axis with downstream effects on CORT secretion and BDNF expression. With chronic stress, continuously elevated CORT decreases BDNF and suppresses the differentiation of progenitor cells into mature granule cells. Furthermore, excess CORT inhibits the migration of progenitor cells, disrupting homeostatic regulation of mature granule cell homeostasis. Chronic stress creates a feedforward cycle that restricts recovery. Here, we contend that BDNF is an initial alteration in a sequence of physiological responses to stress in the DG (rather than CORT). This is likely consequent to the failure of proper stress adaptation, producing chronic hyperactivation of the entorhinal cortex, excessive stimulation of granule cells, and abnormal HPA negative feedback.

Author Biographies

Jacob Huffman is currently working on a PhD at the University of Missouri–St. Louis. His research focuses on the steroidal and immunological contributions of acute/chronic stress and ischemic stroke on models of depression-like behaviors. He is a certified surgeon for the micro-arterial stroke model, middle cerebral artery occlusion, which mimics a focal cerebral ischemic event in humans. He also collaborates with Washington University Medical School on steroidal, anesthetic, and viral models of prenatal and postnatal neurotoxicity.

George T. Taylor is a professor of psychology and director of the behavioral neuroscience graduate program. He received his PhD from the University of New Mexico in animal psychology, and postdoctoral work was conducted at Rockefeller University in New York City in neurophysiology. He also has held appointments as a guest scientist at Bordeaux (France) Medical University in neuroendocrinology, at the German Cancer Research Center–Heidelberg in experimental pathology, and at Tokyo University in biology. Research in his laboratory employs animal models for the study of neuroendocrinology–psychopathology relations. Ongoing projects include neurosteroidal influences on cognition and learning and memory behaviors, steroid receptor functions in the hippocampus, neurobiology of acute/chronic stress and depression-like behaviors, and hormones as neuroprotective agents in stroke. His lab collaborates with colleagues at Heidelberg University Medical School, Washington University Medical School, and University of Missouri–Columbia Veterinary School, allowing his graduate students to gain further research experiences in biomedical settings.

References

- Adachi, M., Barrot, M., Autry, A. E., Theobald, D., & Monteggia, L. M. (2008). Selective loss of brain-derived neurotrophic factor in the dentate gyrus attenuates antidepressant efficacy. *Biological Psychiatry*, 63(7), 642–649.
- Alahmed, S., & Herbert, J. (2008). Strain differences in proliferation of progenitor cells in the dentate gyrus of the adult rat and the response to fluoxetine are dependent on corticosterone. *Neuroscience*, 157(3), 677–682.
- Amaral, D. G., Scharfman, H. E., & Lavenex, P. (2007). The dentate gyrus: Fundamental neuroanatomical organization (dentate gyrus for dummies). *Progress in Brain Research*, 3–790.
- Anacker, C., Cattaneo, A., Luoni, A., Musaelyan, K., Zunszain, P. A., Milanese, E., ... Price, J. (2013). Glucocorticoid-related molecular signaling pathways regulating hippocampal neurogenesis. *Neuropsychopharmacology*, 38(5), 872–883.
- Brummelte, S., & Galea, L. (2010). Chronic high corticosterone reduces neurogenesis in the dentate gyrus of adult male and female rats. *Neuroscience*, 168(3), 680–690.
- Caldeira, M. V., Melo, C. V., Pereira, D. B., Carvalho, R. F., Carvalho, A. L., & Duarte, C. B. (2007). BDNF regulates the expression and traffic of NMDA receptors in cultured hippocampal neurons. *Molecular and Cellular Neuroscience*, 35(2), 208–219.
- Chen, B., Dowlatshahi, D., Macqueen, G. M., Wang, J., & Young, L. (2001). Increased hippocampal BDNF immunoreactivity in subjects treated with antidepressant medication. *Biological Psychiatry*, 50(4), 260–265.
- Cowen, D. S., Takase, L. F., Fornal, C. A., & Jacobs, B. L. (2008). Age-dependent decline in hippocampal neurogenesis is not altered by chronic treatment with fluoxetine. *Brain Research*, 1228, 14–19.
- David, D. J., Samuels, B. A., Rainer, Q., Wang, J. W., Marsteller, D., Mendez, I., ... Artymyshyn, R. P. (2009). Neurogenesis-dependent and-independent effects of fluoxetine in an animal model of anxiety/depression. *Neuron*, 62(4), 479–493.

- Fried, E. I., Nesse, R. M., Zivin, K., Guille, C., & Sen, S. (2014). Depression is more than the sum score of its parts: individual DSM symptoms have different risk factors. *Psychological Medicine*, 44(10), 2067–2076.
- Gartner, A. (2006). Hippocampal long-term potentiation is supported by presynaptic and postsynaptic tyrosine receptor kinase B-mediated phospholipase C signaling. *Journal of Neuroscience*, 26(13), 3496–3504.
- Ibarguen-Vargas, Y., Surget, A., Vourc'h, P., Leman, S., Andres, C. R., Gardier, A. M., & Belzung, C. (2009). Deficit in BDNF does not increase vulnerability to stress but dampens antidepressant-like effects in the unpredictable chronic mild stress. *Behavioural Brain Research*, 202(2), 245–251.
- Jacobs, B. L., Van Praag, H., & Gage, F. H. (2000). Adult brain neurogenesis and psychiatry: A novel theory of depression. *Molecular Psychiatry*, 5(3), 262.
- Jessberger, S., Clark, R. E., Broadbent, N. J., Clemenson, G. D., Consiglio, A., Lie, D. C., ... Gage, F. H. (2009). Dentate gyrus-specific knockdown of adult neurogenesis impairs spatial and object recognition memory in adult rats. *Learning & Memory*, 16(2), 147–154.
- Kramár, E. A., Chen, L. Y., Lauterborn, J. C., Simmons, D. A., Gall, C. M., & Lynch, G. (2012). BDNF upregulation rescues synaptic plasticity in middle-aged ovariectomized rats. *Neurobiology of Aging*, 33(4), 708–719.
- Kuczewski, N., Porcher, C., Ferrand, N., Fiorentino, H., Pellegrino, C., Kolarow, R., ... Gaiarsa, J. (2008). Backpropagating action potentials trigger dendritic release of BDNF during spontaneous network activity. *Journal of Neuroscience*, 28(27), 7013–7023.
- Larsen, M. H., Mikkelsen, J. D., Hay-Schmidt, A., & Sandi, C. (2010). Regulation of brain-derived neurotrophic factor (BDNF) in the chronic unpredictable stress rat model and the effects of chronic antidepressant treatment. *Journal of Psychiatric Research*, 44(13), 808–816.
- Lee, H. J., Kim, J. W., Yim, S. V., Kim, M. J., Kim, S. A., Kim, Y. J., ... Chung, J. H. (2001). Fluoxetine enhances cell proliferation and prevents apoptosis in dentate gyrus of maternally separated rats. *Molecular Psychiatry*, 6(6), 725–728.
- Lucassen, P. J., Fuchs, E., & Czeh, B. (2004). Antidepressant treatment with tianeptine reduces apoptosis in the hippocampal dentate gyrus and temporal cortex. *Biological Psychiatry*, 55(8), 789–796.
- Lyons, D. M., Buckmaster, P. S., Lee, A. G., Wu, C., Mitra, R., Duffey, L. M., ... Schatzberg, A. F. (2010). Stress coping stimulates hippocampal neurogenesis in adult monkeys. *Proceedings of the National Academy of Sciences*, 107(33), 14823–14827.
- Milaneschi, Y., Lamers, F., Peyrot, W. J., Abdellaoui, A., Willemsen, G., Hottenga, J. J., ... Boomsma, D. I. (2016). Polygenic dissection of major depression clinical heterogeneity. *Molecular Psychiatry*, 21(4), 516–522.
- Montaron, M., Drapeau, E., Dupret, D., Kitchener, P., Aurousseau, C., Moal, M. L., ... Abrous, D. (2006). Lifelong corticosterone level determines age-related decline in neurogenesis and memory. *Neurobiology of Aging*, 27(4), 645–654.
- Murakami, S., Imbe, H., Morikawa, Y., Kubo, C., & Senba, E. (2005). Chronic stress, as well as acute stress, reduces BDNF mRNA expression in the rat hippocampus but less robustly. *Neuroscience Research*, 53(2), 129–139.
- NIMH. (2015). *Major depression among adults*. Retrieved from <http://www.nimh.nih.gov/health/statistics/prevalence/major-depression-among-adults.shtml>.
- Parihar, V. K., Hattiangady, B., Kuruba, R., Shuai, B., & Shetty, A. K. (2011). Predictable chronic mild stress improves mood, hippocampal neurogenesis and memory. *Molecular Psychiatry*, 16(2), 171–183.
- Rapp, P. R., & Gallagher, M. (1996). Preserved neuron number in the hippocampus of aged rats with spatial learning deficits. *Proceedings of the National Academy of Sciences*, 93(18), 9926–9930.
- Scharfman, H., Goodman, J., Macleod, A., Phani, S., Antonelli, C., & Croll, S. (2005). Increased neurogenesis and the ectopic granule cells after intrahippocampal BDNF infusion in adult rats. *Experimental Neurology*, 192(2), 348–356.
- Snyder, J. S., Soumier, A., Brewer, M., Pickel, J., & Cameron, H. A. (2011). Adult hippocampal neurogenesis buffers stress responses and depressive behaviour. *Nature*, 476(7361), 458–461.

- Wong, E., & Herbert, J. (2006). Raised circulating corticosterone inhibits neuronal differentiation of progenitor cells in the adult hippocampus. *Neuroscience*, 137(1), 83–92.
- Xu, B., Gottschalk, W., Chow, A., Wilson, R. I., Schnell, E., Zang, K., ... Reichardt, L. F. (2000). The role of brain-derived neurotrophic factor receptors in the mature hippocampus: Modulation of long-term potentiation through a presynaptic mechanism involving TrkB. *The Journal of Neuroscience*, 20(18), 6888–6897.
- Zhang, Y., Gu, F., Chen, J., & Dong, W. (2010). Chronic antidepressant administration alleviates frontal and hippocampal BDNF deficits in CUMS rat. *Brain Research*, 1366, 141–148.

An Overview of Neuropsychiatric Symptoms and Pathogenesis in MS

Emily Evans¹, Elizabeth Silbermann^{1,2}, and Soe Mar³

¹*Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA*

²*Department of Neurology, Oregon Health and Science University, Portland, OR, USA*

³*Neurology and Pediatrics, Division of Pediatric Neurology, Washington University in St. Louis, St. Louis, MO, USA*

Pathophysiology of MS

Multiple sclerosis (MS) is a chronic inflammatory disorder affecting the central nervous system. MS affects almost 2.3 million people worldwide and approximately 250,000–400,000 people in the United States. MS primarily affects the white matter with pathology characterized by the inflammatory plaque. Inflammatory plaques exist throughout the brain, spinal cord, and optic nerves with their locations corresponding to a myriad of clinical symptoms including weakness, numbness, bowel and bladder symptoms, vision loss, or other neuropsychiatric symptoms.

Inflammation in MS occurs in several phases and manifests with considerable variability. Our initial understanding of the pathophysiology of this disease focused on the inflammatory plaque. Inflammatory plaques are often characterized by their temporal pattern and their degree of inflammation. Early inflammatory plaques feature monocytes and lymphocytes with T-cell predominant perivascular cuffing and demyelination. Early on, the blood–brain barrier remains intact with limited inflammation. Between six and twenty weeks of disease progression, lesions begin to show inflammatory infiltrates. Chronic plaques, on the other hand, feature hypo-cellularity and glial scarring with varying degrees of active inflammation present at the plaque margin (Wu & Alvarez, 2011).

The robust presence of T cells in inflammatory lesions prompted investigation into their involvement in neuronal destruction. Several studies in animal models of MS have demonstrated that autoreactive CD4 cells and to a lesser extent CD8 cells lead to a phenotype similar to MS. There are ongoing investigations into the role of a particular lineage of CD4 T cells, TH17 cells, which secrete IL17, a proinflammatory cytokine. In experimental autoimmune

encephalomyelitis (EAE), a commonly utilized animal model of MS, TH17 cells, participate in the robust inflammatory response seen in early CNS lesions (Ontaneda, Hyland, & Cohen, 2012). Further, human studies have shown that both the peripheral blood and cerebral spinal fluid (CSF) of patients with MS contain a greater proportion of TH17 cells than do controls. TH17 cells are found in perivascular infiltrates that further support their role in allowing penetration of the blood–brain barrier. B cells are also involved in CNS damage. While acute plaques feature robust active inflammation, chronic plaques often feature complement deposition and plasma cells (Weissert, 2013). This involvement is not entirely surprising; the presence of oligoclonal bands in the CSF of patients is highly suggestive of, although not specific for, a diagnosis of MS. In addition, B-cell follicles have been reported in the meninges of patients with secondary progressive MS and have also been reported underlying pathology in the corresponding cortical gray matter. The exact target of these clonal B cells and their antibodies remains unclear but is an important area of ongoing research.

While MS was initially defined by abnormalities noted in plaques, there is now substantial evidence that pathology exists in normal-appearing white matter (NAWM) and dirty-appearing white matter (DAWM) (Moore et al., 2008). These extra-lesional areas feature perivascular and parenchymal inflammation and have disruption of the blood–brain barrier that promotes proinflammatory cascades, fibrinogen deposition, and resultant demyelination. Similarly, DAWM also features demyelination and axonal loss. While the implications of NAWM and DAWM remain unclear, it does provide further evidence that MS exists as a more global inflammatory process as opposed to a clearly focal process.

In addition to white matter disease, postmortem studies have provided robust evidence that MS also involves gray matter structures (Gilmore et al., 2009). The cortex is frequently disrupted in MS with cortical pathology demonstrating demyelination, inflammation, and cell death. Similar to white matter lesions, this damage occurs both within discrete lesions as well as in normal-appearing gray matter. The functioning of areas such as the prefrontal cortex, cingulate cortex, and hippocampus is preferentially affected, which explains the dysfunction in domains of executive function, learning, information processing, and memory displayed by MS patients. Finally, disruption of deep gray matter structures, including the thalamus and basal ganglia, is also seen. Atrophy in both the cortex and deeper gray matter regions leads to more global cognitive dysfunction that ultimately contributes to significant morbidity and potentially mortality in MS patients.

Cognitive Impairment in MS

Cognitive impairment is a common complaint among MS patients and leads to considerable disability. The true prevalence of cognitive impairment varies by study; however, it is generally accepted that between 40 and 65% of MS patients experience it (Bobholz & Rao, 2003). Cognitive changes in MS are not clearly tied to disease duration or physical disability. In fact, cognitive changes can occur very early in the disease course and can even precede the diagnosis. Cognitive issues can worsen acutely during a relapse but persist even between relapses and often progress over time. Cognitive impairment seems to be more profound in the progressive subtypes of MS than in the relapsing subtypes.

The cognitive impairment in MS is not global, but rather preferentially affects individual cognitive domains. For example, processing speed is commonly slow in patients with MS. Processing speed involves one's ability to take in new information, manipulate it, and apply it to his environment. Attentional domains are also very commonly affected, especially sustained and

shifting attention. When tested, patients with MS score poorly on tasks requiring them to divide their attention and attend to multiple tasks simultaneously. Other very commonly affected domains include visual memory, executive functioning, visual perception, and long-term memory. Visual perception is often confounded by poor visual acuity as many patients with MS have suffered optic neuritis and have residual deficits in visual acuity. However visual perception deficits also occur independently of visual acuity loss. With regard to long-term memory, the problem with memory is usually one of encoding memories and reflective of the poor attention of said patients. Conversely domains like language processing and naming are usually spared.

Cognitive impairment has real consequences for patients with MS and results in decreased productivity at work, high rates of unemployment, disrupted social relationships, and less ability to live independently. Cognitive deficits correlate with objective difficulties completing instrumental activities of daily living, such as managing one's finances and medications, and with worsened scores on subjective quality of life (QOL) measures as reported from a patient's perspective (Cutajar et al., 2000). QOL can be quantified using such tests as the Multiple Sclerosis Quality of Life Inventory or SF36 (part of health status questionnaire). Cognitive deficits can also interfere with the patient–physician therapeutic alliance or patient adherence to prescribed pharmacological or non-pharmacological treatment regimens.

Cognitive impairment can be measured in various ways. Traditional clinical measures of screening cognition such as the Mini-Mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA) are insensitive to the specific deficits affected in MS. If these are the only screens used to assess MS patients, their deficits and complaints can go unrecognized and underappreciated for years.

The gold standard for diagnosing cognitive impairment in MS patients is formal neuropsychiatric testing, which involves exposing patients to a battery of tests assessing multiple domains of cognition. However, such testing is time consuming, costly, and not available in all regions. A recent focus in the field has been on developing more clinic-friendly cognitive screening assessments. Several validated screening assessments are now available, and there has been considerable debate regarding which are the “best” screens. Each varies in regard to sensitivities and specificities depending on the specific domain of cognition one is interested in analyzing. Two of the most commonly utilized assessments include the Minimal Assessment of Cognitive Function in MS (MACFIMS) and Brief International Cognitive Assessment for MS (BICAMS). The MACFIMS is a ninety-minute battery, consisting of seven tests that was created by the consortium of MS centers that has been validated both in MS patients and in healthy controls (Benedict et al., 2006). The BICAMS is a fifteen-minute test created by an international expert consensus committee and includes the symbol digit modalities test (SDMT), California Verbal Learning Test second edition (CVLT-II), and revised Brief Visuospatial Memory Test (BVM-T-R) (Langdon et al., 2012). The BICAMS is designed to be administered by any healthcare practitioner and would not require formal neuropsychiatric training. It is currently undergoing validation studies in multiple countries.

One recent focus in the field has been on developing a computerized or Internet-based screening test to ameliorate some of the cost and availability issues of our current screening methods. Several such screens have been developed and validated, such as the thirty-minute Cognitive Stability Index (Younes et al., 2007), but none have yet been widely incorporated into clinical practice.

Pathophysiologically, it is not known what the exact cause of the cognitive dysfunction in MS is. Likely it is a combination of both gray matter and white matter damage (as detailed earlier in the pathophysiology of MS section) and the resultant atrophy associated with these inflammatory changes in the brain.

The cognitive changes in MS do correlate with quantifiable radiographic changes such as MRI patterns of cerebral atrophy. Both gray and white matter atrophy have been implicated. A recent review article published in *Lancet* (Rocca et al., 2015) compiled a table of more than thirty recently published studies reporting associations between cognitive function and radiographic measures of either white matter lesions, gray matter lesions, or total brain atrophy. For example, associations have been demonstrated between cognitive impairment and total lesion areas, T1 lesion volumes, T2 lesion volumes, FLAIR lesion volumes, the size of the corpus callosum, and the third ventricular width (Rocca et al., 2015). Moreover, cortical lesions burden has been associated with cognitive disability in MS. No one measure has been shown to fully explain or predict cognitive performance, and research remains ongoing in the field.

Several agents including disease-modifying therapies aimed at tempering the inflammatory process in the CNS have been shown to decrease acute clinical relapses and decrease radiographic lesion burden among patients with MS. It is possible that these therapies slow the rate of progressive cognitive impairment; however no studies yet exist that clearly demonstrate this. No pharmacological therapies have yet been proven to reverse cognitive impairment among MS patients. Nonetheless several agents are used for symptomatic treatment of cognitive impairment in MS. These include stimulants (such as methylphenidate, amphetamine, and modafinil), cholinesterase inhibitors (such as donepezil, galantamine, and rivastigmine), and *N*-methyl-D-aspartate (NMDA) receptor antagonists (such as memantine). A recent Cochrane review of the literature concluded that there is no convincing evidence that any of these agents were effective (He, Zhou, Guo, Hao, & Wu, 2011). Further investigations into pharmacotherapy to treat MS-related cognitive impairment are warranted.

Cognitive rehabilitation is a non-pharmacological form of treatment being increasingly explored to combat MS-related cognitive impairment. Cognitive rehabilitation is an individualized treatment regimen, which combines restorative techniques with compensatory activities. Restorative techniques are designed to help restore normal brain functioning to a previously damaged area and focuses on repeated exposures and practice to harness the brain's natural repair mechanisms and plasticity. Conversely compensatory techniques focus on finding ways around the cognitive difficulties a patient has by utilizing what remaining domains a patient has. For example, behavioral interventions like employing reminders such as personal organizers, calendars, or alarms can be useful for patients with attentional or working memory difficulties. Studies have showed that cognitive rehabilitation strategies can both improve a patient's quality of life and lead to improved performance on memory tasks.

Depression and Fatigue

Both mood disorders and fatigue are very prevalent in MS patients with estimates that up to sixty and ninety percent of patients suffer from these conditions, respectively. Depression and other emotional disorders are associated with reduced functioning, reduced adherence to medical therapies, and overall decreased quality of life. The lifetime prevalence of major depression in patients with MS is elevated and estimated at 46–54% versus 16.2% in the general public (Minden et al., 2014). In addition, MS patients are more likely to have clinical diagnoses of anxiety, and suicide is twice as common in the MS population relative to controls. Recent studies suggest a relationship between depression and demyelination, particularly when demyelination involves the limbic system. Functional MRI studies have shown differences in activation between the prefrontal cortex and the amygdala in patients with concurrent MS and depression. Similar studies have also shown patients with concurrent MS and affective disorders had overall higher global disease burdens suggesting a link between structural damage and comorbid psychiatric disease.

Fatigue is also highly prevalent in MS patients. Patients complain of both cognitive and global fatigue. Researchers have demonstrated an association between cognitive fatigue and slower information processing (Andreasen, Spliid, Andersen, & Jakobsen, 2010). The impact of global fatigue, however, has been more difficult to quantify. Radiographic studies have correlated global fatigue with preferential frontoparietal cortical atrophy and disruption of frontal and parietal pathways (Sepulcre et al., 2009). Recent studies have utilized fMRI scanning to explore MS subjects undergoing cognitive evaluation of working memory and attention via Paced Auditory Serial Addition Test (PASAT) or the Paced Visual Serial Addition Test (PVSAT). Subjects with MS have demonstrated more sustained and widespread cortical activation despite worse task performance. It was hypothesized that this may contribute to “neural fatigue.” The interplay between fatigue and cognitive dysfunction requires further investigation.

Screening for fatigue and affective disorders like depression is very important, because when untreated, such conditions lead to disability. Independent evaluation of these disorders is challenging as fatigue, depression, and cognitive impairment are intricately linked. A few screening tools are currently available. Fatigue can be assessed using validated scales such as the Modified Fatigue Impact Scale (MFIS), Fatigue Severity Scale (FSS), or the Chalder Fatigue Scale. Depression can be assessed using the Beck Depression Inventory. In addition, the General Health Questionnaire can be useful in evaluating more globally for difficulty in regulating emotions. Finally, the Center for Neurologic Study Emotional Lability Scale can be useful in detecting pseudobulbar affect, which can be another important consideration in evaluating depression in MS patients. Clinicians must carefully consider the confounding impact of other related factors, such as insomnia, restless legs syndrome, and circadian rhythm abnormalities, all of which are experienced by MS patients and can influence both fatigue and depression.

Author Biographies

Emily Evans, MD, completed her undergraduate studies at Tulane University and earned a medical degree from the Perelman School of Medicine at the University of Pennsylvania. She completed her residency training in neurology at Washington University in Saint Louis. She is currently a clinical fellow in neuroimmunology at the John L. Trotter MS Center associated with Washington University in Saint Louis where she is specializing in the clinical care of patients with MS and the conduct of clinical trials related to MS.

Elizabeth Silbermann, MD, completed her undergraduate studies at Brown University and earned a medical degree at Warren Alpert Medical School, Providence, RI. She completed a residency in neurology at Washington University School of Medicine in Saint Louis, where she distinguished herself as a neurology chief resident. She is currently a neuroimmunology fellow at Oregon Health and Science University. She is the recipient of a National MS Society sponsored Sylvia Lawry Physician Fellowship grant and is currently involved in Phase I, Phase II, and observational trials of drug and rehabilitation interventions.

Soe Mar, MD, completed her undergraduate and medical school training in Myanmar. She completed her residency and fellowship training at the Albert Einstein College of Medicine in New York, NY. She is the program director of the Pediatric Neurology Residency Training Program at Saint Louis Children’s Hospital (SLCH) associated with Washington University in Saint Louis. She is also the director of the Pediatric Multiple Sclerosis & Demyelinating Disease Center at SLCH, which is one of twelve nationwide National MS Society (NMMS) recognized pediatric MS centers. She has been the recipient of numerous distinguished teaching awards and published more than seventy papers relating not only to MS but also to leukodystrophies, headache disorders, and CNS lymphomas to name a few.

References

- Andreasen, A. K., Spliid, P. E., Andersen, H., & Jakobsen, J. (2010). Fatigue and processing speed are related in multiple sclerosis. *European Journal of Neurology*, 17(2), 212–218. doi:10.1111/j.1468-1331.2009.02776.x
- Benedict, R. H. B., Cookfair, D., Gavett, R., Gunther, M., Munschauer, F., Garg, N., & Weinstock-Guttman, B. (2006). Validity of the Minimal Assessment of Cognitive Function in Multiple Sclerosis (MACFIMS). *Journal of the International Neuropsychological Society: JINS*, 12(4), 549–558. doi:10.1017/S1355617706060723
- Bobholz, J. A., & Rao, S. M. (2003). Cognitive dysfunction in multiple sclerosis: A review of recent developments. *Current Opinion in Neurology*, 16(3), 283–288. doi:10.1097/01.wco.0000073928.19076.84
- Cutajar, R., Ferriani, E., Scandellari, C., Sabattini, L., Trocino, C., Marchello, L. P., & Stecchi, S. (2000). Cognitive function and quality of life in multiple sclerosis patients. *Journal of Neurovirology*, 6, S186–S190.
- Gilmore, C. P., Donaldson, I., Bö, L., Owens, T., Lowe, J., & Evangelou, N. (2009). Regional variations in the extent and pattern of grey matter demyelination in multiple sclerosis: A comparison between the cerebral cortex, cerebellar cortex, deep grey matter nuclei and the spinal cord. *Journal of Neurology, Neurosurgery, and Psychiatry*, 80(2), 182–187. doi:10.1136/jnnp.2008.148767
- He, D., Zhou, H., Guo, D., Hao, Z., & Wu, B. (2011). Pharmacologic treatment for memory disorder in multiple sclerosis. *Cochrane Database of Systematic Reviews*, (10), CD008876. doi:10.1002/14651858.CD008876.pub2
- Langdon, D., Amato, M., Boringa, J., Brochet, B., Foley, F., Fredrikson, S., et al. (2012). Recommendations for a Brief International Cognitive Assessment for Multiple Sclerosis (BICAMS). *Multiple Sclerosis Journal*, 18(6), 891–898. doi:10.1177/1352458511431076
- Minden, S. L., Feinstein, A., Kalb, R. C., Miller, D., Mohr, D. C., Patten, S. B., et al. (2014). Evidence-based guideline: Assessment and management of psychiatric disorders in individuals with MS: Report of the guideline development subcommittee of the American academy of neurology. *Neurology*, 82(2), 174–181. doi:10.1212/WNL.0000000000000013
- Moore, G. R. W., Laule, C., MacKay, A., Leung, E., Li, D. K. B., Zhao, G., ... Paty, D. W. (2008). Dirty-appearing white matter in multiple sclerosis: Preliminary observations of myelin phospholipid and axonal loss. *Journal of Neurology*, 255(11), 1802–1811. doi:10.1007/s00415-008-0002-z
- Ontaneda, D., Hyland, M., & Cohen, J. A. (2012). Multiple sclerosis: New insights in pathogenesis and novel therapeutics. *Annual Review of Medicine*, 63, 389–404. doi:10.1146/annurev-med-042910-135833
- Rocca, M. A., Amato, M. P., De Stefano, N., Enzinger, C., Geurts, J. J., Penner, I. K., ... Filippi, M. (2015). Clinical and imaging assessment of cognitive dysfunction in multiple sclerosis. *The Lancet Neurology*. doi:10.1016/S1474-4422(14)70250-9
- Sepulcre, J., Masdeu, J. C., Goñi, J., Arrondo, G., Vélaz de Mendizábal, N., Bejarano, B., & Villoslada, P. (2009). Fatigue in multiple sclerosis is associated with the disruption of frontal and parietal pathways. *Multiple Sclerosis (Houndmills, Basingstoke, England)*, 15(3), 337–344. doi:10.1177/1352458508098373
- Weissert, R. (2013). The immune pathogenesis of multiple sclerosis. *Journal of Neuroimmune Pharmacology*, 8(4), 857–866. doi:10.1007/s11481-013-9467-3
- Wu, G. F., & Alvarez, E. (2011). The immunopathophysiology of multiple sclerosis. *Neurologic Clinics*. doi:10.1016/j.ncl.2010.12.009
- Younes, M., Hill, J., Quinless, J., Kilduff, M., Peng, B., Cook, S. D., & Cadavid, D. (2007). Internet-based cognitive testing in multiple sclerosis. *Multiple Sclerosis (Houndmills, Basingstoke, England)*, 13(8), 1011–1019. doi:10.1177/1352458507077626

Psychoneuroimmunology: Immune Markers of Psychopathology

Rachel A. Wamser-Nanney and Brittany F. Goodman

*Department of Psychological Sciences, Center for Trauma Recovery,
University of Missouri-St. Louis, St. Louis, MO, USA*

Overview

In recent years, the role of inflammation as a disease mechanism for both physical and psychological disorders has been increasingly recognized. Immune activation appears to play a critical role in numerous physical disorders and has been observed in conditions such as cardiovascular disease, diabetes, cancer, asthma, and obesity. Accumulating evidence suggests that inflammation also may be a key factor in the development and treatment of psychological disorders (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Raison, Capuron, & Miller, 2006). Although research regarding inflammation and psychological disorders is still emerging, abnormal levels of immune markers have been associated with psychiatric conditions including major depressive disorder (MDD), bipolar disorder (BPD), schizophrenia, and posttraumatic stress disorder (PTSD). The immune system may be a potential treatment target for these disorders. This paper will provide a brief overview of inflammation and describe how inflammation may be a factor in the development of psychopathology. We will then characterize the prior research regarding the role of immune markers in four psychological disorders where inflammation has been more extensively examined: MDD, BPD, schizophrenia, and PTSD.

Inflammation

Inflammation is the immune system's response to both internal and external stimuli that are potentially harmful to the organism. Acute inflammation is protective, preventing spread of harmful agents to nearby tissue and initiating the healing process for damaged tissue. However, chronic inflammation becomes toxic, resulting in neuronal death, and has been linked to

neurodegenerative disorders and psychopathology (Dantzer et al., 2008). Cytokines are one family of proteins that are heavily involved in the inflammatory response and are routinely measured to assess levels of inflammation. Cytokines include interferons, interleukins (IL), and growth factors that have a diverse set of functions and can be proinflammatory or anti-inflammatory or both. Inflammation research is therefore complicated, as inflammation can be helpful or harmful, depending on the context (Yirmiya & Goshen, 2011).

As the brain is responsible for processes integral to the immune response, cytokines are expressed in the brain. The vast majority of the inflammation research has utilized peripheral indices of inflammation, such as measuring levels of cytokines in the body, as opposed to directly studying immune markers in the brain. Peripheral inflammation indices are thought to be a proxy for central nervous system (CNS) inflammation due to the significant interconnections between the peripheral and central immune systems. Proinflammatory cytokines, such as IL-6, IL-1, and tumor necrosis factor- α (TNF- α), can be transported across the blood-brain barrier (BBB) and thus are directly able to alter CNS functioning. Peripheral proinflammatory signals can also be propagated across the BBB by cross-talk between the peripheral and central immune systems. While there is a considerable amount of interaction between the central and peripheral immune systems, the exact degree to which peripheral inflammation indicates neuroinflammation remains unclear.

The immune system has been found to be critically involved in brain development and functioning. Cytokines play a role in shaping brain development through cell signaling, neuronal growth and development, and modulation of neurotransmission (Yirmiya & Goshen, 2011). Proinflammatory cytokines aid in the migration of neurons and regulate the process of pruning during development (Dantzer et al., 2008). Indeed, animal studies and nonhuman primate models have found that immune activation in the prenatal and early postnatal period is related to brain volume and neurogenesis. Levels of inflammatory markers have been inversely linked with brain volumes. For example, higher levels of immune markers such as IL-6 have been associated with reduced hippocampal gray matter volume. Immune cells, such as T cells and microglia, are critical for brain and cognitive functioning and help maintain brain plasticity, particularly hippocampal neurogenesis. The brain and the immune system are strongly interconnected; yet, the precise nature of these relationships is complex and not fully understood. Future research in both animal and human models that examines these complex interactions will continue to provide additional insights regarding the effects of immune activation on the brain and behavior.

Immune Markers and Psychopathology

The robust links between the immune system and the brain create numerous opportunities for inflammation to be involved in the development and maintenance of psychopathology. Peripheral immune modulators can induce psychiatric symptoms in both animal and human models (Dantzer et al., 2008). Treatments with proinflammatory cytokines can result in “sickness behavior,” a constellation of depressive-like symptoms including decreased appetite, weight loss, fatigue, sleep disturbances, and social withdrawal. In animal models, the injection of lipopolysaccharide (LPS), a marker for the immune system to identify a foreign body as toxic, induces inflammation and results in depressive-like symptoms. Immune activation via LPS is associated with depression, anxiety, and memory impairment as well as higher levels of serum IL-1b and TNF- α . Therefore, there appears to be a direct link between inflammation and the emergence of psychopathology.

Physical health conditions associated with immune system abnormalities such as obesity, diabetes, rheumatoid arthritis, and multiple sclerosis are risk factors for mood disorders.

Similarly, depression is frequently observed among patients who have been diagnosed with a medical condition that is associated with cytokine activation, such as viral infections, rheumatoid arthritis, cancer, and neurodegenerative diseases. Other research has found that approximately half of individuals with hepatitis C and cancer develop depressive symptoms associated with increased levels of IL-6 and TNF- α (Dantzer et al., 2008). In addition to depression, a 30-year population study reported that having an autoimmune disease or being hospitalized from a serious infection increased the risk of developing schizophrenia by 29 and 60%, respectively (Benros et al., 2011). Although there are alternative explanations for why individuals with a physical health condition may develop a psychological disorder (i.e., situational stress, difficulties coping with illness, or lifestyle changes as a result of illness), the relationship between physical and psychological conditions suggests that inflammation is likely important in the etiology of psychopathology.

The exact mechanisms by which inflammation results in psychopathology are unclear; however, stress may be a critical factor (Raison et al., 2006). The stress response system includes the release of immune markers such as TNF- α and IL-6, which increase the release of corticotropin-releasing hormone (CRH), adrenocorticotrophic hormone (ACTH), and cortisol by acting directly on hypothalamic and pituitary cells. The seminal work of Kiecolt-Glaser and colleagues has demonstrated that stress, induced experimentally or in response to an external event, is related to compromised immune function and physical health conditions. Stress predicts higher rates of illness following pathogen exposure, protracted wound healing, poorer response to immunization, and alterations in several indices of immune functioning (Glaser & Kiecolt-Glaser, 2005). Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, the major stress response system, is also associated with depression. Stress may activate the immune response in the CNS via enhanced neuronal activity. The large and compelling literature regarding stress and the immune system has established clear links between stress, the brain, and the immune system, although again, the precise nature of these relationships still merits attention.

Another explanation for how inflammation may increase the risk for psychopathology is the ability of cytokines to regulate hippocampal neurogenesis. Hippocampal neurogenesis has been implicated as a key mechanism in the pathophysiology and treatment of depression, through the modulation of brain-derived neurotrophic factor (BDNF). Selective serotonin reuptake inhibitors (SSRIs) upregulate the expression of BDNF in the hippocampus, which promotes the proliferation and survival of neural progenitor cells, neural stem cells that differentiate according to location and function.

Inflammation may also create a vulnerability for psychological illness via serotonin (5-HT). Serotonin is a neurotransmitter that is heavily involved in mood functioning and has been implicated in the development and treatment of numerous psychological disorders. Inflammatory cytokines have been found to alter the metabolism and release of central 5-HT. Proinflammatory cytokines IL-1B and TNF- α have also been shown to activate 5-HT transporters. Therefore, immune-driven alterations in 5-HT are likely an important factor in understanding how inflammation may contribute to psychiatric illnesses.

Inflammation and Psychiatric Conditions

Major Depressive Disorder

The inflammation hypothesis posits that MDD is associated with sustained activation of the immune system, specifically increased production of proinflammatory cytokines and acute phase proteins (Maes, 1999). Numerous studies have suggested that depression is

accompanied by immune dysregulation. Depressed patients have exhibited increased serum levels of numerous immune markers including IL-1, IL-1B, IL-2, IL-6, interferon gamma (IFN- γ), and TNF- α . One meta-analysis of 58 studies found that IL-6, CRP, and TNF- α were significantly associated with MDD, whereas IL-1B was not tied to MDD (Haapakoski, Mathieu, Ebmeier, Alenius, & Kivimäki, 2015). It is important to note that elevated levels of inflammation were not consistently reported in all studies or for all immune markers and there was extensive heterogeneity in study-specific estimates. Children and adolescents diagnosed with MDD have also exhibited elevated levels of immune markers such as IL-6, IFN- γ , and IL-1B. Increased levels of IL-1B, IL-2, and IL-10 have been reported among unmedicated children with MDD. Elevated protein expression levels of IL-1B and TNF- α in the prefrontal cortex were observed in adolescents who completed suicide than controls.

Immune activation may differ depending on the presence of suicidality. Most individuals who have attempted suicide or had suicidal ideation have shown an imbalance of the immune system. IL-6 and TNF- α may be significantly higher, whereas IL-2 may be lower among suicidal patients versus depressed patients without suicidality. Although further research is needed in this area to draw conclusions regarding suicidality and inflammation, suicidality should be assessed in the context of MDD inflammation research. Relatively little research has investigated the influence of treatment on inflammation; however, cytokines may be reduced to normal levels following successful antidepressant treatment.

Bipolar Disorder

Inflammatory markers, combined with oxidative damage and neurotrophins, are theorized to be one of the three key pillars in a multifactorial approach to predicting BPD among those at risk (Brietzke et al., 2012). Immune alterations have been associated with many aspects of BPD including symptom severity, mood episodes, stages, and medication effects. TNF- α , IL-6, and IL-8 have been shown to be elevated during manic and depressive phase, whereas IL-2, IL-4, and IL-6 were increased during manic episodes. Other markers, such as IL-1B and IL-1, may be normal among individuals with BPD.

A large meta-analysis of thirty studies concluded that IL-4, IL-10, and TNF- α are elevated in patients with BPD compared with healthy controls (Modabbernia, Taslimi, Brietzke, & Ashrafi, 2013). Interestingly, levels of IL-2, IFN- γ , and IL-4 were unrelated to medication status. Phasic differences were present for TNF- α and IL-6, but not IL-4 and IL-10. Levels of TNF- α and IL-6 appear to normalize during the depressive phase, whereas other markers such as IL-2R remain abnormal. Larger studies examining phasic differences are needed to elucidate trait and state markers of BPD. The research with child and adolescent samples is too limited to draw conclusions at this time; however, CRP may be related to mania symptom severity.

Regarding treatment effects, a meta-analysis reported that medication did not significantly impact markers such as IL-2, IL-4, and IFN- γ (Modabbernia et al., 2013). Medication effects of other cytokines could not be determined due to the small number of studies. While there is substantial support, the immune system being involved in BPD, at this time, it is unclear which markers are most related to specific phases and the impact of treatment on the immune system.

Schizophrenia

The development of schizophrenia may be due, in part, to immune dysregulation (Miller, Buckley, Seabolt, Mellor, & Kirkpatrick, 2011). Early work revealed that prenatal infections

with bacterial or viral agents were associated with an increased risk of adult-onset schizophrenia. A substantial portion of cases diagnosed with schizophrenia spectrum disorders had mothers exposed to influenza in the first trimester of pregnancy than controls. Further, increased maternal levels of proinflammatory cytokines have been related to schizophrenia in offspring. A number of reviews describe the mechanisms by which infection alters neurodevelopment that increases the risk of developing schizophrenia (e.g., Miller et al., 2011).

However, the risk of developing schizophrenia from infection is not restricted to the prenatal period. Exposure to neurotropic virus in early childhood is associated with increased risk of subclinical psychosis during adolescence. In a 30-year population study, childhood autoimmune conditions were related to subclinical psychotic experiences in adolescents and schizophrenia in adults (Benros et al., 2011). Here, the risk of schizophrenia had a positive linear relationship with the number of severe infections among those with a history of autoimmune disease, and autoimmune conditions were more prevalent in individuals with schizophrenia than controls. One meta-analytic study revealed that the risk of schizophrenia was nearly double among adult survivors of childhood CNS viral infections (Khandaker, Zimbron, Dalman, Lewis, & Jones, 2012). Interestingly, the prevalence of autoimmune conditions is increased in people with schizophrenia as well as their unaffected first-degree relatives.

Increased levels of inflammatory markers have been recorded in those with schizophrenia and associated symptoms. First-episode psychosis and acute psychotic relapse are linked with increased concentrations of IL-6, TNF- α , IL-1 β , and IFN- γ and decreased levels of IL-10. Several meta-analyses have concluded that schizophrenia is related to altered immune functioning, with a propensity toward increased production of proinflammatory cytokines (e.g., Miller et al., 2011). It is important to note that the literature may be more conflicting, as some studies have found both decreased proinflammatory markers (e.g., IL-2, IFN- γ) and increased serum and cerebral spinal fluid (CSF) anti-inflammatory cytokines (e.g., IL-10). Nonetheless, there appears to be a dominance of proinflammatory findings. Miller and colleagues reported that levels of inflammation between controls, first-episode patients, and acutely relapsed patients were similar, suggesting that abnormal cytokine levels in schizophrenia are not a result of antipsychotic treatment. Comparatively little work has been conducted with child populations, although children with schizophrenia may have comparable levels of TNF- α to controls, both before and after clozapine treatment. More studies are needed in child populations as childhood-onset schizophrenia may present with a different inflammatory profile than adult-onset schizophrenia.

In a longitudinal study of inflammatory markers and subsequent psychotic disorders, the Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort revealed that higher concentrations of IL-6 at age 9 corresponded to a twofold increased risk of developing a psychotic disorder at age 18 (Khandaker, Pearson, Zammit, Lewis, & Jones, 2014). A robust dose-response relationship was also reported between IL-6 levels in childhood and later risk of psychotic experiences in young adulthood, even after controlling for sex, body mass, and psychological and behavioral problems. Initial CRP levels were not related to later psychosis. Additional longitudinal studies will help to confirm whether which markers are tied to the development of schizophrenia and related psychosis.

The hypothesized immune-mediation etiology for schizophrenia is supported by genome-wide association studies that report significant relationships between schizophrenia and markers close to the major histocompatibility complex region, which contains many immune-related genes, including inflammatory mediators. Genome-wide studies have identified numerous genetic loci associated with schizophrenia, many of which represent genes expressed in the brain and immune cells involved in adaptive immunity. In a recent review, inflammation-related

genes such as SERPINA3 and IFITM were consistently increased in schizophrenic brains (Trépanier, Hopperton, Mizrahi, Mechawar, & Bazinet, 2016). These findings suggest increased expression for immune-mediating genes in schizophrenia.

Further support for the role of the immune system in schizophrenia is found in treatment studies. Immunotherapy has led to symptomatic improvement in some cases of first-episode psychosis. TNF- α , IL-1 β , IFN- γ , and IL-10 may normalize after remission of symptoms from antipsychotic treatment. Treatment of acute psychosis has been related to significant reductions in IL-1 β , IL-4, and IL-6 and increases in IL-12. Thus, it appears that changes in cytokines are likely following successful treatment.

Posttraumatic Stress Disorder

Individuals with PTSD are at an increased risk for a myriad of health issues, including autoimmune disorders, diabetes, and cardiovascular disease. However, the etiology of this relationship is unknown, and studies regarding inflammation and PTSD appear more conflicting than other disorders. The first meta-analysis regarding PTSD and inflammation was recently conducted by Passos et al. (2015), which included 20 studies that contrasted inflammatory markers among individuals with PTSD compared with healthy controls. Concentrations of IL-6, IL-1B, and IFN- γ were higher in the PTSD group than controls, with large effect sizes for IL-1B and IL-6 and a medium effect size for IFN- γ . PTSD duration was positively related to IL-1B, and symptom severity was tied to IL-6. Higher levels TNF- α were observed in the unmedicated PTSD group. In the studies that excluded individuals with comorbid MDD, levels of TNF- α , IL-1B, and IL-6 remained elevated. Consequently, use of medication and comorbid conditions may account for conflicting results. It is important to note that research studies with trauma-exposed individuals, who may or may not have PTSD, have also reported higher levels of CRP, IL-1B, IL-6, and TNF- α , suggesting full PTSD diagnostic criteria need not be met for immune dysfunction to manifest.

Coelho, Viola, Walss-Bass, Brietzke, and Grassi-Oliveira (2014) reviewed the nascent literature regarding child maltreatment and inflammation. A history of child maltreatment was related to increased levels of CRP, fibrinogen, TNF- α , and IL-6. CRP was the most robust marker. However, many of the studies included individuals with MDD, only one study was specific to PTSD, and most of the studies utilized small samples ($ns < 20$). Thus, it is premature to draw conclusions regarding immune activation and child maltreatment at this time. There is also very limited information regarding the impact of treatment on immune markers among patients with PTSD.

Conclusions and Future Directions

There is substantial support for the role of the immune system in the development and maintenance of psychopathology, and altered levels of inflammatory markers have been observed in MDD, BPD, schizophrenia, and PTSD. Physical health issues have been consistently associated with multiple psychological disorders, and there is evidence that this relationship is mediated by immune functioning. Yet, our ability to determine the role of inflammation on psychological conditions is limited by the caliber of the current published studies. Numerous early studies had relatively poor quality, including small sample sizes and failing to control for important confounds such as body mass index and other psychiatric conditions. Further, as

most studies currently use peripheral markers to establish these connections, more longitudinal studies are needed connecting inflammatory markers in the CNS before confidence can be placed on this etiological explanation.

Study differences complicate the field's ability to draw conclusions regarding inflammation and specific psychiatric disorders. Rates of comorbidity are high among patients with MDD, BPD, schizophrenia, and PTSD, which makes it more challenging to determine if there is a unique inflammatory profile associated with a specific disorder. Some studies exclude all comorbid conditions, whereas others only exclude psychosis and/or bipolar. Not all studies provide information regarding other psychiatric conditions. For example, only 47% of studies included data regarding other Axis I or Axis II diagnoses, and 26% of studies did not assess or report other Axis I or Axis II disorders (Haapakoski et al., 2015). Further, many studies do not include information on the different subtypes of MDD and PTSD, which is important in light of the variability across phenotypes of these conditions. A shift in research focus from a diagnostic construct to specific symptoms and dimensions of symptoms may shed light on how immune markers interact with psychiatric difficulties.

The majority of the evidence linking immune function to psychopathology is correlational. Research targeting the precise mechanisms by which immune dysfunction leads to psychopathology is necessary before causal conclusions can be drawn. It may be that the immune response contributes to the development of psychopathology, but it is also possible that the presence of psychopathology leads to dysfunction in the immune system or that another factor induces both immune dysregulation and psychological symptoms. There is some support for a causal relationship as studies have demonstrated that administration of proinflammatory cytokines and LPS leads to disordered behavioral symptoms and immune markers may normalize as symptoms resolve after treatment (Dantzer et al., 2008; O'Connor & Spagnola, 2009). However, these studies are relatively few and not sufficient for demonstrating causal influence. Findings in human correlational research are also inconsistent, and results vary across samples and specific immune markers. This research area is relatively young, with the first meta-analysis in some disorders being published within the last 2 years (i.e., Passos et al., 2015).

The long-term effects and development of alterations in the immune system are still unclear. Longitudinal and prospective studies of immune markers and specific psychiatric conditions are needed to more definitively conclude that inflammation is a key factor in the development of psychopathology. Given the importance of stressful life events and traumatic exposure, research is also needed to examine immune markers and indices of stress (i.e., cortisol) and psychiatric conditions. Despite these limitations, there is a preponderance of data pointing to the importance of inflammation in numerous psychiatric conditions. Immune markers represent critical putative biomarkers and intervention targets that may ultimately allow us to better ameliorate psychiatric symptoms.

Author Biographies

Rachel A. Wamser-Nanney, PhD, is an assistant professor in the Department of Psychological Sciences at the University of Missouri–St. Louis. Her research interests are specific to traumatic stress, particularly traumatic events that begin in childhood, complex trauma, trauma-focused assessment and treatments, and the cognitive and immune alterations associated with trauma exposure.

Brittany F. Goodman, MA, is a graduate student in the Behavioral Neuroscience Program at the University of Missouri–St. Louis. Research interests include the psychophysiology of traumatic stress and PTSD. Specific interests include changes in the nervous system reflected by measures of heart rate variability and their relationship to social behaviors. She is currently a graduate research assistant at the Center for Trauma Recovery.

References

- Benros, M. E., Nielsen, P. R., Nordentoft, M., Eaton, W. W., Dalton, S. O., & Mortensen, P. B. (2011). Autoimmune diseases and severe infections as risk factors for schizophrenia: A 30-year population-based register study. *American Journal of Psychiatry*, 168(12), 1303–1310.
- Brietzke, E., Mansur, R. B., Soczynska, J. K., Kapczynski, F., Bressan, R. A., & McIntyre, R. S. (2012). Towards a multifactorial approach for prediction of bipolar disorder in at risk populations. *Journal of Affective Disorders*, 140(1), 82–91.
- Coelho, R., Viola, T. W., Walss-Bass, C., Brietzke, E., & Grassi-Oliveira, R. (2014). Childhood maltreatment and inflammatory markers: A systematic review. *Acta Psychiatrica Scandinavica*, 129(3), 180–192.
- Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature reviews neuroscience*, 9, 46–56.
- Glaser, R., & Kiecolt-Glaser, J. K. (2005). Stress-induced immune dysfunction: Implications for health. *Nature Reviews Immunology*, 5(3), 243–251.
- Haapakoski, R., Mathieu, J., Ebmeier, K. P., Alenius, H., & Kivimäki, M. (2015). Cumulative meta-analysis of interleukins 6 and 1 β , tumour necrosis factor α and C-reactive protein in patients with major depressive disorder. *Brain, Behavior, and Immunity*, 49, 206–215.
- Khandaker, G. M., Pearson, R. M., Zammit, S., Lewis, G., & Jones, P. B. (2014). Association of serum interleukin 6 and C-reactive protein in childhood with depression and psychosis in young adult life: A population-based longitudinal study. *JAMA psychiatry*, 71(10), 1121–1128.
- Khandaker, G. M., Zimbron, J., Dalman, C., Lewis, G., & Jones, P. B. (2012). Childhood infection and adult schizophrenia: A meta-analysis of population-based studies. *Schizophrenia Research*, 139(1–3), 161.
- Maes, M. (1999). Major depression and activation of the inflammatory response system. *Advances in Experimental Medicine and Biology*, 461, 25.
- Miller, B. J., Buckley, P., Seabolt, W., Mellor, A., & Kirkpatrick, B. (2011). Meta-analysis of cytokine alterations in schizophrenia: Clinical status and antipsychotic effects. *Biological Psychiatry*, 70(7), 663–671.
- Modabbernia, A., Taslimi, S., Brietzke, E., & Ashrafi, M. (2013). Cytokine alterations in bipolar disorder: A meta-analysis of 30 studies. *Biological Psychiatry*, 74(1), 15–25.
- O'Connor, T. G., & Spagnola, M. E. (2009). Early stress exposure: Concepts, findings, and implications, with particular emphasis on attachment disturbances. *Child and Adolescent Psychiatry and Mental Health*, 3(1), 24.
- Passos, I. C., Vasconcelos-Moreno, M. P., Costa, L. G., Kunz, M., Brietzke, E., Quevedo, J., ... Kauer-Sant'Anna, M. (2015). Inflammatory markers in post-traumatic stress disorder: A systematic review, meta-analysis, and meta-regression. *The Lancet Psychiatry*, 2(11), 1002–1012.
- Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: Inflammation and the pathogenesis of depression. *Trends in Immunology*, 27(1), 24–31.
- Trépanier, M. O., Hopperton, K. E., Mizrahi, R., Mechawar, N., & Bazinet, R. P. (2016). Postmortem evidence of cerebral inflammation in schizophrenia: A systematic review. *Molecular psychiatry*.
- Yirmiya, R., & Goshen, I. (2011). Immune modulation of learning, memory, neural plasticity and neurogenesis. *Brain, Behavior, and Immunity*, 25(2), 181–213.

Suggested Reading

- Dowlati, Y., Herrmann, N., Swardfager, W., Liu, H., Sham, L., Reim, E. K., & Lanctôt, K. L. (2010). A meta-analysis of cytokines in major depression. *Biological Psychiatry*, 67(5), 446–457.
- Goldsmith, D. R., Rapaport, M. H., & Miller, B. J. (2016). A meta-analysis of blood cytokine network alterations in psychiatric patients: Comparisons between schizophrenia, bipolar disorder and depression. *Molecular Psychiatry*, 21(12), 1696–1709. doi:10.1038/mp.2016.3
- Schwartz, M., & Kipnis, J. (2011). A conceptual revolution in the relationships between the brain and immunity. *Brain, Behavior, and Immunity*, 25(5), 817.
- Tursich, M., Neufeld, R. W. J., Frewen, P. A., Harricharan, S., Kibler, J. L., Rhind, S. G., & Lanius, R. A. (2014). Association of trauma exposure with proinflammatory activity: A transdiagnostic meta-analysis. *Translational Psychiatry*, 4, e413.
- Dantzer, R., & Kelley, K. W. (2007). Twenty years of research on cytokine-induced sickness behavior. *Brain, Behavior, and Immunity*, 21(2), 153–160.

Genetic Polymorphisms

Mikhail Votinov^{1,2} and Katharina Sophia Goerlich¹

¹*Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany*

²*Institute of Neuroscience and Medicine (INM-10), Jülich, Germany*

The Study of Genetics

DNA molecules consist of two strands that are coiled around each other like a twisted ladder, forming a double helix. The two DNA strands are called polynucleotides since they consist of smaller units called nucleotides. Each nucleotide is composed of a nitrogen-containing nucleobase: either adenine (A) and thymine (T), creating A–T pairs, or cytosine (C) and guanine (G), creating G–C pairs, as well as sugar (deoxyribose) and a phosphate group.

Polymorphisms are variations of a particular DNA sequence. The most common type of polymorphisms involves variation at a single base pair. These are called single nucleotide polymorphisms (SNPs). For example, an SNP may replace the cytosine (C) with thymine (T) at a certain DNA locus. When such SNPs occur within a gene, they may change the amino acid sequence of the gene's protein product or change the timing, location, or level of gene expression. Another type of polymorphism is a variation (e.g., repeats) in tandems of two or more adjacent nucleotides (see Figure 1).

Despite intense research on the role of genetics in human health and behavior in the past decades, we are still far from fully understanding the complex interactions between genes and behavior. Here, we provide an overview of several genes whose polymorphisms have been extensively studied due to their role in the regulation of molecular mechanisms and modulation of neurotransmitter systems in the brain, leading to changes in human cognition and behavior.

Dopamine System

Numerous studies across a wide range of species demonstrated that the dopaminergic system plays a key role in reward, behavioral flexibility, working memory (WM), positive reinforcement, and learning processes. Malfunction in this system is associated with behavioral

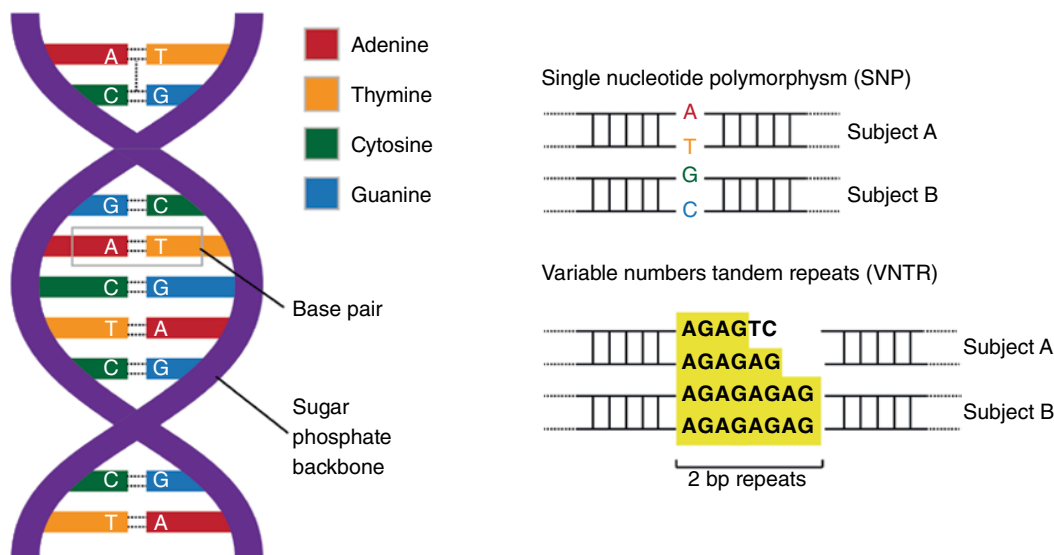


Figure 1 Overview of DNA structure and types of gene polymorphisms 225 × 128 mm (300 × 300 DPI).

abnormalities and diseases such as impulsive behaviors, addiction, Parkinson's disease, and schizophrenia.

Dopamine (DA) is a monoamine neurotransmitter that is produced by DA neurons in two regions of the midbrain, the ventral tegmental area (VTA) and substantia nigra pars compacta (SNc), and projected to a broad number of brain regions. There are three major dopaminergic projections in the central nervous system: the nigrostriatal pathway, regulating motor functions; the mesocorticolimbic pathway, which plays a key role in reward, motivation, learning, emotions, and memory; and the tuberoinfundibular pathway, involved in prolactin secretion regulation and sensory processing.

DA transmission in the brain is mediated by five (D1–D5) subtypes of DA receptors, dopamine transporters (DAT), and enzyme catabolizers, which show different patterns of distribution across brain regions and therefore affect different cognitive functions. For instance, the concentration of D1 receptors is higher in prefrontal areas, while D2 receptor concentration is most prevalent in the striatum. DAT density is highest in subcortical regions, and catechol-*O*-methyltransferase (COMT) plays a major role in DA clearance in the prefrontal cortex (PFC).

Genetic polymorphisms of the DA system include SNPs and variable number tandem repeat (VNTR) polymorphisms of the metabolizing enzymes, receptors and transporters, which modulate tonic DA (the average amount of DA available over a relatively long period of time) or phasic DA levels (the amount of DA being released in a given burst of neuronal firing). The most important genes modulating DA transmission comprise the enzyme catabolizers COMT and monoamine oxidase A (MAOA), dopamine- and cAMP-regulated phosphoprotein (DARPP-32), the dopamine receptor D2 (DRD2) and D4 (DRD4) genes, and DAT.

Serotonin System

A further important monoamine neurotransmitter is serotonin (5HT or 5-hydroxytryptamine), which is produced in the raphe nuclei of the midbrain and transported to the limbic system, cerebellum, and cortex. The serotonin system plays an important role in sensory processing,

sleep, memory, mood, and arousal. Pathology in this system has been associated with diseases such as depression, antisocial behavior, bipolar disorder, and schizophrenia.

The best-investigated gene modulating serotonin transmission is the serotonin transporter-linked polymorphic region (5-HTTLPR) gene. Genetic variations in this gene have been linked to personality traits such as impulsivity and neuroticism and to a range of psychiatric illnesses as well as the treatment success of such disorders.

5HT, catecholamines, and histamine are metabolized by the COMT and monoamine oxidase (MAO) enzymes. There are two MAO isoforms (MAOA and MAOB) that are encoded by distinct genes on the X chromosome. MAOA and MAOB differ in their substrate specificity, anatomical distribution, regulation by pharmacological agents, and influence on behavior. Serotonin is mostly degraded by MAOA, while DA is degraded by both MAOA and MAOB.

Opioid System

The opioid system has been extensively studied due to its association to pain, analgesia, reward sensitivity, and substance abuse. There are several endogenous opioids—dynorphins, enkephalins, endorphins, and endomorphins—which bind with high affinity to particular μ -, δ -, and κ -opioid receptors. Important genes modulating the opioid system are the μ -opioid receptor gene (OPRM1) and prodynorphin (PDYN), a gene coding for dynorphin opioid peptides, which have a high affinity to κ -opioid receptors. Genetic variations in these genes have been associated with individual differences in the reward processing as well as different forms of addiction, such as nicotine and alcohol dependence.

Neuroendocrine System

The hypothalamus and pituitary gland are the center of integration for the nervous and the endocrine system. The neuroendocrine system plays a role in social behavior, mating, pair bonding, trust, aggression, and empathy. A well-studied gene modulating the neuroendocrine system is the oxytocin receptor (OXTR) gene, which has been implicated in a wide range of social behaviors.

In addition to the genes mentioned above, the proteins apolipoprotein E (ApoE) and the brain-derived neurotrophic factor (BDNF) have been extensively investigated due to their involvement in memory, aging, depression, anxiety, and mood disorders. ApoE is a major cholesterol carrier that supports lipid transport and injury repair in the brain, whose allele number 4 ApoE ϵ 4 is the most important genetic risk factor for Alzheimer's disease (AD) and dementia. BDNF is a protein involved in the proliferation, neurogenesis, differentiation, survival of neuronal cells, and neuroplasticity, which is involved in the pathogenesis of a wide range of psychiatric disorders. Table 1 provides an overview of the here discussed genes and polymorphisms and their involvement in functional domains of cognition and behavior.

Polymorphisms and Behavior

Reward Processing

Rewards may be defined as events that generate approach and consummatory behavior, evoking positive emotions and leading to learning of this behavior. Rewards that are crucial for survival

Table 1 A list of genes associated with neurotransmitter systems and their related polymorphisms and the proposed mechanism of those polymorphisms on cognitive function.

<i>Gene</i>	<i>Polymorphism</i>	<i>Mechanism</i>	<i>Functional domain</i>
Dopamine system			
COMT (<i>catechol-O-methyltransferase</i>)	SNP Val158Met (rs4680) Substitution of methionine (Met) in place of valine (Val) at codon 158	Met/Met carriers have a 2–4 fold decrease of COMT activity, compared with the Val/Val genotype, leading to higher extracellular dopamine levels in the prefrontal cortex	Reward, working memory, learning, attention
Enzyme with high availability in prefrontal cortex that degrades catecholamines			
DRD2 (<i>dopamine receptor D2</i>)/ANKK1 (<i>ankyrin repeat and kinase domain</i>)	DRD2/ANKK1-Taq1A (rs1800497) Located in a putative substrate-binding domain of the ANKK1 gene, resulting in a Glu713Lys substitution	This polymorphism modulates the density of DA D2 receptors. A1 allele carriers show a reduction in striatal D2 receptor density compared to the noncarriers	Reward, learning, neuroticism, addiction, schizophrenia
Closely linked to the dopamine receptor D2 (DRD2) on chromosome band 11q23.1			
DRD4 (<i>dopamine receptor D4</i>)	48-bp tandem repeat (VNTR)	The 7-repeat variant has been linked to decreased postsynaptic inhibition and lower dopaminergic tone	Impulsivity, novelty seeking, addiction, obsessive–compulsive disorder, ADHD
Located on chromosome 11p, predominantly expressed in prefrontal cortex	Codes for the third cytoplasmic loop of the receptor		
DAT (<i>dopamine transporter</i>) SLC6A3	40-bp VNTR (rs28313670)	This polymorphism produces two common alleles with 9 and 10 repeats (9R and 10R), which affect the basal level of expression of the transporter. The 9R allele is associated with increased DAT activity in human adults	Reward, addiction, learning, working memory, cognitive flexibility, ADHD
High availability in striatum, regulates dopamine signaling at the synapse through reuptake of dopamine into presynaptic terminals, thus terminating signaling	Located in the 3'-untranslated (UTR) region of the gene with repeat numbers between 3 and 13		
DARPP-32 (<i>dopamine- and cAMP-regulated phosphoprotein</i>)	DARPP-32 Richly expressed protein in the striatum, regulated by D1 and D2 dopamine receptors in opposing directions	The protein modulates striatal dopamine cellular excitability and synaptic plasticity related to the dopamine receptors. AA homozygotes have higher D1 dopamine receptor efficacy	Learning, cognitive flexibility, anger
PPP1R1B (protein phosphatase 1 regulatory inhibitor subunit 1B), located on chromosome 17q12			

Table 1 (Continued)

<i>Gene</i>	<i>Polymorphism</i>	<i>Mechanism</i>	<i>Functional domain</i>
Serotonin system			
5-HTT/SLC6A4 (<i>serotonin transporter gene</i>)	5-HTTLPR Short and long repeats in the 5-HTT-linked polymorphic region. The short variation has 14 repeats of a sequence, the long variation has 16 repeats	The short allele is associated with lower levels of serotonin uptake and lower transcriptional efficiency of the serotonin transporter protein compared with the long allele	Impulsivity, aggression, antisocial behavior, anxiety, depression, bipolar disorder
MAOA (<i>monoamine oxidase A</i>) An enzyme that metabolizes catecholamines, 5HT and histamine	30-bpVNTR Located 1.2 kb upstream from exon 1 in the 5' UTR of MAOA gene	Common are 3 and 4 repeats, less frequent are 2, 3.5, and 5 repeats. The alleles with 3.5 or 4 repeats (high activity alleles) have 2–10 times more transcriptional activity than those with 2 or 3 copies (low-activity alleles)	Aggression, antisocial behavior, depression, reward, addiction
Opioid system			
OPRM1	SNP (rs1799971) Often referred to as A118G, situated in exon 1	The variant leads to the conversion of the amino acid asparagine to aspartate at codon 40	Addiction, alcoholism, Smoking
PDYN (<i>prodynorphin</i>) Codes the dynorphin opioid peptides, which have a high affinity to κ receptors	This polymorphism is located in the PDYN gene promoter region with one to four repeats of a 68-bp element containing one binding site per repeat for the transcription factor AP-1 (c-Fos/c-Jun)	Alleles with 3 or 4 repeats are associated with higher levels of dynorphins expression than alleles with 1 or 2 repeats	Reward, addiction, alcoholism, memory
Neuroendocrine system			
OXTR (<i>oxytocin receptor</i>)	SNP (rs53576), which consists of a G to A change within the third intron	Research usually compares individuals homozygous for the G allele (GG) with individuals with A allele carriers (AA/AG)	Prosocial behavior, empathy, trust, mating, depression, personality traits

(Continued)

Table 1 (Continued)

<i>Gene</i>	<i>Polymorphism</i>	<i>Mechanism</i>	<i>Functional domain</i>
Proteins			
ApoE (<i>apolipoprotein E</i>)	ApoE-ε2, ApoE-ε3, ApoE-ε4	ApoE ε4 is associated with an increased risk of developing Alzheimer's disease,	Memory, Alzheimer's disease, mild cognitive impairment
Produces a protein that, when combined with fat, becomes a lipoprotein	Three allelic variants of ApoE as defined by the two SNPs rs429358 and rs7412	ApoE ε2 with a reduced risk, relative to the more common ε3 allele	
BDNF (<i>brain-derived neurotrophic factor</i>)	BDNF Val66Met	The Met allele results in reduced activity-dependent release of BDNF and inefficient BDNF trafficking	Memory, aging, depression
Supports neuronal growth and differentiation, induces long-lasting changes in synaptic plasticity	Substitution of methionine (Met) for valine (Val) at codon 66 of the pro-BDNF protein		

such as eating, drinking, and reproduction are called primary rewards. Rewards that are attributed to, for instance, money, gambling, and aesthetic stimuli are called secondary rewards.

The ability to predict future rewards and losses and to assess the value of upcoming outcomes is a fundamental aspect of learning and adaptive behavior. Numerous neurophysiological and neuroimaging studies demonstrated that anticipating and receiving rewards activates the brain's reward circuitry, including the dopaminergic midbrain, striatum, and PFC. Given the fundamental role of DA in reward processing, studies investigating reward anticipation and feedback, approach motivation, goal-directed behavior, and pathological aspects of the reward system focused on several genetic variations in the dopaminergic system to reveal how those variations modulate reward-related behavior. These investigations confirmed significant effects of polymorphisms in the COMT, DAT, DRD2/Taq1A, and DRD4 genes on reward behavior and its underlying brain activity.

One of the best-investigated genetic variations in the dopaminergic system is the COMT Val158Met polymorphism, which contributes to a degradation of DA in PFC and modulates activity in the cortico-striatal loop connecting the frontal cortex and the brain's core reward center, the striatum. Imaging genomics studies found that there is a linear relationship between the number of met158 alleles and ventral striatal activity and that the activation of the ventral striatum and anterior cingulate cortex is modulated by the COMT polymorphism. Further, an interaction between the COMT Val158Met polymorphism and stressful life events was observed, such that Met/Met carriers (who have higher levels of DA in PFC) with low exposure to lifetime stress showed a lower sensitivity to reward than Met/Met carriers with high exposure to lifetime stress. A study investigating how the interaction between the COMT and DAT genes influences reward processing revealed that both variants modulated activation in PFC and striatum and that the combinations COMT Met/Met DAT 10R and COMT Val/Val 9R were associated with blunted responses to rewards in the ventral striatum. People with the 22q11.2 deletion syndrome (a microdeletion on chromosome 22q11.2, where the

gene coding for COMT is located) displayed reduced activity in medial frontal regions during reward anticipation.

Studies of the DAT1 gene revealed that carriers of the 9-repeat allele, associated with high levels of DA in the striatum, showed greater activity in the ventromedial striatum during reward anticipation than homozygotes for the 10-repeat allele. Individual differences in extraversion and the presence of the DRD2 Taq1A allele predicted a significant amount of inter-subject variability in the magnitudes of reward-related brain activity. Moreover, the DRD2 gene was linked to the interindividual variations in neural responses to expectations of reward and negative emotional processing, as well as stress-induced DA release.

These studies demonstrated that genetic variations in the COMT gene as well as the DAT1 and DRD2 genes alter the processing of rewards in the brain. However, effects of polymorphisms in other neurotransmitter systems have also been observed. For example, T homozygotes carriers of the oxytocin (OXTR) gene demonstrated relatively decreased activation in the mesolimbic circuitry when anticipating rewards. Moreover, individuals with the HH genotype of the 68-bp VNTR PDYN gene showed higher activation in the medial orbitofrontal cortex (mOFC) when anticipating monetary rewards and stronger functional coupling of mOFC with the ventral striatum and ventral medial PFC than individuals with the LL genotype, indicating an increased sensitivity for upcoming rewards.

Besides leading to individual differences in how rewards are processed in the healthy brain, genetic variations in neurotransmitters implicated in the reward system, genetic variations may contribute to a hyper- or hyposensitivity to rewards that can become pathological. Hypersensitivity to potential rewards may lead to an excessive increase in approach behavior in the presence of rewards, while a decreased sensitivity may lead to seeking ever more stimulating experiences in order to compensate for low levels of reward experience. Such malfunctions of the reward system can contribute to pathological forms of reward behavior, such as sensation seeking, risk-taking, pathological gambling (PG), anhedonia, and addiction.

Drugs induce a pleasurable state and are therefore experienced as rewarding and thus reinforcing. In addition, repeated self-administration of drugs causes cellular and molecular changes in neurons as well as homeostatic adaptations, which further contribute to increased tolerance of the drug and, eventually, addiction. Drugs act via different systems: opioid drugs via binding with μ -opioid receptors, cocaine and amphetamine through the DAT, and ethanol through GABA_A receptors and the inhibition of NMDA glutamate receptors. Understanding genetic risk factors for individual vulnerability to addiction is of high importance for the development of prevention strategies.

There is evidence that individual variations in the opioid OPRM1 and PDYN genes are related to the development of opioid and ethanol substance dependence. Methamphetamine and heroin-dependent individuals exhibited a significantly higher frequency of the 3- or 4-repeat allele 68-bp VNTR polymorphism in the PDYN gene than healthy individuals. Similarly, several other PDYN polymorphisms (e.g., rs1022563, rs2235749) were associated with cocaine, heroin, and alcohol. Comparable findings were observed for an SNP of the μ -opioid receptor gene OPRM1 (rs1799971), which has been linked to heroin abuse, alcoholism, and smoking. A recent meta-analysis confirmed that the OPRM1 SNP contributes to mechanisms of addiction liability and is shared across different addictive substances. Moreover, studies investigating DA receptor candidate genes suggested that the DRD2 A1 allele (DRD2 Taq1A polymorphism) was associated with a higher consumption of alcohol and heroin.

A well-studied example of addiction not including substance abuse (i.e., behavioral addiction) is PG. In the recently released fifth edition of the Diagnostic and Statistical Manual

of Mental Disorders (DSM-5), pathological gambling (renamed “disordered gambling”) was moved from the “Impulse Control Disorders Not Elsewhere Classified” category into the new “Substance-Related and Addictive Disorders” category. PG is a chronic, progressive disorder that is associated with the urge to gamble continuously despite harmful negative consequences or a desire to stop. Often, gambling is used as a model for investigating mechanism of developing a behavioral addiction. The heritability of PG is estimated to be 50–60%. PG shares genetic vulnerability factors with alcohol dependence and depressive disorder, with the genetic correlation between PG and alcohol dependence ranging from 0.29 to 0.44, with men showing a higher correlation than women.

Genetic studies have associated PG with the DRD2 TaqA1 polymorphism, the 10/10 alleles of the DAT1 gene, and polymorphisms of DRD2/ANKK1, revealing that pathological gamblers with genetic predispositions toward lower availability of DA and D2 receptor density are at a higher risk of cognitive flexibility difficulties. Other candidate genes that have been the focus of genetic association studies of PG are the serotonin transporter gene and the MAOA genes. Allelic frequency of the 3-copy allele MAOA polymorphism was observed to be significantly higher in male gamblers than in men without PG, and the less functional S allele of the 5-HTTLPR polymorphism was more frequent in male gamblers. However, the only genome-wide association study of PG to date in 1,312 twins found no single candidate gene reaching genome-wide significance, rendering current results of genetic variations underlying PG preliminary and in need of replication.

Substance abuse and behavioral addictions are highly comorbid with other maladaptive behaviors, such as impulsivity, risk-taking, and sensation and novelty seeking. Impulsivity is characterized as the inability to withhold an action for the time needed to estimate consequences. An example of an impulsive choice is when an individual preferentially chooses an immediately available small reward rather than enduring a certain time delay for a larger reward (“delay discounting”), a behavior that is frequently observed in individuals with substance use disorders. Independent twin and longitudinal twin studies have demonstrated a heritability of impulsivity in the range of 40–50% and implicated the DA-related genes COMT, DRD2, DRD4, and DAT in impulsivity, delay discounting, and risk-taking. However, the most extensively studied polymorphism in the context of impulsivity is the serotonin transporter 5-HTTLPR, revealing that carriers of the less active S allele score higher on attentional impulsiveness and are less sensitive to punishment-related information.

Learning

Based on the reinforcing role of rewards in learning processes, the dopaminergic system is also a key component of learning and motivation, as evidenced by a wealth of findings across a range of different species from rats to humans. In the striatum, DA is critical for reinforcing actions that are most likely to lead to a reward. Demonstrating this, striatal activation has been observed to predict the amount of learning effects regarding WM updating. In response to positive “prediction errors” (events that are better than expected), DA cells burst fire, whereas the same cells remain silent in response to negative prediction errors (events that are worse than expected). Reinforcement learning models assume that these bursts and dips act as teaching signals by modifying synaptic plasticity in the structures involved. Studies in rodents have shown that phasic (but not tonic) stimulation of dopaminergic cells induces behavioral conditioning, and conversely, selective genetic disruption of phasic dopaminergic burst firing produces behavioral learning deficits, demonstrating a direct influence between DA activity on learning in animals.

In humans, several genes controlling dopaminergic function in the frontostriatal circuitry have been investigated in the context of learning, including COMT, DARPP-32, DAT1, DRD2, and DRD4. The COMT Val158Met polymorphism has repeatedly been found to influence cognitive processing in a variety of tasks requiring prefrontal function, and a recent meta-analysis confirmed the involvement of the COMT Val158Met polymorphism in reward learning. Genetic polymorphisms modulating DARPP-32 mRNA expression and cognition in humans are associated with changes in activation in the striatum and with frontostriatal connectivity. Biophysical models showed that DARPP-32 serves to integrate DA signals across time, demonstrating a key role of this gene in probabilistic reinforcement learning. The DAT1 polymorphism is predictive of neural and behavioral responses to cognitive flexibility and has further been shown to modulate striatal activity as a function of WM load in an updating task. The DRD2 gene polymorphism affects D2 receptor density in the striatum, the brain region in which D2 receptors are by far most prevalent. DRD2 has been demonstrated to be strongly predictive of learning from negative reward prediction errors, that is, avoiding responses that lead to negative outcomes. Lastly, the DRD4 gene is associated with dopaminergic function in PFC, in which the D4 receptor is primarily expressed, and has been found predictive of error-related prefrontal activity and subsequent behavioral adjustments.

Besides the DA system, also the serotonin system has been implicated in individual differences in learning and decision making, revealing a differential association of the serotonin transporter 5-HTTLPR polymorphism with reflexive and reflective optimal learning. Moreover, the Val66Met polymorphism of the neurotrophin BDNF, which is involved in regulating synaptic plasticity in many brain areas including the hippocampus, has been suggested to affect sensorimotor cortex activity as well as motor learning in humans. Individuals with the BDNF Met allele have been reported to show increased cognitive performance and to commit more errors during short-term learning during a driving-based motor learning task. Furthermore, a recent study provided first evidence that dynorphins contribute to individual differences in reversal learning: carriers of the HH genotype (alleles with 3 or 4 repeats) of the nucleotide tandem repeat (68-bp VNTR) functional polymorphism of the prodynorphin (PDYN) made more perseverative errors than carriers of the LL genotype in a reversal learning task requiring flexible adaptation to stimulus–response associations and showed less engagement of the orbitofrontal cortex (OFC) and cortico-striatal circuitry as well as lower effective connectivity of the OFC with the anterior midcingulate cortex and the anterior insula/ventrolateral PFC during reversal learning and the processing of negative feedback.

Taken together, individual differences in human learning capacity have been robustly associated with genetic variations in the DA system. While many studies have linked single DA-related candidate genes to individual differences in learning, it has to be considered that the well-studied COMT val/met polymorphism can itself be moderated by other dopaminergic polymorphisms such as DAT1 and DRD2 as well as within the COMT gene itself, requiring future investigations taking more complex gene×gene interactions into account. Besides the COMT Val158Met polymorphism, individual differences in learning seem further modulated by the BDNF Val66Met polymorphism and the PDYN VNTR polymorphism.

Memory

The apolipoprotein E (ApoE) gene is the most important known genetic risk factor for AD, a chronic neurodegenerative disease causing dementia. ApoE has three major alleles: ApoE ϵ 2, ϵ 3, and ϵ 4. ApoE ϵ 4 is associated with an increased risk of AD, ApoE ϵ 2 with a reduced risk, relative to the more common ϵ 3 allele. Healthy carriers of *APOE* ϵ 4 show an accelerated

decline in memory tests starting at the age of 55–60 years. While the underlying mechanism of this effect of ApoE $\epsilon 4$ is not fully understood, evidence suggests an interaction with amyloid: AD is characterized by buildups of aggregates of the peptide beta-amyloid. ApoE enhances proteolytic breakdown of this peptide, and ApoE $\epsilon 4$ appears less effective than the other alleles at promoting these reactions, resulting in increased vulnerability to AD in individuals carrying the ApoE $\epsilon 4$ allele. Regarding protective factors, evidence suggests that a high education level, active leisure activities and exercise, and maintenance of vascular health are beneficial in reducing the risk of AD and cognitive decline, particularly in APOE $\epsilon 4$ carriers.

Besides ApoE, individual differences in memory performance are influenced by the neurotrophin BDNF, whose levels of expression are particularly high in the hippocampus, the brain's memory center, in which the consolidation of information from short-term to long-term memory takes place. The BDNF Val66Met polymorphism produces a nonconservative substitution of amino acids at codon 66 of the gene, involving a valine (Val) being replaced by a methionine (Met). The presence of a copy of the Met allele is associated with less activity-dependent BDNF secretion, as well as abnormal BDNF localization. A number of behavioral and neuroimaging studies observed that Met homozygotes perform worse on verbal episodic memory tests as well as on visual sensory memory tests than Val homozygotes and that carriers of the Met allele activate the hippocampus less during memory encoding and retrieval, suggesting weaker memory trace formation in carriers of the Met allele.

The extracellular enzyme COMT is known to degrade DA in the synapses. In humans, COMT is mostly expressed in the PFC and thus has a direct influence on prefrontal DA level. The COMT Val158Met polymorphism, which is known to affect prefrontal DA levels and D1 receptor binding, influences cognitive processing in various tasks that depend on prefrontal function. Regarding memory performance, COMT Val158Met modulates the activity and connectivity of brain regions within the WM network. This effect is stronger with increasing age, contributing to an age-related decline in memory performance. The relation between DA and WM depends on a person's baseline DA level and is thus characterized by an inverted U-shape function: administration of DA increases WM performance in individuals with low WM capacity but decreases performance in those with high WM capacity. These differences likely reflect differences in baseline DA levels, with low DA levels in individuals with low WM capacity and high DA levels in those with high WM capacity, such that additional DA administration either optimizes the DA level or exceeds it, depending on a person's baseline DA. Thus, there is an optimum concentration of DA in the PFC to perform WM tasks. These findings demonstrate that individual differences in WM performance are genetically determined by DA level and modified by the COMT Val158Met polymorphism.

A number of animal studies and some human studies have shown that memory is further modulated by dynorphins. Dynorphins are members of the opioid peptide family that are located in the hippocampus, amygdala, hypothalamus, striatum, and spinal cord and preferentially bind to κ -opioid receptors. Their functions are related to memory, learning, emotional control, pain, and stress response. Individuals with the minor dynorphin alleles rs1997794 and rs910080 have been observed to show better episodic memory performance than homozygote carriers of the major allele. Furthermore, carriers of the prodynorphin T allele at rs1997794 showed reduced fear extinction and a significantly diminished functional connectivity between amygdala and ventromedial PFC, demonstrating a role of dynorphin κ -opioid receptor signaling in fear memory and extinction in humans.

In sum, differences in individual memory capacities seem to be significantly influenced by the $\epsilon 4$ allele of the ApoE gene, the BDNF Val66Met polymorphism, basic DA levels and the COMT Val158Met polymorphism, and minor alleles of the opioid peptide dynorphin.

Personality

Personality traits are individual differences in general patterns of thoughts, feelings, and behavior that form the core of an individual's nature. Although it is known that both genetic and environmental factors as well as interactions between them contribute to the development of one's personality in a complex manner, the stable core of a person's nature—the behavioral continuity that makes someone unique, recognizable, and predictable—is owned to over 80% to genetic factors (McGue, Bacon, & Lykken, 1993). In the following, we will provide an overview of the current knowledge regarding genetic influences on personality.

Extraversion and introversion are part of one-dimensional continuum of the “big 5” personality traits. Extraversion is linked to positive emotionality and describes people who are outgoing and fun loving and who love to socialize, whereas introverts have lower social engagement and energy levels than extroverts and prefer to spend time alone. Whether someone is more extraverted or introverted seems to be partly determined by variations in the BDNF Val66Met polymorphism and the COMT Val158Met polymorphism coding dopaminergic activity in the brain, as recent genome-wide association studies revealed.

Neuroticism, the tendency to experience negative emotions, such as anger, anxiety, or depression, appears to be linked to a variety of genetic influences. This personality trait has been associated with low BDNF serum concentrations and variations in the BDNF Val66Met polymorphism as well as with interactions between BDNF Val66Met and the dopamine D2 receptor gene (DRD2)/ANKK1 and the serotonin transporter promoter polymorphism 5-HTTLPR. A meta-analysis summarizing 26 genetic association studies confirmed a significant association between 5-HTTLPR and neuroticism, demonstrating that individual differences in neuroticism are linked to serotonin transmission in the brain. Moreover, high neuroticism and low extraversion have been linked to several functional variants of the COMT gene, indicating an association of these two traits with dopaminergic activity.

Harm avoidance, a further personality trait that is characterized by excessive worrying and fear of uncertainty, has been found to be influenced by a combination of the BDNF Val66Met and the dopamine-coding DRD2/ANKK1 Taq1a polymorphisms. Moreover, higher levels of harm avoidance in women have been associated with the neuropeptide oxytocin as well as with MAOA, an enzyme encoding the MAOA gene catalyzing serotonin, DA, and norepinephrine.

Neuroticism, harm avoidance, and low levels of extraversion belong to the anxiety-related personality traits, which—in extreme form—may predispose individuals to develop psychopathological conditions. Meta-analytic evidence on the serotonin-coding 5-HTTLPR polymorphism and psychiatric disorders suggests that the relationship between 5-HTTLPR and anxiety-related personality traits is mediated by hyperreactivity of the amygdala during the processing of emotions. Particularly the S allele of the 5-HTTLPR polymorphism appears to be the “risk allele” for a variety of mood disorders, such as anxiety, depression, and bipolar disorder, putatively due to decreased transcriptional activity of the 5-HTTLPR S allele. In contrast, increased transcriptional activity of the 5-HTTLPR L allele has been associated with nicotine dependence and attention-deficit hyperactivity disorder (ADHD). Not only the development of psychiatric disorders but also their treatment seems to be partially dependent upon the 5-HTTLPR polymorphism: treatment responses in disorders such as posttraumatic stress disorder (PTSD), major depressive disorder, and alcohol dependence reportedly vary by 5-HTTLPR allele, suggesting a significant influence of the 5-HTTLPR polymorphism on treatment success.

A further personality trait describing how a person deals with feelings is alexithymia. Alexithymia characterizes individual differences in the ability to consciously experience and

regulate one's emotions. The more alexithymic an individual, the more difficulty they will experience identifying, describing, and verbalizing their feelings to others. Similarly to anxiety-related personality traits, alexithymia is linked to aberrant functioning and structure of the amygdala and other brain areas involved in emotion processing and thus constitutes a further risk factor for a range of psychiatric conditions. Preliminary evidence suggests that alexithymia may be linked to the serotonin polymorphism 5-HTTLPR (with carriers of the L/L allele having significantly higher alexithymia levels than S/S and L/S carriers) and to the dopamine-coding COMT Val108/158Met polymorphism (with carriers of the Val/Val genotype having significantly higher alexithymia levels than Met/Met or Met/Val carriers). Furthermore, an interaction between BDNF and the dopamine DRD2/ANKK1 gene has been reported to contribute to individual differences in alexithymia. However, these findings were based on rather small sample sizes and thus warrant replication in future research.

As opposed to alexithymia, which refers to one's own emotions, trait empathy describes individual differences in people's sensitivity to the feelings of others, that is, their capacity to share and understand other persons' feelings. Many studies have linked empathic abilities to oxytocin, a neuropeptide implicated in a wide range of social behaviors. Individuals homozygous for the G allele of OXTR SNP rs53576 exhibit greater empathy for others and show more activity in their own pain-related brain areas when seeing others suffering. Moreover, cognitive empathy (the capacity to understand another's perspective or mental state) and affective empathy (the ability to respond with an appropriate emotion to another's mental state) have been linked to different SNPs of OXTR, suggesting on the one hand an involvement of several OXTR SNPs in empathy and on the other hand a genetically differential disposition for cognitive and affective empathic abilities. In addition, this disposition seems to be influenced by gender: more empathic concern for others, an aspect of affective empathy, appears to be significantly linked to OXTR SNP rs53576 in women, but in men, this association may be weaker or even absent. Moving beyond genetic association studies, treatment studies intranasally administering oxytocin indicated some promise of this neuropeptide in treating psychiatric symptoms involving deficits in social functioning, such as autism, social anxiety disorders, borderline personality disorder (BPD), and schizophrenia, possibly by increasing the saliency of social stimuli, thus helping social attunement. Besides oxytocin, other genes appearing to influence human empathic abilities are the BDNF Val66Met polymorphism, which correlates with self-reported empathy; the serotonin transporter polymorphism 5-HTTLPR, which has been linked to levels of emotional reactivity (affective empathy); and the dopamine D4 receptor gene (DRD4 exon 3 polymorphism), which has been related to cognitive empathy.

Impulsivity, antisocial behavior, aggression, and violence form a group of negative personality traits, which have been linked to the MAOA gene located on the X chromosome, giving rise to this gene being popularly referred to as the "warrior gene." This term is based on repeated observations that the low-activity MAOA genotype in men, causing MAO deficiency, is associated with offending behavior, conduct problems, and hostility and that male carriers of this genotype who experienced early abuse are more prone to exhibiting antisocial behaviors throughout their lifetime. A recent meta-analysis on the 30-base pair variable number of tandem repeats of MAOA-uVNTR confirmed the link between the MAOA gene and antisocial behavior in humans. Regarding the question whether the effect of the MAOA gene on antisocial and aggressive behavior is driven by modulations in the DA or serotonin system, recent research suggests an inconclusive role of DA release in relation to MAOA. In contrast, a disrupted serotonergic system linked to the low functioning MAOA genotype has been concluded to predispose individuals to aggressive behavior by increasing impulsive reactivity to negative affect.

The important role of the serotonin system in impulsivity, antisocial behavior, and aggression is corroborated by other studies investigating the relationship between serotonin and psychiatric disorders marked by such behaviors. Several studies showed that the serotonin 5-HTTLPR polymorphism predisposes individuals not only to mood disorders such as anxiety and depression as addressed above but also to psychiatric problems involving impulsivity and aggressive behaviors, such as BPD. Children and teenagers in the ages of 9–15 years, who are carriers of the two S alleles of the serotonin 5-HTTLPR polymorphism, have the highest levels of BPD traits, even when controlling for depressive symptoms, providing evidence for a developmental etiological risk for BPD among youth. Such findings were replicated in adults with BPD, showing the carriers of the two S alleles of 5-HTTLPR suffered from the most severe affective symptoms of BPD.

In summary, research has linked a number of candidate genes to personality traits and the risk for developing psychiatric illness. However, no known gene is either necessary or sufficient to produce mental disease. Instead, there are many susceptibility genes with small effects, each increasing an individual's risk of illness to a small percentage. This is illustrated, for instance, by a recent genome-wide association study of 36,989 cases by the Schizophrenia Working Group of the Psychiatric Genomics Consortium, which found that 108 genetic loci were involved in the heritability of schizophrenia. Moreover, genes interact with each other and with environmental factors, and phenotypes are thus more than the sum of independent genetic and environmental factors. Certain life events may interact with brain vulnerabilities created by particular genes to produce behavioral patterns. For instance, a recent study aiming to identify genetic contributions to delinquency in teenagers found that such behaviors were not linked to one specific gene and also not to gene×gene interactions, but instead to four-way interactions between three genes (BDNF Val66Met, 5-HTTLPR, MAOA) and the environmental risk factors family conflict and sexual abuse, suggesting that genotypes per se do not confer risk for delinquent behaviors of the teenagers but rather alter their susceptibility to environmental factors. Importantly, this was not only true for negative but for positive environmental factors as well: interactions between the same genes and a positive child–parent relationship were associated with the lowest delinquency scores, highlighting the power of a positive environment on human behavior and demonstrating that it is ultimately the interaction with the environment a person is surrounded with that eventually determines their behavior, despite the presence of potential risk genes.

Conclusion

Despite their limitations, candidate gene and genome-wide association studies have demonstrated their potential in helping scientists understand the effects of changes in our genetic endowment on brain functions and systems on various aspects of human cognition and behavior as well as their potential as risk factors for a range of diseases. With the costs of full genomic sequencing becoming more and more affordable, the collection of big sets of genomics data and their integration with behavioral, environmental, clinical, and functional and structural neuroimaging data will become possible in the near future. Together with modern technologies of “big data” processing, analysis, sharing, and visualization, such future studies will enable us to move to another level of understanding the highly complex patterns of interactions between multiple genes and between our genetic disposition and the environment in which we grow up and surround ourselves with. Understanding the complex patterns of genetics and behavior will provide indispensable knowledge regarding the workings of the human mind and the mechanisms underlying our mental health.

Author Biographies

Mikhail Votinov is a postdoctoral researcher at Research Center Jülich (Germany) in the Institute of Neuroscience and Medicine (INM-10). He received his PhD in neuroimaging and cognitive neuroscience from Kyoto University (Japan), Faculty of Medicine. His research investigates genetic and hormonal influences on the neural mechanisms underlying reward processing, decision making, and social interactions.

Katharina Sophia Goerlich is a postdoctoral researcher at RWTH Aachen University (Germany) in the Department of Psychiatry, Psychotherapy, and Psychosomatics. She received her PhD in neuropsychiatry from the University of Groningen (Netherlands), Faculty of Medical Science. Her research investigates the neural mechanisms underlying the processing of social and emotional information in the brain and in what way these mechanisms are influenced by personality differences and psychopathology.

Reference

McGue, M., Bacon, S., & Lykken, D. T. (1993). Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental psychology*, 29(1), 96.

Suggested Reading

- Anokhin, A. P., Golosheykin, S., Grant, J. D., & Heath, A. C. (2011). Heritability of delay discounting in adolescence: A longitudinal twin study. *Behavior Genetics*, 41(2), 175–183.
- Aspé-Sánchez, M., Moreno, M., Rivera, M. I., Rossi, A., & Ewer, J. (2015). Oxytocin and vasopressin receptor gene polymorphisms: Role in social and psychiatric traits. *Frontiers in Neuroscience*, 9, 510. doi:10.3389/fnins.2015.00510
- Bevilacqua, L., & Goldman, D. (2013). Genetics of impulsive behaviour. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 368(1615), 20120380.
- Corral-Frías, N. S., Pizzagalli, D. A., Carré, J. M., Michalski, L. J., Nikolova, Y. S., Perlis, R. H., ... Haddad, S. (2016). COMT Val158Met genotype is associated with reward learning: A replication study and meta-analysis. *Genes, Brain and Behavior*, 15(5), 503–513.
- Dincheva, I., Glatt, C. E., & Lee, F. S. (2012). Impact of the BDNF Val66Met polymorphism on cognition implications for behavioral genetics. *The Neuroscientist*, 18(5), 439–451.
- Dreber, A., Apicella, C. L., Eisenberg, D. T., Garcia, J. R., Zamore, R. S., Lum, J. K., & Campbell, B. (2009). The 7R polymorphism in the dopamine receptor D 4 gene (DRD4) is associated with financial risk taking in men. *Evolution and Human Behavior*, 30(2), 85–92.
- El Haj, M., Antoine, P., Amouyel, P., Lambert, J. C., Pasquier, F., & Kapogiannis, D. (2016). Apolipoprotein E (APOE) ε4 and episodic memory decline in Alzheimer's disease: A review. *Ageing Research Reviews*, 27, 15–22.
- Faraone, S. V., Spencer, T. J., Madras, B. K., Zhang-James, Y., & Biederman, J. (2014). Functional effects of dopamine transporter gene genotypes on in vivo dopamine transporter functioning: A meta-analysis. *Molecular Psychiatry*, 19(8), 880–889.
- Ficks, C. A., & Waldman, I. D. (2014). Candidate genes for aggression and antisocial behavior: A meta-analysis of association studies of the 5HTTLPR and MAOA-uVNTR. *Behavior Genetics*, 44(5), 427–444.
- Frank, M. J., & Fossella, J. A. (2011). Neurogenetics and pharmacology of learning, motivation, and cognition. *Neuropsychopharmacology*, 36(1), 133–152.
- Frustaci, A., Pozzi, G., Gianfagna, F., Manzoli, L., & Boccia, S. (2008). Meta-analysis of the brain-derived neurotrophic factor gene (BDNF) Val66Met polymorphism in anxiety disorders and anxiety-related personality traits. *Neuropsychobiology*, 58(3–4), 163–170.

- Garcia-Garcia, M., Barcelo, F., Clemente, I. C., & Escera, C. (2010). The role of the dopamine transporter DAT1 genotype on the neural correlates of cognitive flexibility. *European Journal of Neuroscience*, 31(4), 754–760.
- Goerlich-Dobre, K. S., Lamm, C., Pripfl, J., Habel, U., & Votinov, M. (2015). The left amygdala: A shared substrate of alexithymia and empathy. *NeuroImage*, 122, 20–32.
- Goerlich-Dobre, K. S., Votinov, M., Habel, U., Pripfl, J., & Lamm, C. (2015). Neuroanatomical profiles of alexithymia dimensions and subtypes. *Human Brain Mapping*, 36(10), 3805–3818.
- Gyollai, Á., D Griffiths, M., Barta, C., Vereczkei, A., Urbán, R., Kun, B., ... Demetrovics, Z. (2014). The genetics of problem and pathological gambling: A systematic review. *Current Pharmaceutical Design*, 20(25), 3993–3999.
- Ham, B. J., Lee, M. S., Lee, Y. M., Kim, M. K., Choi, M. J., Oh, K. S., ... Choi, I. G. (2005). Association between the catechol O-methyltransferase Val108/158Met polymorphism and alexithymia. *Neuropsychobiology*, 52(3), 151–154.
- Hankin, B., Barrocas, A. L., Jenness, J., Oppenheimer, C., Badanes, L., Abela, J., ... Smolen, A. (2011). Association between 5-HTTLPR and borderline personality disorder traits among youth. *Frontiers in Psychiatry*, 2, 6.
- Keller, M. C., Coventry, W. L., Heath, A. C., & Martin, N. G. (2005). Widespread evidence for non-additive genetic variation in Cloninger's and Eysenck's personality dimensions using a twin plus sibling design. *Behavior Genetics*, 35(6), 707–721.
- Kenna, G. A., Roder-Hanna, N., Leggio, L., Zywiak, W. H., Clifford, J., Edwards, S., ... Swift, R. M. (2012). Association of the 5-HTT gene-linked promoter region (5-HTTLPR) polymorphism with psychiatric disorders: Review of psychopathology and pharmacotherapy. *Pharmacogenomics and Personalized Medicine*, 5, 19–35. doi:10.2147/PGPM.S23462
- Kreek, M. J., Nielsen, D. A., Butelman, E. R., & LaForge, K. S. (2005). Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. *Nature Neuroscience*, 8(11), 1450–1457.
- Le Foll, B., Gallo, A., Le Strat, Y., Lu, L., & Gorwood, P. (2009). Genetics of dopamine receptors and drug addiction: A comprehensive review. *Behavioural Pharmacology*, 20(1), 1–17.
- Lehto, K., Akkermann, K., Parik, J., Veidebaum, T., & Harro, J. (2013). Effect of COMT Val158Met polymorphism on personality traits and educational attainment in a longitudinal population representative study. *European Psychiatry*, 28(8), 492–498.
- Lind, P. A., Zhu, G., Montgomery, G. W., Madden, P. A., Heath, A. C., Martin, N. G., & Slutske, W. S. (2013). Genome-wide association study of a quantitative disordered gambling trait. *Addiction Biology*, 18(3), 511–522.
- Liu, C. C., Kanekiyo, T., Xu, H., & Bu, G. (2013). Apolipoprotein E and Alzheimer disease: Risk, mechanisms and therapy. *Nature Reviews Neurology*, 9(2), 106–118.
- Meyer-Lindenberg, A., Straub, R. E., Lipska, B. K., Verchinski, B. A., Goldberg, T., Callicott, J. H., ... Kleinman, J. E. (2007). Genetic evidence implicating DARPP-32 in human frontostriatal structure, function, and cognition. *The Journal of Clinical Investigation*, 117(3), 672–682.
- Montag, C., Jurkiewicz, M., & Reuter, M. (2012). The role of the catechol-O-methyltransferase (COMT) gene in personality and related psychopathological disorders. *CNS & Neurological Disorders-Drug Targets*, 11(3), 236–250.
- Montag, C., Markett, S., Basten, U., Stelzel, C., Fiebach, C., Canli, T., & Reuter, M. (2010). Epistasis of the DRD2/ANKK1 Taq Ia and the BDNF Val66Met polymorphism impacts novelty seeking and harm avoidance. *Neuropsychopharmacology*, 35(9), 1860–1867.
- Peciña, M., Mickey, B. J., Love, T., Wang, H., Langenecker, S. A., Hodgkinson, C., ... Stohler, C. S. (2013). DRD2 polymorphisms modulate reward and emotion processing, dopamine neurotransmission and openness to experience. *Cortex*, 49(3), 877–890.
- Ripke, S., Neale, B. M., Corvin, A., Walters, J. T., Farh, K. H., Holmans, P. A., ... Pers, T. H. (2014). Biological insights from 108 schizophrenia-associated genetic loci. *Nature*, 511(7510), 421.
- Savitz, J., Solms, M., & Ramesar, R. (2006). The molecular genetics of cognition: Dopamine, COMT and BDNF. *Genes, Brain and Behavior*, 5(4), 311–328.

- Schlüter, T., Winz, O., Henkel, K., Eggermann, T., Mohammadkhani-Shali, S., Dietrich, C., ... Piel, M. (2016). MAOA-VNTR polymorphism modulates context-dependent dopamine release and aggressive behavior in males. *NeuroImage*, 125, 378–385.
- Schwarzer, C. (2009). 30 years of dynorphins—New insights on their functions in neuropsychiatric diseases. *Pharmacology & Therapeutics*, 123(3), 353–370.
- Sen, S., Burmeister, M., & Ghosh, D. (2004). Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality traits. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 127(1), 85–89.
- Stein, M. B., Fallin, M. D., Schork, N. J., & Gelernter, J. (2005). COMT polymorphisms and anxiety-related personality traits. *Neuropsychopharmacology*, 30(11), 2092–2102.
- Terracciano, A., Sanna, S., Uda, M., Deiana, B., Usala, G., Busonero, F., ... Distel, M. A. (2010). Genome-wide association scan for five major dimensions of personality. *Molecular Psychiatry*, 15(6), 647–656.
- van der Velde, J., Servaas, M. N., Goerlich, K. S., Bruggeman, R., Horton, P., Costafreda, S. G., & Aleman, A. (2013). Neural correlates of alexithymia: A meta-analysis of emotion processing studies. *Neuroscience & Biobehavioral Reviews*, 37(8), 1774–1785.
- Votinov, M., Pripfl, J., Windischberger, C., Kalcher, K., Zimprich, A., Zimprich, F., ... Sailer, U. (2014). A genetic polymorphism of the endogenous opioid dynorphin modulates monetary reward anticipation in the corticostriatal loop. *PLoS One*, 9(2), e89954.
- Votinov, M., Pripfl, J., Windischberger, C., Moser, E., Sailer, U., & Lamm, C. (2015). A functional polymorphism in the prodynorphin gene affects cognitive flexibility and brain activation during reversal learning. *Frontiers in Behavioral Neuroscience*, 9, 172.
- Wagels, L., Votinov, M., Radke, S., Clemens, B., Montag, C., Jung, S., & Habel, U. (2017). Blunted insula activation reflects increased risk and reward seeking as an interaction of testosterone administration and the MAOA polymorphism. *Human Brain Mapping*. doi:10.1002/hbm.23685
- Yacubian, J., Sommer, T., Schroeder, K., Gläscher, J., Kalisch, R., Leuenberger, B., ... Büchel, C. (2007). Gene–gene interaction associated with neural reward sensitivity. *Proceedings of the National Academy of Sciences*, 104(19), 8125–8130.

Epigenetics in Developmental Disorders

Tiffany S. Doherty and Tania L. Roth

Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA

Introduction

An array of disorders find their origin in early development and profoundly affect brain function and cognitive health throughout the lifetime. Several years of research have contributed to our understanding of how these disorders arise, and epigenetic modifications have emerged as a primary mechanism in many cases. Epigenetics refers to chemical marking of the DNA that changes transcriptional and translational outcomes (i.e., gene and protein expression) without changing the underlying sequence of DNA. This marking can be accomplished in more than one manner, and each process involves an assortment of molecular characters.

One of the best understood epigenetic mechanisms is DNA methylation. Generally associated with silencing of genes, methylation is carried out by DNA methyltransferases (DNMTs). DNMTs attach methyl groups to cytosines, typically at cytosine–guanine pairings. These methyl groups are most well known for blocking transcription factor access to DNA and for their ability to recruit repressor proteins (Moore, Le, & Fan, 2013). One of these proteins, methyl-CpG-binding protein 2 (MeCP2), possesses a transcriptional repression domain that interacts with the Sin3/HDAC complex (Nan et al., 1998). The *SIN3* gene encodes a transcriptional regulatory protein whereas histone deacetylases (HDACs) are enzymes that remove acetyl groups from proteins, including histones. MeCP2 can also recruit coactivators such as CREB1 (Chahrour et al., 2008) though less is known about this occurrence.

Histones are the proteins around which DNA is packaged, and modifications to these histones are another type of epigenetic mechanism. The two best-studied histone modifications involve the addition or removal of acetyl and/or methyl groups, that is, histone acetylation and histone methylation. These alterations determine whether the DNA/histone complex (chromatin) is accessible (euchromatin) or inaccessible (heterochromatin) to transcription. The addition of acetyl groups to histones is associated with euchromatin (Grunstein, 1997;

Hebbes, Thorne, & Crane-Robinson, 1988) whereas the effects of histone methylation depend on the location and number of methyl groups. Histone acetylation is catalyzed by histone acetyltransferases (Grunstein, 1997) and is reversed by HDACs (De Ruijter, Van Gennip, Caron, Kemp, & van Kuilenburg, 2003). Histone methylation is catalyzed by histone methyltransferases and is reversed by histone demethylases (Bannister & Kouzarides, 2005).

Epigenetic mechanisms are responsible for a large portion of developmental events including critical processes such as cell fate and germ cell imprinting. Developmental epigenetic changes are tightly controlled as typical patterns are necessary for optimal development and continued function. Thus, many developmental disorders are attributed, at least in part, to atypical epigenetic activity.

Fragile X Syndrome

The clinical features of fragile X syndrome range from slight cognitive deficits to severe mental retardation. The disorder preferentially affects males due to its X-linked inheritance (i.e., the mutation responsible for the phenotype exists on the X chromosome, of which males inherit only one). In the case of fragile X, DNA methylation is responsible for silencing the fragile X mental retardation 1 gene, or *FMR1* (Sutcliffe et al., 1992). Normally, this gene encompasses a small number of repeats of a specific DNA segment (the trinucleotide CGG). However, the genome of individuals with the full mutation responsible for fragile X syndrome can contain between 200 and 1,000 repeats of this segment. This results in hypermethylation of *FMR1* resulting in a lack of the FMR protein and subsequent alterations of synaptic structure and function. These alterations underlie the cognitive difficulties associated with the disorder, likely due to the regulatory role the FMR protein plays in translation, especially at the synapse (Zalfa et al., 2003).

Angelman and Prader–Willi Syndrome

The clinical features of Angelman Syndrome include intellectual impairment, jerky movements, seizures, and joyful demeanor (Williams, Driscoll, & Dagli, 2010). This disease is attributed to genomic imprinting, a phenomenon responsible for the expression of certain genes in a parent-of-origin manner accomplished via epigenetic means (Reik & Walter, 2001). In such cases, either the allele inherited from the mother or the allele inherited by the father is imprinted, leading to expression only of the non-imprinted parent allele. In Angelman syndrome, the paternally inherited allele of ubiquitin-protein ligase E3A (*UBE3A*) is silenced via methylation, leaving expression to the maternally inherited allele. *UBE3A* is involved in cell maintenance by producing an enzyme involved in the degradation of unwanted or unnecessary proteins. In Angelman syndrome however, the maternally inherited allele is typically lost to a chromosomal deletion, leaving no expression to take place (Kishino, Lalonde, & Wagstaff, 1997). Though far less common, Angelman can be caused by imprinting defects (i.e., methylation) of the maternal allele, likewise disrupting expression (Bürger et al., 1997).

The clinical features of Prader–Willi syndrome include intellectual impairment and obesity caused by constant hunger. Like Angelman syndrome, Prader–Willi syndrome is due to genomic imprinting, but in this case maternal alleles are silenced by hypermethylation, and

paternal alleles have been deleted (or in rare cases methylated), leaving no expression of several genes along a section of chromosome 15 (Cassidy, Schwartz, Miller, & Driscoll, 2012).

Rett Syndrome

The clinical features of Rett syndrome include a small head due to improper brain development, abnormal hand movements, cognitive deficits, impaired social and language abilities, breathing problems, and coordination issues. The syndrome preferentially affects females and is the result of mutations to MeCP2, located on the X chromosome (Amir et al., 1999). It is the most common cause of mental retardation in females; affected males rarely survive past birth. Both nonsense and missense mutations have been uncovered. The former involve incomplete protein formation while the latter involve nucleotide deletions or substitutions. MeCP2 missense mutations found in Rett syndrome may reduce or halt methyl-CpG binding, result in abnormal protein folding, or disturb protein interactions within the SIN3A/HDAC silencing complex (Wan et al., 1999). Various phenotypes are associated with each mutation and range from mild cognitive deficits to the classic Rett symptoms described above.

Autism Spectrum Disorders

Some clinical features of autism spectrum disorders (ASD) include impairments in language and social skills as well as highly repetitive behaviors. While genetic factors have been strongly associated with this disorder, evidence increasingly supports a significant role for epigenetic modifications as either biomarkers or causal mechanisms. Differentially methylated sites have been reported for several genes in relation to ASDs. A reduction in the methyl-binding protein MeCP2, which is generally associated with decreased transcription, has been reported in the frontal cortex of autistic brains (Nagarajan, Hogart, Gwye, Martin, & LaSalle, 2006). This may be indicative of overactive gene expression in the frontal cortex. In addition, the manner in which MeCP2 is silenced is most likely epigenetic as the decreased expression in these ASD brains is accompanied by increased promoter methylation. Disrupted patterns of methyl-binding domain genes 3 and 4 have also been reported in autistic individuals (Cukier et al., 2010).

In the cerebellum of ASD patients, MeCP2 binding to glutamate decarboxylase 1 (*GAD1* or *GAD67*) and reelin (*RELN*) is significantly increased, a change that presumably (though not definitely) decreases expression (Zhubi et al., 2014). The primary inhibitory neurotransmitter gamma-aminobutyric acid (GABA) is produced from glutamate by an enzyme encoded by *GAD67*. *RELN* is heavily involved in processes of synaptic plasticity and also plays a role in neuronal migration during brain development. Both of these genes have suggested roles in ASD etiology.

Epigenetic regulation of the oxytocin receptor gene (*OXTR*) may be another critical mechanism underlying ASD etiology. Oxytocin is a known promoter of social behavior, and *OXTR* hypermethylation has been reported in human ASD brains (Gregory et al., 2009). Moreover, hypermethylation was reported to take place at more *OXTR* loci in males than in females, a compelling piece of evidence for a disorder that preferentially affects males (Gregory et al., 2009).

The scaffolding protein SHANK3 has been heavily implicated in autism etiology. This protein is found in high levels at neuronal postsynaptic densities where they play a critical role

in synaptic function (Wang et al., 2011). Several CpG sites are located within the *SHANK* genes, but *SHANK3* expression in particular appears to be highly epigenetically regulated (Beri et al., 2007). Examination of postmortem brain tissue of ASD patients revealed significantly higher methylation levels at multiple *SHANK3* CpG islands (Zhu et al., 2013), presumably indicating a decrease in expression and thus in synaptic function.

Finally, investigation of the maternal methylome has provided some correlative evidence for the contribution of the prenatal epigenetic landscape to the development of ASDs. Overall decreases in methylation levels as well as the methylation precursors homocysteine and *S*-adenosylhomocysteine (SAH) have been reported in mothers of autistic children (James et al., 2008).

Fetal Alcohol Spectrum Disorder

The clinical features of fetal alcohol spectrum disorders (FASD) include a range of atypical growth patterns and facial features and cognitive deficits resulting from disturbance of the central nervous system. This disorder is a result of alcohol consumption during the gestational period, an act that disrupts many essential developmental processes. Methyl group metabolism is highly affected by alcohol consumption (Schalinske & Nieman, 2005). Thus, bidirectional disruption of epigenetic patterns is hypothesized to be one manner through which alcohol exerts its teratogenic effects. It is difficult to pinpoint a specific epigenetic abnormality at fault as they vary based on factors such as the time during gestation when alcohol consumption occurs.

Altered methylation is seen at several genes associated with cellular and developmental processes in mouse embryos exposed to alcohol during early neurulation, the folding process whereby the neural tube forms from the neural plate. In alcohol-exposed embryos expressing neural tube defects, the number of genes exhibiting increased levels of methylation on chromosomes 10 and X increased more than 10-fold (Liu, Balaraman, Wang, Nephew, & Zhou, 2009). Perinatal exposure to alcohol significantly decreases DNMT activity and expression in the rodent hippocampus (Perkins, Lehmann, Lawrence, & Kelly, 2013), providing a mechanism by which methylation alterations occur in response to alcohol. Evidence has also emerged for the role of histone modifications following developmental alcohol exposure. Increased histone methyltransferase activity and alterations of both repressive and activating histone marks occur in the early alcohol-exposed rodent brain (Subbanna et al., 2013, 2014).

In male and female rodent offspring exposed to alcohol during gestation, methylation of the proopiomelanocortin (*POMC*) gene is significantly increased (Govorko, Bekdash, Zhang, & Sarkar, 2012). The *POMC* gene codes for the protein proopiomelanocortin, which is used to form peptides like adrenocorticotrophic hormone (ACTH). These peptides serve various functions throughout the body by influencing signaling pathways via protein binding. These signaling pathways are critical to systems involved in stress response, immune function, and reward. Alcohol exposure and craving are associated with *POMC* dysregulation (Muschler et al., 2010; Zhou et al., 2000), and evidence points to epigenetic alterations as the mechanism underlying these associations.

Gain-of-function studies have also provided support for epigenetic mechanisms of FASD. Typical epigenetic patterns, including the presence of methylation at specific locations in the genome, are critical to developmental processes. Alcohol consumption decreases levels of the methyl donor *S*-adenosylmethionine (SAM) by inhibiting the absorption of folic acid. Methylation of DNA is dependent on SAM and is thus disrupted by alcohol consumption,

leading to aberrant developmental trajectories. Choline supplementation may rescue some of these trajectories. For example, choline administration attenuates learning impairments in alcohol-exposed rodents (Ryan, Williams, & Thomas, 2008). Given that choline is one of the nutrients from which methyl groups are derived (Niculescu & Zeisel, 2002), it is hypothesized that altered DNA methylation is the mechanism of behavioral rescue, a hypothesis that has continued to be supported. Alcohol-induced epigenetic modifications in the hippocampus and frontal cortex (Otero, Thomas, Saski, Xia, & Kelly, 2012) and hypothalamus (Bekdash, Zhang, & Sarkar, 2013) are also attenuated with choline supplementation.

Author Biographies

Tiffany S. Doherty is a doctoral candidate in the Department of Psychological and Brain Sciences at the University of Delaware, where she conducts research in the lab of Dr. Tania Roth. Her dissertation research is focused on the utility of drug intervention in preventing aberrant epigenetic and behavioral trajectories following developmental exposure to adversity.

Dr. Tania L. Roth is an associate professor and director of Graduate Education in Psychological and Brain Sciences at the University of Delaware, where she teaches upper-level and graduate courses in psychology and neuroscience. Her research program is focused on defining epigenetic mechanisms responsible for environmental influences on CNS gene activity, development of behavior, and psychiatric disorder.

References

- Amir, R. E., Van den Veyver, I. B., Wan, M., Tran, C. Q., Francke, U., & Zoghbi, H. Y. (1999). Rett syndrome is caused by mutations in X-linked MECP2, encoding methyl-CpG-binding protein 2. *Nature Genetics*, 23(2), 185–188.
- Bannister, A. J., & Kouzarides, T. (2005). Reversing histone methylation. [10.1038/nature04048]. *Nature*, 436(7054), 1103–1106.
- Bekdash, R. A., Zhang, C., & Sarkar, D. K. (2013). Gestational choline supplementation normalized fetal alcohol-induced alterations in histone modifications, DNA methylation, and proopiomelanocortin (POMC) gene expression in β -endorphin-producing POMC neurons of the hypothalamus. *Alcoholism: Clinical and Experimental Research*, 37(7), 1133–1142.
- Beri, S., Tonna, N., Menozzi, G., Bonaglia, M. C., Sala, C., & Giorda, R. (2007). DNA methylation regulates tissue-specific expression of Shank3. *Journal of Neurochemistry*, 101(5), 1380–1391.
- Bürger, J., Buiting, K., Dittrich, B., Groß, S., Lich, C., Sperling, K., ... Reis, A. (1997). Different mechanisms and recurrence risks of imprinting defects in Angelman syndrome. *The American Journal of Human Genetics*, 61(1), 88–93.
- Cassidy, S. B., Schwartz, S., Miller, J. L., & Driscoll, D. J. (2012). Prader-Willi syndrome. *Genetics in Medicine*, 14(1), 10–26.
- Chahrouh, M., Jung, S. Y., Shaw, C., Zhou, X., Wong, S. T. C., Qin, J., & Zoghbi, H. Y. (2008). MeCP2, a key contributor to neurological disease, activates and represses transcription. *Science*, 320(5880), 1224–1229. doi:10.1126/science.1153252
- Cukier, H. N., Rabionet, R., Konidari, I., Rayner-Evans, M. Y., Baltos, M. L., Wright, H. H., ... Pericak-Vance, M. A. (2010). Novel variants identified in methyl-CpG-binding domain genes in autistic individuals. *Neurogenetics*, 11(3), 291–303.
- De Ruijter, A., Van Gennip, A., Caron, H., Kemp, S., & van Kuilenburg, A. (2003). Histone deacetylases (HDACs): characterization of the classical HDAC family. *The Biochemical Journal*, 370, 737–749.

- Govorko, D., Bekdash, R. A., Zhang, C., & Sarkar, D. K. (2012). Male germline transmits fetal alcohol adverse effect on hypothalamic proopiomelanocortin gene across generations. *Biological Psychiatry*, 72(5), 378–388. doi:10.1016/j.biopsych.2012.04.006
- Gregory, S. G., Connelly, J. J., Towers, A. J., Johnson, J., Biscocho, D., Markunas, C. A., ... Ellis, P. (2009). Genomic and epigenetic evidence for oxytocin receptor deficiency in autism. *BMC Medicine*, 7(1), 1.
- Grunstein, M. (1997). Histone acetylation in chromatin structure and transcription. [10.1038/38664]. *Nature*, 389(6649), 349–352.
- Hebbes, T. R., Thorne, A. W., & Crane-Robinson, C. (1988). A direct link between core histone acetylation and transcriptionally active chromatin. *The EMBO Journal*, 7(5), 1395.
- James, S. J., Melnyk, S., Jernigan, S., Hubanks, A., Rose, S., & Gaylor, D. W. (2008). Abnormal transmethylation/transsulfuration metabolism and DNA hypomethylation among parents of children with autism. *Journal of Autism and Developmental Disorders*, 38(10), 1966–1975.
- Kishino, T., Lalande, M., & Wagstaff, J. (1997). UBE3A/E6-AP mutations cause Angelman syndrome. *Nature Genetics*, 15(1), 70–73.
- Liu, Y., Balaraman, Y., Wang, G., Nephew, K. P., & Zhou, F. C. (2009). Alcohol exposure alters DNA methylation profiles in mouse embryos at early neurulation. *Epigenetics*, 4(7), 500–511.
- Moore, L., Le, T., & Fan, G. (2013). DNA methylation and its basic function. *Neuropsychopharmacology*, 38(1), 23–38.
- Muschler, M. A. N., Hillemecher, T., Kraus, C., Kornhuber, J., Bleich, S., & Frieeling, H. (2010). DNA methylation of the POMC gene promoter is associated with craving in alcohol dependence. *Journal of Neural Transmission*, 117(4), 513–519. doi:10.1007/s00702-010-0378-7
- Nagarajan, R., Hogart, A., Gwyne, Y., Martin, M. R., & LaSalle, J. M. (2006). Reduced MeCP2 expression is frequent in autism frontal cortex and correlates with aberrant MECP2 promoter methylation. *Epigenetics*, 1(4), 172–182.
- Nan, X., Ng, H.-H., Johnson, C. A., Laherty, C. D., Turner, B. M., Eisenman, R. N., & Bird, A. (1998). Transcriptional repression by the methyl-CpG-binding protein MeCP2 involves a histone deacetylase complex. [10.1038/30764]. *Nature*, 393(6683), 386–389.
- Niculescu, M. D., & Zeisel, S. H. (2002). Diet, methyl donors and DNA methylation: interactions between dietary folate, methionine and choline. *The Journal of Nutrition*, 132(8), 2333S–2335S.
- Otero, N. K., Thomas, J. D., Saski, C. A., Xia, X., & Kelly, S. J. (2012). Choline supplementation and DNA methylation in the hippocampus and prefrontal cortex of rats exposed to alcohol during development. *Alcoholism: Clinical and Experimental Research*, 36(10), 1701–1709.
- Perkins, A., Lehmann, C., Lawrence, R. C., & Kelly, S. J. (2013). Alcohol exposure during development: Impact on the epigenome. *International Journal of Developmental Neuroscience*, 31(6), 391–397. doi:10.1016/j.ijdevneu.2013.03.010
- Reik, W., & Walter, J. (2001). Genomic imprinting: Parental influence on the genome. [10.1038/35047554]. *Nature Reviews. Genetics*, 2(1), 21–32.
- Ryan, S. H., Williams, J. K., & Thomas, J. D. (2008). Choline supplementation attenuates learning deficits associated with neonatal alcohol exposure in the rat: Effects of varying the timing of choline administration. *Brain Research*, 1237, 91–100.
- Schalinske, K. L., & Nieman, K. M. (2005). Disruption of methyl group metabolism by ethanol. *Nutrition Reviews*, 63(11), 387–391.
- Subbanna, S., Nagre, N. N., Shivakumar, M., Umashathy, N. S., Psychoyos, D., & Basavarajappa, B. S. (2014). Ethanol induced acetylation of histone at G9a exon1 and G9a-mediated histone H3 dimethylation leads to neurodegeneration in neonatal mice. *Neuroscience*, 258, 422–432. doi:10.1016/j.neuroscience.2013.11.043
- Subbanna, S., Shivakumar, M., Umashathy, N. S., Saito, M., Mohan, P. S., Kumar, A., ... Basavarajappa, B. S. (2013). G9a-mediated histone methylation regulates ethanol-induced neurodegeneration in the neonatal mouse brain. *Neurobiology of Disease*, 54, 475–485.
- Sutcliffe, J. S., Nelson, D. L., Zhang, F., Pieretti, M., Caskey, C. T., Saxe, D., & Warren, S. T. (1992). DNA methylation represses FMR-1 transcription in fragile X syndrome. *Human Molecular Genetics*, 1(6), 397–400.

- Wan, M., Lee, S. S. J., Zhang, X., Houwink-Manville, I., Song, H.-R., Amir, R. E., ... Francke, U. (1999). Rett syndrome and beyond: Recurrent spontaneous and familial MECP2 mutations at CpG hotspots. *The American Journal of Human Genetics*, 65(6), 1520–1529. doi:10.1086/302690
- Wang, X., McCoy, P. A., Rodriguiz, R. M., Pan, Y., Je, H. S., Roberts, A. C., ... Jiang, Y.-h. (2011). Synaptic dysfunction and abnormal behaviors in mice lacking major isoforms of Shank3. *Human Molecular Genetics*. doi:10.1093/hmg/ddr212
- Williams, C. A., Driscoll, D. J., & Dagli, A. I. (2010). Clinical and genetic aspects of Angelman syndrome. *Genetics in Medicine*, 12(7), 385–395.
- Zalfa, F., Giorgi, M., Primerano, B., Moro, A., Di Penta, A., Reis, S., ... Bagni, C. (2003). The fragile X syndrome protein FMRP associates with BCL RNA and regulates the translation of specific mRNAs at synapses. *Cell*, 112(3), 317–327.
- Zhou, Y., Franck, J., Spangler, R., Maggos, C. E., Ho, A., & Kreek, M. J. (2000). Reduced hypothalamic POMC and anterior pituitary CRF1 receptor mRNA levels after acute, but not chronic, daily “binge” intragastric alcohol administration. *Alcoholism: Clinical and Experimental Research*, 24(10), 1575–1582.
- Zhu, L., Wang, X., Li, X.-L., Towers, A., Cao, X., Wang, P., ... Li, Y.-J. (2013). Epigenetic dysregulation of SHANK3 in brain tissues from individuals with autism spectrum disorders. *Human Molecular Genetics*, 23(6), ddt547.
- Zhubi, A., Chen, Y., Dong, E., Cook, E. H., Guidotti, A., & Grayson, D. R. (2014). Increased binding of MeCP2 to the GAD1 and RELN promoters may be mediated by an enrichment of 5-hmC in autism spectrum disorder (ASD) cerebellum. [Original Article]. *Translational Psychiatry*, 4, e349. doi:10.1038/tp.2013.123

Epigenetics in Behavior and Mental Health

Tiffany S. Doherty and Tania L. Roth

Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA

Introduction

Exposure to adversity during development significantly increases the risk of manifesting a mental health disorder. Examination of the consequences of early adversity in rodent and human models has brought epigenetic mechanisms to the forefront of adversity-induced mental health outcomes, with research continuously revealing a significant relationship between psychopathology and the epigenome following early life adversity.

Epigenetic mechanisms are chemical modifications of chromatin (the DNA/histone complex) that result in genetic modulation without altering the DNA sequence. The most heavily studied of these are DNA methylation and histone modifications.

DNA methylation occurs upon the addition of a methyl group to a cytosine base in the DNA sequence, typically at cytosine–guanine dinucleotides. This modification generally results in gene silencing via blockage of transcription factor access or recruitment of repressive proteins (Moore, Le, & Fan, 2013). DNA hydroxymethylation occurs via oxidation of methylated cytosines, a modification that may be involved in the process of demethylation but may also play a stable role in gene expression depending on the context in which it occurs (Kato & Iwamoto, 2014).

Histones, the proteins around which DNA is packaged, are subject to several posttranslational modifications. These include acetylation, methylation, phosphorylation, SUMOylation, and ubiquitylation. Acetylation and methylation are the best understood of these modifications and have thus far been the focus of studies on epigenetic mechanisms in mental health. Acetylation results in a more open chromatin structure and increased gene expression. Histone methylation has both activating and repressive effects on gene expression depending on the number and location of methyl groups.

Epigenetic regulators are enzymes responsible for the addition or removal of epigenetic markers. DNA methyltransferases (DNMTs) are responsible for the addition of methyl groups to DNA, and various demethylases (of which much less is currently known) are responsible for their removal (Chen, Wang, & Shen, 2013). Hydroxymethylation is catalyzed by the ten-eleven translocation (TET) family of proteins (Kato & Iwamoto, 2014). Histone acetyltransferases (HATs) and histone methyltransferases (HMTs) are responsible for the addition of acetyl and methyl groups to histones, respectively. The removal of these groups from histones is accomplished by histone deacetylases (HDACs) and histone demethylases (Bolger, Cohen, & Yao, 2006).

Epigenetic Consequences of Prenatal Stress

Both adverse and enriching experiences during gestation are capable of catalyzing epigenetic changes in the developing brain. These changes precede an array of behavioral alterations ranging from altered stress responses to deficits in cognitive performance. Epigenetic alterations affecting the stress response are generally found to interrupt function of the hypothalamic–pituitary–adrenal gland (HPA) axis and are known to occur as early as the gestational period. Upon exposure to a prenatal stressor, the enzyme responsible for protecting the fetus against maternally derived glucocorticoids, 11 β -hydroxysteroid dehydrogenase type 2 (*Hsd11b2*), is decreased. This decrease occurs alongside significantly increased promoter methylation and DNMT expression in the placenta (Peña, Monk, & Champagne, 2012). In other rodent models of prenatal stress, aberrant stress responses in adult offspring correspond to epigenetic alterations of genes coding for the stress-related glucocorticoid receptor (*GR*) and corticotropin-releasing factor (*Crf*) (Mueller & Bale, 2008).

Cognitive deficits resulting from stress exposure in the prenatal period are also correlated with epigenetic remodeling. Spatial memory impairments in adult male and female rats that were stressed while in utero are accompanied by significantly elevated levels of the epigenetic regulator DNMT1 and significantly decreased levels of histone acetylation in the hippocampus (Benoit, Rakic, & Frick, 2015). Neurotrophins, which promote healthy brain development and plasticity across the lifespan, are also epigenetically modified by gestational stress. For example, brain-derived neurotrophic factor (*Bdnf*) methylation is increased in adult offspring in a rodent model of prenatal stress, an epigenetic change accompanied by behavioral alterations in both male and female offspring (St-Cyr & McGowan, 2015).

The link between prenatal stress and mental health outcomes also appears to be epigenetic in nature. Following gestational restraint stress, mice displaying a schizophrenia-like phenotype exhibit epigenetic alterations of genes that have been implicated in schizophrenia (Matrisciano et al., 2013). In humans, mother-reported anxiety during pregnancy correlates with increased *11BHSD2* methylation in offspring, whereas mother-reported depressive symptoms during pregnancy correlate with increased *GR* methylation (Conradt, Lester, Appleton, Armstrong, & Marsit, 2013). Disrupted epigenetic patterns at these genes, both of which are involved in the stress response, have implications for behavioral outcomes relevant to mental health disorders. In mothers carrying a specific variant of the *MTHFR* gene, a gene that codes for an enzyme critical in the production of methionine, depressive symptoms during pregnancy are associated with epigenetic changes within the serotonin system (Devlin, Brain, Austin, & Oberlander, 2010). The serotonin transporter gene *SLC6A4*, implicated in many mental health disorders, exhibits decreased methylation in these mothers and their offspring.

Epigenetic Consequences of Postnatal Stress

Postnatal stressors, often occurring within the context of the caregiving relationship, exert many of the same effects as gestational stressors and similarly disrupt the HPA axis. In rodent models of subprime caregiving, altered methylation of the *GR* gene is found in offspring hippocampus and is accompanied by exaggerated stress responses in adulthood (Weaver et al., 2004). Pharmacological alteration of the epigenome is sufficient to prevent these outcomes. In a mouse model of postnatal stress wherein pups are repeatedly separated from their mother, offspring exhibit increased HPA axis activity due to decreased methylation of arginine vasopressin (*Avp*). The behavioral correlates of this epigenetic alteration include memory deficits and decreased ability to cope with stress (Murgatroyd et al., 2009). Maternally separated mice also exhibit decreased methylation of proopiomelanocortin (*Pomc*), the precursor hormone for ACTH (Wu, Patchev, Daniel, Almeida, & Spengler, 2014).

Genes associated with neuroplasticity are also epigenetically altered following postnatal stress exposure. In a rodent model of adverse caregiving, maltreated offspring exhibit altered *Bdnf* methylation patterns in several brain regions across development (Doherty & Roth, 2016). The associated behavioral deficits expressed by maltreated offspring in adulthood (Doherty, Keller, Blaze, & Roth, 2017) implicate epigenetic mechanisms in the cognitive and mental health outcomes that often result from early adversity.

In human studies, postnatal adversity is also associated with epigenetic change. Increased *GR* methylation is found in infants of mothers who report postpartum depression (Murgatroyd, Quinn, Sharp, Pickles, & Hill, 2015). Adverse parental care is associated with methylation levels of *BDNF* and *OXTR* (oxytocin receptor, associated with bonding and other social behaviors) in offspring (Unternahrer et al., 2015). The same has been found for *SLC6A4* (the serotonin transporter gene) (Vijayendran, Beach, Plume, Brody, & Philibert, 2012). Offspring of Holocaust survivors also exhibit increased *GR* methylation and altered stress responses, as well as altered methylation of FK506 binding protein 5 (*FKBP5*) (Bowers & Yehuda, 2016). *FKBP5* plays a role in *GR* regulation and is associated with major depressive and posttraumatic stress disorder (PTSD).

Epigenetic Consequences Stress Outside of Development

The epigenome remains responsive outside of early life developmental windows. Acute stress applied to adult rodents produces short-term alterations in *Bdnf* methylation (Fuchikami, Morinobu, Kurata, Yamamoto, & Yamawaki, 2009). Stress of varying intensities (acute to chronic) applied to adult rodents results in varying patterns of histone methylation (Hunter, McCarthy, Milne, Pfaff, & McEwen, 2009). Decreased *Crf* methylation is found in adult mice that develop a socially avoidant phenotype following chronic social stress (Elliott, Ezra-Nevo, Regev, Neufeld-Cohen, & Chen, 2010). Further, following chronic social defeat, adult rodents express significantly more DNMT3A in the nucleus accumbens (Covington et al., 2009), an area associated with reward.

The sensitivity of the human epigenome beyond childhood continues to be empirically supported. For example, the *GR* gene exhibits increased methylation in teenagers following a social stress task, a change that is associated with slower post-task cortisol recovery (Van Der Knaap, Oldehinkel, Verhulst, Van Oort, & Riese, 2015). In addition, *OXTR* methylation is altered in adult participants following the Trier social stress test (Unternahrer et al., 2012).

Mental Health and the Epigenome

Compelling evidence in humans supports the association between epigenetics and mental health outcomes first suggested in rodent models. In suicide victims with a history of child abuse, *GR* methylation is significantly increased in postmortem hippocampal tissue (McGowan et al., 2009). Increased methylation of *Reelin*, a gene implicated in schizophrenia, is found in post-mortem cortical tissue of schizophrenia patients (Grayson et al., 2005). In postmortem parietal cortex tissue of psychotic patients, there is an upregulation of TET1, one of the enzymes involved in hydroxymethylation (Dong, Gavin, Chen, & Davis, 2012). Furthermore, the antipsychotic medication haloperidol is known to alter the epigenetic state in rodents (Bertran-Gonzalez et al., 2008; Shimabukuro, Jinno, Fuke, & Okazaki, 2006). In patients diagnosed with borderline personality disorder, *BDNF* is hypermethylated, an epigenetic change that correlates with childhood trauma (i.e., the more trauma the more methylation) (Perroud et al., 2013).

Epigenetic mechanisms also appear to play a role in depression and anxiety-related disorders. Altered epigenetic patterns have been associated with depressive states, and the epigenome is known to be affected by antidepressants (Vialou, Feng, Robison, & Nestler, 2013). Epigenetic markers have also been heavily associated with risk for and development of PTSD (Yehuda & Bierer, 2009).

Epigenomic Remodeling Outside of Adverse Experience: Therapeutic Implications

The epigenome changes in response to therapeutic experiences. In rodents, enrichment of the environment with toys, increased opportunity for social interaction, and/or increased exercise results in epigenetic remodeling. Environmental enrichment during the gestational period leads to a decrease in hippocampal and frontal cortex methylation in rodent offspring (Mychasiuk et al., 2012). Enrichment of the environment of adult rodents is associated with more open chromatin and increased expression of *Bdnf* in the hippocampus (Kuzumaki et al., 2011). Voluntary exercise in rodents results in improved stress coping abilities and chromatin remodeling (Collins et al., 2009) as well as downregulation of epigenetic regulators (Abel & Rissman, 2013).

Therapeutic and meditative activities also impact humans at the level of the epigenome. In individuals diagnosed with borderline personality disorder, aberrant *BDNF* methylation patterns can be rescued with intensive behavioral therapy in some patients (Perroud et al., 2013). In addition, children with anxiety disorders who exhibit positive responses to cognitive behavioral therapy exhibit increased *SLC6A4* methylation (Roberts et al., 2014). In females, long-term tai-chi practice remodels the epigenome such that it exhibits less typical age-related changes (Ren et al., 2012). It stands to reason that treatment and intervention strategies for mental health disorders can benefit from this epigenomic malleability.

Author Biographies

Tiffany S. Doherty is a doctoral candidate in the Department of Psychological and Brain Sciences at the University of Delaware, where she conducts research in the lab of Dr. Tania Roth. Her dissertation research is focused on the utility of drug intervention in preventing aberrant epigenetic and behavioral trajectories following developmental exposure to adversity.

Dr. Tania L. Roth is an associate professor and director of Graduate Education in Psychological and Brain Sciences at the University of Delaware, where she teaches upper-level and graduate courses in psychology and neuroscience. Her research program is focused on defining epigenetic mechanisms responsible for environmental influences on CNS gene activity, development of behavior, and psychiatric disorder.

References

- Abel, J. L., & Rissman, E. F. (2013). Running-induced epigenetic and gene expression changes in the adolescent brain. *International Journal of Developmental Neuroscience*, 31(6), 382–390.
- Benoit, J. D., Rakic, P., & Frick, K. M. (2015). Prenatal stress induces spatial memory deficits and epigenetic changes in the hippocampus indicative of heterochromatin formation and reduced gene expression. *Behavioural Brain Research*, 281, 1–8.
- Bertran-Gonzalez, J., Bosch, C., Maroteaux, M., Matamalas, M., Hervé, D., Valjent, E., et al. (2008). Opposing patterns of signaling activation in dopamine D1 and D2 receptor-expressing striatal neurons in response to cocaine and haloperidol. *The Journal of Neuroscience*, 28(22), 5671–5685.
- Bolger, T. A., Cohen, T., & Yao, T.-P. (2006). HATs and HDACs. In *Gene expression and regulation* (pp. 111–133). Springer.
- Bowers, M. E., & Yehuda, R. (2016). Intergenerational transmission of stress in humans. *Neuropsychopharmacology: Official publication of the American College of Neuropsychopharmacology*, 41(1), 232–244.
- Chen, C.-C., Wang, K.-Y., & Shen, C.-K. J. (2013). DNA 5-methylcytosine demethylation activities of the mammalian DNA methyltransferases. *Journal of Biological Chemistry*, 288(13), 9084–9091.
- Collins, A., Hill, L. E., Chandramohan, Y., Whitcomb, D., Droste, S. K., & Reul, J. (2009). Exercise improves cognitive responses to psychological stress through enhancement of epigenetic mechanisms and gene expression in the dentate gyrus. *PLoS One*, 4(1), e4330.
- Conradt, E., Lester, B. M., Appleton, A. A., Armstrong, D. A., & Marsit, C. J. (2013). The roles of DNA methylation of NR3C1 and 11 β -HSD2 and exposure to maternal mood disorder in utero on newborn neurobehavior. *Epigenetics*, 8(12), 1321–1329.
- Covington, H. E., Maze, I., LaPlant, Q. C., Vialou, V. F., Ohnishi, Y. N., Berton, O., et al. (2009). Antidepressant actions of histone deacetylase inhibitors. *The Journal of Neuroscience*, 29(37), 11451–11460.
- Devlin, A. M., Brain, U., Austin, J., & Oberlander, T. F. (2010). Prenatal exposure to maternal depressed mood and the MTHFR C677T variant affect SLC6A4 methylation in infants at birth. *PLoS One*, 5(8), e12201.
- Doherty, T. S., Keller, S., Blaze, J., & Roth, T. L. (2017). Phenotypic outcomes in adolescence and adulthood in the scarcity-adversity model of low nesting resources outside the home cage. *Developmental Psychobiology*, 59(6), 703–714.
- Doherty, T. S., & Roth, T. L. (2016). Insight from animal models of environmentally driven epigenetic changes in the developing and adult brain. *Development and Psychopathology*, 28(4pt2), 1229–1243.
- Dong, E., Gavin, D. P., Chen, Y., & Davis, J. (2012). Upregulation of TET1 and downregulation of APOBEC3A and APOBEC3C in the parietal cortex of psychotic patients. *Translational Psychiatry*, 2(9), e159.
- Elliott, E., Ezra-Nevo, G., Regev, L., Neufeld-Cohen, A., & Chen, A. (2010). Resilience to social stress coincides with functional DNA methylation of the Crf gene in adult mice. *Nature Neuroscience*, 13(11), 1351–1353.
- Fuchikami, M., Morinobu, S., Kurata, A., Yamamoto, S., & Yamawaki, S. (2009). Single immobilization stress differentially alters the expression profile of transcripts of the brain-derived neurotrophic factor (BDNF) gene and histone acetylation at its promoters in the rat hippocampus. *International Journal of Neuropsychopharmacology*, 12(1), 73–82.

- Grayson, D. R., Jia, X., Chen, Y., Sharma, R. P., Mitchell, C. P., Guidotti, A., et al. (2005). Reelin promoter hypermethylation in schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 102(26), 9341–9346.
- Hunter, R. G., McCarthy, K. J., Milne, T. A., Pfaff, D. W., & McEwen, B. S. (2009). Regulation of hippocampal H3 histone methylation by acute and chronic stress. *Proceedings of the National Academy of Sciences of the United States of America*, 106(49), 20912–20917.
- Kato, T., & Iwamoto, K. (2014). Comprehensive DNA methylation and hydroxymethylation analysis in the human brain and its implication in mental disorders. *Neuropharmacology*, 80, 133–139.
- Kuzumaki, N., Ikegami, D., Tamura, R., Hareyama, N., Imai, S., Narita, M., et al. (2011). Hippocampal epigenetic modification at the brain-derived neurotrophic factor gene induced by an enriched environment. *Hippocampus*, 21(2), 127–132.
- Matriciano, F., Tueting, P., Dalal, I., Kadriu, B., Grayson, D. R., Davis, J. M., et al. (2013). Epigenetic modifications of GABAergic interneurons are associated with the schizophrenia-like phenotype induced by prenatal stress in mice. *Neuropharmacology*, 68, 184–194.
- McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonte, B., Szyf, M., et al. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience*, 12(3), 342–348.
- Moore, L., Le, T., & Fan, G. (2013). DNA methylation and its basic function. *Neuropsychopharmacology*, 38(1), 23–38.
- Mueller, B. R., & Bale, T. L. (2008). Sex-specific programming of offspring emotionality after stress early in pregnancy. *The Journal of Neuroscience*, 28(36), 9055–9065.
- Murgatroyd, C., Patchev, A. V., Wu, Y., Micale, V., Bockmühl, Y., Fischer, D., et al. (2009). Dynamic DNA methylation programs persistent adverse effects of early-life stress. *Nature Neuroscience*, 12(12), 1559–1566.
- Murgatroyd, C., Quinn, J. P., Sharp, H. M., Pickles, A., & Hill, J. (2015). Effects of prenatal and postnatal depression, and maternal stroking, at the glucocorticoid receptor gene. *Translational Psychiatry*, 5, e560.
- Mychasiuk, R., Zahir, S., Schmol, N., Ilnytsky, S., Kovalchuk, O., & Gibb, R. (2012). Parental enrichment and offspring development: Modifications to brain, behavior and the epigenome. *Behavioural Brain Research*, 228(2), 294–298.
- Peña, C. J., Monk, C., & Champagne, F. A. (2012). Epigenetic effects of prenatal stress on 11 β -hydroxysteroid dehydrogenase-2 in the placenta and fetal brain. *PLoS One*, 7(6), e39791.
- Perroud, N., Salzmann, A., Prada, P., Nicastro, R., Hoeppli, M., Furrer, S., et al. (2013). Response to psychotherapy in borderline personality disorder and methylation status of the BDNF gene. *Translational Psychiatry*, 3(1), e207.
- Ren, H., Collins, V., Clarke, S. J., Han, J.-S., Lam, P., Clay, F., et al. (2012). Epigenetic changes in response to tai chi practice: A pilot investigation of DNA methylation marks. *Evidence-based Complementary and Alternative Medicine*.
- Roberts, S., Lester, K., Hudson, J., Rapee, R., Creswell, C., Cooper, P., et al. (2014). Serotonin transporter methylation and response to cognitive behaviour therapy in children with anxiety disorders. *Translational Psychiatry*, 4(9), e444.
- Shimabukuro, M., Jinno, Y., Fuke, C., & Okazaki, Y. (2006). Haloperidol treatment induces tissue- and sex-specific changes in DNA methylation: A control study using rats. *Behavioral and Brain Functions*, 2(1), 1–5.
- St-Cyr, S., & McGowan, P. (2015). Programming of stress-related behavior and epigenetic neural gene regulation in mice offspring through maternal exposure to predator odor. *Frontiers in Behavioral Neuroscience*, 9, 145.
- Unternaehrer, E., Luers, P., Mill, J., Dempster, E., Meyer, A. H., Staehli, S., et al. (2012). Dynamic changes in DNA methylation of stress-associated genes (OXTR, BDNF[thinsp]) after acute psychosocial stress. *Translational Psychiatry*, 2, e150.
- Unternaehrer, E., Meyer, A. H., Burkhardt, S. C., Dempster, E., Staehli, S., Theill, N., et al. (2015). Childhood maternal care is associated with DNA methylation of the genes for brain-derived

- neurotrophic factor (BDNF) and oxytocin receptor (OXTR) in peripheral blood cells in adult men and women. *Stress*, 18(4), 451–461.
- Van Der Knaap, L. J., Oldehinkel, A. J., Verhulst, F. C., Van Oort, F. V., & Riese, H. (2015). Glucocorticoid receptor gene methylation and HPA-axis regulation in adolescents. The TRAILS study. *Psychoneuroendocrinology*, 58, 46–50.
- Vialou, V., Feng, J., Robison, A. J., & Nestler, E. J. (2013). Epigenetic mechanisms of depression and antidepressant action. *Annual Review of Pharmacology and Toxicology*, 53, 59–87.
- Vijayendran, M., Beach, S., Plume, J. M., Brody, G., & Philibert, R. (2012). Effects of genotype and child abuse on DNA methylation and gene expression at the serotonin transporter. *Frontiers in Psychiatry*, 3, 55.
- Weaver, I. C., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., et al. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience*, 7(8), 847–854.
- Wu, Y., Patchev, A. V., Daniel, G., Almeida, O. F., & Spengler, D. (2014). Early-life stress reduces DNA methylation of the pomc gene in male mice. *Endocrinology*, 155(5), 1751–1762.
- Yehuda, R., & Bierer, L. M. (2009). The relevance of epigenetics to PTSD: Implications for the DSM-V. *Journal of Traumatic Stress*, 22(5), 427–434.

Diffusion MRI: Introduction

Naiara Demnitz and Claire E. Sexton

Department of Psychiatry, University of Oxford, Oxford, UK

Diffusion MRI: An Introduction

Diffusion-weighted imaging (DWI) is a noninvasive magnetic resonance imaging (MRI) technique that enables the study of the brain *in vivo*. It is used frequently to investigate the integrity of the brain's white matter. This chapter aims to introduce the basic principles of diffusion and how it is measured and to summarize some of the inferences that can (or cannot) be drawn from this technique. We then briefly discuss some of the contributions that may come from applying diffusion imaging to the understanding of white matter.

The Basics of Diffusion Imaging

Measuring Diffusion

Over a century ago, Albert Einstein described the random motion of particles due to thermal energy (Einstein, 1905). Due to this random movement, also known as Brownian motion, molecules collide with each other and cause a net displacement. This process is called diffusion.

DWI is an MRI technique that can be used to measure the diffusion process. In diffusion-weighted MR sequences, the magnetic field varies along a direction of interest (Stejskal & Tanner, 1965). Its gradients label the location of water molecules along this direction. As water moves along the direction of the applied gradient, there is a decrease in the MR signal from the water molecules in each voxel. The greater the displacement of the molecules, the greater the signal loss. This signal loss is then used to calculate the amount of diffusion, or apparent diffusion coefficient.

Box 1 The Parameters of a DTI Sequence

DTI sequences vary immensely between studies. One crucial parameter is the number of diffusion-encoding gradient directions. Although only three directions are required for DWI, a minimum of six is necessary to calculate the diffusion tensor. As a larger number of gradient directions result in a more accurate estimate of the diffusion tensor, DTI sequences often include more directions. Since increasing the number of gradient directions requires an increase in scan time, it is not always feasible to adopt these optimized parameters though. Another important consideration is the voxel dimension. Ideally, the smallest voxel size should be used. In practice, however, decreasing voxel size comes at an increasingly larger cost to the signal-to-noise ratio and must therefore be carefully considered. In a 3 Tesla scanner, a suggested frame of reference is that DWI images maintain the signal-to-noise ratio above 3 : 1, with an isotropic voxel size between 1.5 and 2 mm (Jones et al., 2013).

Three-dimensional diffusivity is frequently modeled using a symmetrical matrix of numbers or tensor—thus giving rise to the name diffusion tensor imaging (DTI). When molecules are equally likely to move in any direction (i.e., isotropic diffusion), the diffusion pattern can be thought of as a sphere. However, in an anisotropic environment, this spherical representation is no longer suitable because diffusion in one direction is more likely than the others. Accordingly, the diffusion tensor is “rugby-ball” shaped, an ellipsoid in which the major axis reflects the principle direction of diffusion within a voxel (Basser, 1995).

By fitting the tensor model to measurements at each voxel, several useful parameters can be estimated (Box 1). Fractional anisotropy (FA) is an index of the degree of diffusion directionality and can range from 0 to 1. While higher values reflect increased diffusion directionality, lower FA values are indicative of more isotropic diffusion. Other diffusivity metrics include axial diffusivity (AD) (also referred to as parallel diffusivity), mean diffusivity (MD), radial diffusivity (RD) (also referred to as perpendicular diffusivity), and mode of anisotropy (MO) (Table 1).

Table 1 Overview of diffusion measurements estimated from the tensor model.

<i>Metric</i>	<i>Interpretation</i>
Fractional anisotropy (FA)	Index of the degree of diffusion directionality. Ranges from 0 (isotropic diffusion) to 1 (increased directionality, anisotropic diffusion)
Axial diffusivity (AD)	Measure of the diffusivity in the direction of maximum diffusion in a voxel. May relate to axonal health (Kinoshita, Ohnishi, Kohshi, & Yokota, 1999)
Radial diffusivity (RD)	Average of the diffusivity values in the two axes perpendicular to the principle direction of diffusion. May be indicative of myelin content (Klawiter et al., 2011; Song et al., 2005)
Mean diffusivity (MD)	Mean amount of diffusion of the three directions calculated by the tensor
Mode of anisotropy (MO)	Index of the type or shape of anisotropy. Ranges from -1 (planar shape, e.g. in regions of crossing fibers) to 1 (linear shape, e.g. in regions with one dominating fiber orientation)

Diffusion in White Matter

The diffusion measures obtained with DTI can be used to examine the integrity of white matter in the brain *in vivo*. Within white matter, a variety of biological boundaries restrict the diffusion process and create a more directional (anisotropic) pattern of water molecule displacement. For instance, axonal membranes result in diffusion occurring more easily along a fiber bundle than across it. Further, increased myelination and axonal density can enhance the degree of anisotropy, while increased axon caliber can decrease anisotropy (Beaulieu, 2002, 2009). Consequently, the measures of anisotropy and overall molecule displacement obtained with DTI can serve as quantitative indices of the orientation and relative integrity of white matter tracts in the brain.

Crucially, it is important to note that, as with MRI parameters in general, there is no direct relationship between diffusion MRI measures and any particular anatomical feature. Measures reflect a number of tissue properties (e.g., membrane integrity, myelin thickness, axon caliber, and axon density) without being specific to any. DTI measures are also sensitive to fiber architecture. In areas containing crossing fiber bundles such as the centrum semiovale, FA values are naturally low because any given voxel will include fiber bundles going in different directions. Moreover, DTI measures can also be affected by factors such as partial volume contamination (mixing of tissue types within a voxel) and spatial filtering. For instance, since diffusion within the cerebrospinal fluid (CSF) is isotropic and approximately four times larger than that in white matter (Jones, Knosche, & Turner, 2013), any partial volume of CSF within the recorded voxel will contaminate findings. This is particularly problematic at the borders between tissue and CSF-filled spaces. It is, therefore, not possible to hold specific biological microstructures (e.g., myelin sheaths) accountable for observed changes in DTI measures.

More recent DWI techniques might be able to provide more direct markers of tissue microstructure than DTI metrics. Although the tensor model is currently the most frequently employed technique in diffusion MRI, DWI can be modeled in a number of ways (Winston, 2015). For example, neurite orientation dispersion and density imaging (NODDI) is a novel DWI technique that aims to overcome the nonspecificity of DTI parameters (Zhang, Schneider, Wheeler-Kingshott, & Alexander, 2012). For example, by providing additional estimates of tissue microstructure, NODDI can disentangle two factors that contribute to FA: fiber orientation and neurite density (Table 2).

Table 2 Overview of diffusion measurements estimated through neurite orientation dispersion and density imaging (NODDI).

<i>Metric</i>	<i>Definition</i>	<i>Interpretation</i>
Intracellular volume fraction (ICVF)	An index of white matter neurite density	Ranges from 0 (complete extraneurite diffusion) to 1 (complete intraneurite diffusion)
Isotropic or free water volume fraction (ISOVF)	Volume within a voxel occupied by isotropic water diffusion like freely moving cerebrospinal fluid (CSF)	Ranges from 0 (no CSF-like fluid) to 1 (complete CSF-like fluid)
Orientation dispersion index (ODI)	A measure of within-voxel tract disorganization	Ranges from 0 (complete directional coherence) to 1 (fully dispersed neurites)

Analysis Approaches

Regardless of the model selected to estimate diffusion metrics, the collected data must undergo a series of processing steps before it can be used in statistical analysis.

This pipeline typically involves three stages: preprocessing (motion correction, eddy current corrections, and brain extraction), estimation of the diffusion tensor (or alternative DWI model), and statistical analysis. A variety of software packages offer tools to perform these processing stages—many of which are freely available (see Hasan, Walimuni, Abid, & Hahn, 2011 for review).

Similarly, a variety of analysis approaches can be used to prepare the individual diffusion data for the final step of the pipeline, the statistical analysis. Diffusion data can be extracted locally in a priori defined regions using region of interest (ROI) analysis or tractography (i.e., using specific regional hypotheses), or globally using the voxel-based method (VBM) or tract-based spatial statistics (TBSS) and controlling for multiple testing a posteriori. With regard to regional analysis approaches, ROI analysis involves delineating specific, predefined, regions of the brain, either manually or using an automated parcellation method. Although manual ROI placement avoids registration errors, it is labor intensive and susceptible to user bias. Automated parcellation, on the other hand, has the advantage of being less time consuming but may be less sensitive to individual anatomical variability. Tractography, an automated method, involves the reconstruction of white matter tracts and thus allows inferences about the connectivity between brain regions.

In terms of global analysis methods, VBM consists of registering individual images to a common template and performing a voxel-by-voxel whole-brain statistical test, either by comparing white matter between groups or by examining correlations between diffusion measurements and a descriptor variable. This method is limited by the quality of the registration between individual images and the amount of spatial smoothing imposed on the data. In order to address these issues, TBSS projects the FA data of all participants onto an average FA tract skeleton before applying voxel-wise cross-participant statistics (Smith et al., 2006). However, since the TBSS skeleton assumes that peak FA values are the center of the tract, TBSS may be insensitive to changes along the width of the tract. While an in-depth description of the various analysis methods is beyond the scope of this chapter, a review of these techniques can be found elsewhere (Bodini & Ciccarelli, 2009; Mueller, Lim, Hemmy, & Camchong, 2015).

Diffusion-Weighted Imaging in Practice

DWI has made substantial contributions to the understanding of the brain *in vivo*, both in health and in disease. This technique has allowed researchers to compare estimates of tissue microstructure between groups (e.g., patients and healthy populations) and to explore correlations between brain and behavior. As previously mentioned, although DWI is not limited to the tensor model, DTI remains the most widely employed method in diffusion imaging. This section focuses, therefore, on findings from DTI studies.

Changes in white matter occur throughout our lifespan: in development, adulthood, and old age. Early development sets the stage for rapid growth and brain maturation, and a substantial amount of myelination occurs in this period (Dubois et al., 2014). DTI parameters are sensitive to the changes during white matter maturation, with studies showing increased FA in the developing white matter, especially between birth and 2 years of age (Dubois et al., 2014). Through childhood and adolescence, the continued development of white matter

microstructure is reflected in an increase in FA (Peters et al., 2012). In late adulthood, this trajectory is reversed. Postmortem studies have shown that brain aging is marked by loss of myelinated fibers (Marner, Nyengaard, Tang, & Pakkenberg, 2003) and degradation of white matter integrity (Peters, 2002). Accordingly, DTI studies have shown a decrease in FA in older adults (Bennett & Madden, 2014). Recent studies have complemented the age-related changes observed in FA with other diffusivity parameters (MD, AD, and RD)—an advisable practice as, in some cases, these measures may be more sensitive to changes in white matter integrity (Bennett & Madden, 2014).

Besides demonstrating the trajectory of white matter development and decline, DTI studies have also highlighted regional differences in these changes. Although functional networks are established early on, the infant brain is not a scaled-down adult brain. Brain maturation occurs successively in a mosaic pattern: while some regions, such as the sensory and motor areas, develop early and quickly, other regions, like the frontal white matter, develop later and at a slower pace (Dubois et al., 2014). In contrast, the decline observed in white matter integrity in late adulthood is most prominent in frontal and parietal white matter and less marked in the occipital and posterior white matter (see Bennett & Madden, 2014 for review).

Understanding the healthy changes that occur in white matter microstructure with age is an essential step for examining the role of white matter changes in illness. Changes in white matter can be seen with DTI not only with conditions that have typically been considered white matter diseases (e.g., multiple sclerosis) but also across a host of conditions. For example, studies have reported indices of reduced white matter integrity in patients with Alzheimer's dementia and mild cognitive impairment, Parkinsonian syndromes, major depression and bipolar disorder, schizophrenia, and obsessive-compulsive disorders (Table 3). These findings indicate a role for white matter changes in psychiatric and neurological disorders. Considerable research is still necessary to understand how pathological changes in white matter structures contribute to each condition.

Changes in white matter structure are not limited to development, aging, and disease, though. Learning new skills and fine-tuning existing abilities can also alter brain structure, a phenomenon known as plasticity. DTI measures are sensitive to these experience-dependent alterations and have contributed to evidence that white matter plasticity is a rapid and dynamic

Table 3 Factors associated with reduced white matter integrity in DTI studies.

<i>Disease</i>	<i>Change in DTI measures</i>	<i>Review of findings</i>
Multiple sclerosis (MS)	Reduced FA and increased MD in lesions and in the normal-appearing white matter	Sbardella, Tona, Petsas, & Pantano (2013)
Alzheimer's disease and mild cognitive impairment	Widespread white matter abnormalities: reduced FA, increased MD	Pini et al. (2016)
Parkinsonian syndromes	Reduced FA in the substantia nigra	Cochrane & Ebmeier (2013)
Major depression and bipolar disorder	Reduced FA in the genu of the corpus callosum	Wise et al. (2016)
Schizophrenia	Widespread reduction in FA; DTI changes may vary with illness duration	Kubicki, Westin, Pasternak and Shenton (2014), Peters & Karlsgodt (2015)
Obsessive-compulsive disorder	Widespread changes in FA	Radua et al. (2014)

process, one that can be observed after weeks (Scholz, Klein, Behrens, & Johansen-Berg, 2009), or even just hours (Sagi et al., 2012), of learning. Moreover, plasticity is not limited to a single type of learning: changes in DTI measures have been associated with training in juggling (Scholz et al., 2009), memory (Takeuchi et al., 2010), reasoning (Mackey, Whitaker, & Bunge, 2012), meditation (Tang, Lu, Fan, Yang, & Posner, 2012), and lifelong musical skills (Bengtsson et al., 2005; for reviews see Wang & Young, 2014; Thomas & Baker, 2013; Zatorre, Fields, & Johansen-Berg, 2012).

DTI studies are likely to benefit from the trend toward larger cohorts and open access imaging data. With increased power, such datasets will allow more robust investigations of the role of white matter integrity in health and disease. For instance, the UK Biobank, the world's largest health imaging study to date, aims to collect brain imaging data in 100,000 adults, alongside measures of cognitive functioning, medical history, lifestyle, and genotypes (Sudlow et al., 2015). This open access dataset, which includes diffusion-weighted images, will enable powerful research into the genotypes and lifestyle factors that may have protective or detrimental effects on white matter structure and overall brain health.

Conclusions

Diffusion MRI provides a unique method for understanding the living brain. While it is important not to overinterpret DTI measures, charting changes through development and aging has provided valuable insight into the changes that normally occur in white matter. Further, equipped with the understanding of healthy age-related white matter changes, clinical studies have identified a wide range of psychiatric and neurological disorders that are associated with white matter abnormalities. While the tensor model has served this field well for over twenty years, it is likely that novel DWI techniques may rise in importance given their ability to provide additional estimates of tissue microstructure.

Author Biographies

Claire E. Sexton and Naiara Demnitz are both based at the University of Oxford, where Dr. Sexton is a postdoctoral researcher at the Nuffield Department of Clinical Neurosciences and Ms. Demnitz is a doctoral student at the Department of Psychiatry. Their research uses magnetic resonance imaging techniques to understand the aging process and factors proposed to promote healthy aging in the brain, including mobility, physical activity, and sleep.

References

- Basser, P. J. (1995). Inferring microstructural features and the physiological state of tissues from diffusion-weighted images. *NMR in Biomedicine*, 8(7-8), 333-344. doi:10.1002/Nbm.1940080707
- Beaulieu, C. (2002). The basis of anisotropic water diffusion in the nervous system—A technical review. *NMR in Biomedicine*, 15(7-8), 435-455. doi:10.1002/nbm.782
- Beaulieu, C. (2009). The biological basis of diffusion anisotropy. In H. Johansen-Berg & T. E. J. Behrens (Eds.), *Diffusion MRI: From quantitative measurement to in vivo neuroanatomy* (pp. 105-126). Academic Press.
- Bengtsson, S. L., Nagy, Z., Skare, S., Forsman, L., Forssberg, H., & Ullen, F. (2005). Extensive piano practicing has regionally specific effects on white matter development. *Nature Neuroscience*, 8(9), 1148-1150.

- Bennett, I. J., & Madden, D. J. (2014). Disconnected aging: Cerebral white matter integrity and age-related differences in cognition. *Neuroscience*, 276, 187–205.
- Bodini, B., & Ciccarelli, O. (2009). Diffusion MRI in neurological disorders. In H. Johansen-Berg & T. E. J. Behrens (Eds.), *Diffusion MRI: From quantitative measurement to in-vivo neuroanatomy* (pp. 175–204). Academic Press.
- Cochrane, C. J., & Ebmeier, K. P. (2013). Diffusion tensor imaging in parkinsonian syndromes: A systematic review and meta-analysis. *Neurology*, 80(9), 857–864. doi:10.1212/WNL.0b013e318284070c
- Dubois, J., Dehaene-Lambertz, G., Kulikova, S., Poupon, C., Huppi, P. S., & Hertz-Pannier, L. (2014). The early development of brain white matter: A review of imaging studies in fetuses, newborns and infants. *Neuroscience*, 276, 48–71.
- Einstein, A. (1905). The theory of the Brownian movement. *Annalen der Physik*, 17, 549.
- Hasan, K. M., Walimuni, I. S., Abid, H., & Hahn, K. R. (2011). A review of diffusion tensor magnetic resonance imaging computational methods and software tools. *Computers in Biology and Medicine*, 41(12), 1062–1072. doi:10.1016/j.combiomed.2010.10.008
- Jones, D. K., Knosche, T. R., & Turner, R. (2013). White matter integrity, fiber count, and other fallacies: The do's and don'ts of diffusion MRI. *NeuroImage*, 73, 239–254. doi:10.1016/j.neuroimage.2012.06.081
- Kinoshita, Y., Ohnishi, A., Kohshi, K., & Yokota, A. (1999). Apparent diffusion coefficient on rat brain and nerves intoxicated with methylmercury. *Environmental Research*, 80(4), 348–354.
- Klawiter, E. C., Schmidt, R. E., Trinkaus, K., Liang, H. F., Budde, M. D., Naismith, R. T., ... Benzinger, T. L. (2011). Radial diffusivity predicts demyelination in ex vivo multiple sclerosis spinal cords. *NeuroImage*, 55(4), 1454–1460.
- Kubicki, M., Westin, C. F., Pasternak, O., & Shenton, M. E. (2014). Diffusion tensor imaging and its application to schizophrenia and related disorders. In *Diffusion MRI* (pp. 317–334). Academic Press.
- Mackey, A. P., Whitaker, K. J., & Bunge, S. A. (2012). Experience-dependent plasticity in white matter microstructure: Reasoning training alters structural connectivity. *Frontiers in Neuroanatomy*, 6.
- Marner, L., Nyengaard, J. R., Tang, Y., & Pakkenberg, B. (2003). Marked loss of myelinated nerve fibers in the human brain with age. *Journal of Comparative Neurology*, 462(2), 144–152.
- Mueller, B. A., Lim, K. O., Hemmy, L., & Camchong, J. (2015). Diffusion MRI and its role in neuropsychology. *Neuropsychology Review*, 25(3), 250–271. doi:10.1007/s11065-015-9291-z
- Peters, A. (2002). The effects of normal aging on myelin and nerve fibers: A review. *Journal of Neurocytology*, 31(8-9), 581–593.
- Peters, B. D., & Karlsgodt, K. H. (2015). White matter development in the early stages of psychosis. *Schizophrenia Research*, 161(1), 61–69.
- Peters, B. D., Szeszko, P. R., Radua, J., Ikuta, T., Gruner, P., DeRosse, P., ... Malhotra, A. K. (2012). White matter development in adolescence: Diffusion tensor imaging and meta-analytic results. *Schizophrenia Bulletin*, 38(6), 1308–1317.
- Pini, L., Pievani, M., Bocchetta, M., Altomare, D., Bosco, P., Cavedo, E., ... Frisoni, G. B. (2016). Brain atrophy in Alzheimer's disease and aging. *Ageing Research Reviews*. doi:10.1016/j.arr.2016.01.002
- Radua, J., Grau, M., van den Heuvel, O. A., Thiebaut de Schotten, M., Stein, D. J., Canales-Rodriguez, E. J., ... Mataix-Cols, D. (2014). Multimodal voxel-based meta-analysis of white matter abnormalities in obsessive-compulsive disorder. *Neuropsychopharmacology*, 39(7), 1547–1557.
- Sagi, Y., Tavor, I., Hofstetter, S., Tzur-Moryosef, S., Blumenfeld-Katzir, T., & Assaf, Y. (2012). Learning in the fast lane: New insights into neuroplasticity. *Neuron*, 73(6), 1195–1203.
- Sbardella, E., Tona, F., Petsas, N., & Pantano, P. (2013). DTI measurements in multiple sclerosis: Evaluation of brain damage and clinical implications. *Multiple Sclerosis International*, 2013, 671730. doi:10.1155/2013/671730
- Scholz, J., Klein, M. C., Behrens, T. E. J., & Johansen-Berg, H. (2009). Training induces changes in white-matter architecture. *Nature Neuroscience*, 12(11), 1370–1371.

- Smith, S. M., Jenkinson, M., Johansen-Berg, H., Rueckert, D., Nichols, T. E., Mackay, C. E., ... Behrens, T. E. J. (2006). Tract-based spatial statistics: Voxelwise analysis of multi-subject diffusion data. *NeuroImage*, 31(4), 1487–1505. doi:10.1016/j.neuroimage.2006.02.024
- Song, S. K., Yoshino, J., Le, T. Q., Lin, S. J., Sun, S. W., Cross, A. H., & Armstrong, R. C. (2005). Demyelination increases radial diffusivity in corpus callosum of mouse brain. *NeuroImage*, 26(1), 132–140.
- Stejskal, E. O., & Tanner, J. E. (1965). Spin diffusion measurements: Spin echoes in the presence of a time-dependent field gradient. *Journal of Chemical Physics*, 42(1), 288–+. doi:10.1063/1.1695690
- Sudlow, C., Gallacher, J., Allen, N., Beral, V., Burton, P., Danesh, J., ... Collins, R. (2015). UK biobank: An open access resource for identifying the causes of a wide range of complex diseases of middle and old age. *PLoS Medicine*, 12(3).
- Takeuchi, H., Sekiguchi, A., Taki, Y., Yokoyama, S., Yomogida, Y., Komuro, N., ... Kawashima, R. (2010). Training of working memory impacts structural connectivity. *Journal of Neuroscience*, 30(9), 3297–3303.
- Tang, Y. Y., Lu, Q. L., Fan, M., Yang, Y. H., & Posner, M. I. (2012). Mechanisms of white matter changes induced by meditation. *Proceedings of the National Academy of Sciences of the United States of America*, 109(26), 10570–10574.
- Thomas, C., & Baker, C. I. (2013). Teaching an adult brain new tricks: A critical review of evidence for training-dependent structural plasticity in humans. *NeuroImage*, 73, 225–236.
- Wang, S., & Young, K. M. (2014). White matter plasticity in adulthood. *Neuroscience*, 276, 148–160.
- Winston, G. P. (2015). The potential role of novel diffusion imaging techniques in the understanding and treatment of epilepsy. *Quantitative Imaging in Medicine and Surgery*, 5(2), 279–287. doi:10.3978/j.issn.2223-4292.2015.02.03
- Wise, T., Radua, J., Nottje, G., Cleare, A. J., Young, A. H., & Arnone, D. (2016). Voxel-based meta-analytical evidence of structural disconnectivity in major depression and bipolar disorder. *Biological Psychiatry*, 79(4), 293–302. doi:10.1016/j.biopsych.2015.03.004
- Zatorre, R. J., Fields, R. D., & Johansen-Berg, H. (2012). Plasticity in gray and white: neuroimaging changes in brain structure during learning. *Nature neuroscience*, 15(4), 528–536.
- Zhang, H., Schneider, T., Wheeler-Kingshott, C. A., & Alexander, D. C. (2012). NODDI: Practical in vivo neurite orientation dispersion and density imaging of the human brain. *NeuroImage*, 61(4), 1000–1016. doi:10.1016/j.neuroimage.2012.03.072

Structural Neuroimaging of Hippocampal Subfields in Healthy Aging, Alzheimer's Disease, Schizophrenia, and Major Depressive Disorder

Lok-Kin Yeung and Adam M. Brickman

Columbia University Medical Center, New York, NY, USA

Introduction

The hippocampus is critically important for episodic memory and spatial representation. It contains a number of distinct subfields, which differ on the basis of neuronal populations and organization (Duvernoy, Cattin, & Risold, 2013). In recent years, a number of segmentation protocols have been developed to map these histological divisions onto high-resolution scans in living individuals using geometric rules derived from anatomical studies (see Yushkevich et al., 2015, for review). Applying this method, researchers have begun to examine how hippocampal subfields differ in the course of healthy aging and along the trajectory of diseases that affect the hippocampus. Among neurological and psychiatric diseases, the most work on hippocampal subfield volume differences has been done in Alzheimer's disease (AD), schizophrenia, and major depressive disorder (MDD), which are well known to involve structural changes in the hippocampus. This chapter will present a brief overview of this research.

A quick anatomical survey of the hippocampus may be helpful to orient the reader (Duvernoy et al., 2013). Unlike much of the brain, which has six-celled layers (i.e., neocortex), the hippocampus is made of allocortex (also called archicortex), with only three cell layers. Internally, the hippocampus is made up of two interlocking lamina. The inner lamina is known as the dentate gyrus, while the outer lamina is known as the cornu Ammonis (CA). In turn, the CA is subdivided into four distinct subfields (labeled CA1–4) due to their cellular

heterogeneity. Along the long axis of the hippocampus, distinctions are also made between the head, body, and tail regions (moving anterior to posterior), which differ in their distribution of hippocampal subfields when viewed cross-sectionally. In addition, the subiculum, which sits between CA1 and the entorhinal cortex, is usually considered part of the hippocampal formation along with the rest of the hippocampus proper.

Within the last few years, a number of MRI segmentation protocols were developed to quantify the volume of hippocampal subfields *in vivo* (Yushkevich et al., 2015). Typically, these segmentation protocols involve taking a non-isotropic T2-weighted fast spin echo (FSE) or turbo spin echo (TSE) scan aligned to the long axis of the hippocampus. These protocols produce “high-resolution” images that have in-plane resolution of 0.4 mm by 0.4 mm or less in the coronal plane, but with a slice gap of 1 mm (using a 7 T magnet) or 2 mm (using a 3 T magnet). By comparison, standard T1-weighted functional scans typically produce images at a resolution of 1 mm by 1 mm. With the higher resolution afforded by these new scanning methods, it is possible to visualize the stratum radiatum, lacunosum, and moleculare (SRLM), a thin band of white matter that separates the dentate gyrus from the CA subfields (i.e., where the CA wraps around the dentate gyrus). This makes it possible to distinguish the two hippocampal lamina with confidence on an MRI scan. Each segmentation protocol then employs histological atlases to devise geometric rules based on anatomical landmarks to subdivide the hippocampus into subfields. For instance, Figure 1 shows manual delineation of three hippocampal subfields (CA1, subiculum, and a combined CA3/dentate gyrus region) using the Olsen–Amaral–Palombo protocol, while Figure 2 shows a 3D reconstruction of the same hippocampal subfields using the same protocol.

It is important to note that this kind of segmentation cannot identify with certainty that a specific location on a particular brain scan belongs to a certain hippocampal subfield. Histological studies typically analyze a large number of hippocampi and define the boundaries

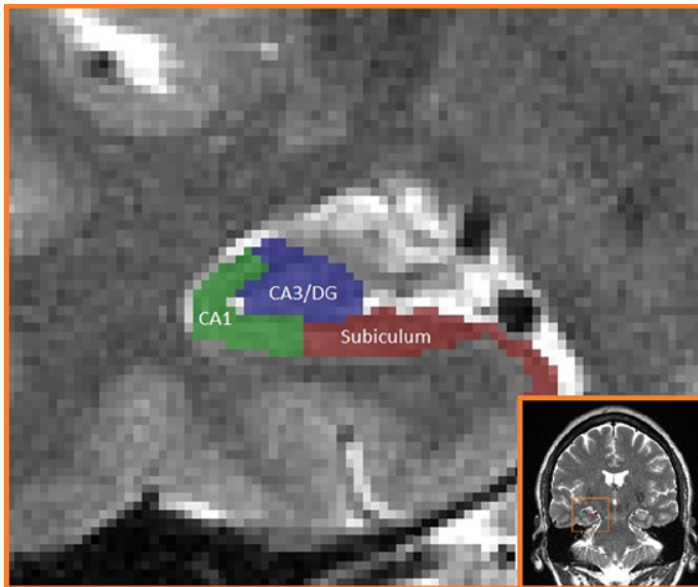


Figure 1 Coronal slice of a high-resolution T2-weighted scan (0.43×0.43 mm in plane) in the hippocampal body, with hippocampus subfields delineated following the Olsen–Amaral–Palombo protocol.

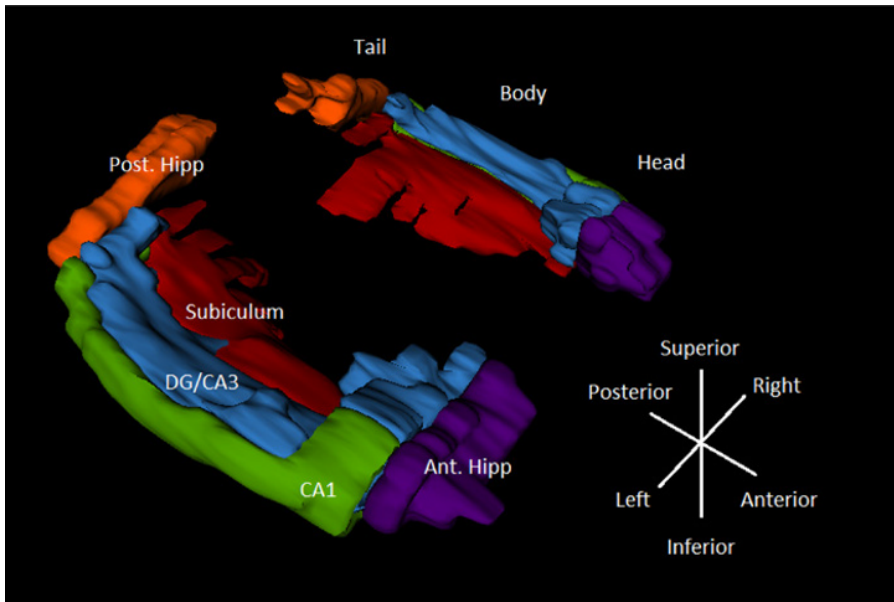


Figure 2 3D reconstruction of manually segmented hippocampal subfields using the Olsen–Amaral–Palombo protocol. Note that subfields were delineated on slices spaced 3 mm apart, causing some apparent discontinuities when viewed in this projection.

of subfields on the basis of anatomical landmarks based on the most typical arrangement observed. However, each individual brain exhibits idiosyncratic variation from the norm. Therefore, it is only possible to say that based on the reference atlases, a particular location on an MRI scan is likely to belong to a certain subfield. This issue is particularly problematic in regions with highly variable anatomical landmarks or a large degree of variation across different brains. Here, subfield identification can only be achieved to a lower degree of accuracy. For example, the irregularity of the digitations in the hippocampal head makes it difficult to differentiate clearly the arrangement of subfields. Many segmentation protocols exclude the hippocampal head entirely for this reason. Moreover, there is some disagreement about where the boundaries between subfields lie (Yushkevich et al., 2015). For instance, one particular area of disagreement is where the boundary between CA1 and subiculum is situated (and how that boundary changes along the long axis of the hippocampus). Further, segmentation protocols differ on which subfields are grouped together because they are difficult to distinguish anatomically. For instance, many segmentation protocols define a combined CA3/dentate gyrus region due to the difficulty of separating those regions visually. However, this decision makes it difficult to ascertain which of these two regions mediates observed effects—this is of particular importance as these two regions have putatively different cognitive functions. It is important to keep in mind these protocol differences when comparing different studies. There is an ongoing effort to create a harmonized segmentation protocol to address these issues (see www.hippocampalsubfields.com for more information).

In addition to manual segmentation, there are a number of automated approaches for hippocampus subfield segmentation. The three most commonly used automated methods are (a) the protocol included in the FreeSurfer segmentation software, (b) the Multiple Automatically Generated Templates Brain Segmentation Algorithm (MAGet-Brain), and (c) the Automated Segmentation of Hippocampal Subfields (ASHS) toolbox. There are two major

advantages of automated hippocampal subfield segmentation. Firstly, manual segmentation of hippocampal subfields requires significant training to achieve a necessary level of skill to produce accurate segmentations reliably. Further, even a skilled tracer requires 4–6 hr to segment hippocampal subfields on each scan. Thus, automated segmentation offers savings in terms of workload and overall processing time. Secondly, automated segmentation follows algorithmic rules far more precisely than human tracers, resulting in more reliable results. Typically, human tracers achieve a reliability of approximately 0.8–0.9, as indexed by the Dice overlap coefficient, with worse reliability in smaller subfields. Automated approaches, on the other hand, theoretically give exactly the same result when run multiple times on the same data. However, these advantages of automated segmentation must be weighed against its disadvantages. At the current moment, the accuracy of a human tracer is still greater than the accuracy of an automated algorithm, and expert manual tracing remains the “gold standard” of hippocampus segmentation. Thus, interpretation of results produced by automated segmentation must keep this in consideration.

This chapter focuses on recent studies that employ high-resolution structural volumetric imaging of hippocampal subfields in the course of normal aging and three clinical disorders associated with reduced hippocampus volume: AD, schizophrenia, and MDD. There is a detailed body of work that focuses on surface deformations of the hippocampus, which will not be discussed here. While surface deformation analysis is sensitive to differences in the CA subfields and the subiculum, the anatomy of the hippocampus prevents this method from being able to detect differences in the dentate gyrus, which lies in the interior of the hippocampus.

There are also a number of studies that use the automated hippocampus subfield segmentation protocol implemented in FreeSurfer 5.3. However, concerns have been expressed about the accuracy of the results produced using this method (Wisse, Biessels, & Geerlings, 2014) for two important reasons. Firstly, FreeSurfer 5.3 performs hippocampal segmentation on T1-weighted images taken at a lower resolution (1 mm³ isotropic). At that resolution, the contrast between white and gray matter is insufficient to be able to demarcate the boundaries between the two hippocampal lamina (i.e., the SRLM between dentate gyrus and the other hippocampal subfields) accurately. Secondly, FreeSurfer 5.3 segments the hippocampus based on its division within a single coronal slice and then projects that division to slices along the length of the hippocampus. However, the placement of subfields is heterogeneous along the hippocampal long axis. Other segmentation protocols rely on anatomical landmarks and internal boundaries to define subfield boundaries for this very reason. Because of these two issues, the subfields identified by FreeSurfer 5.3 do not accurately reflect the internal structure of the hippocampus. A newer version of the FreeSurfer software (v6.0) has addressed these concerns to some degree (Iglesias et al., 2015). The newer software performs segmentation on high-resolution scans and bases its subdivisions on a novel atlas derived from 15 postmortem brains. As far as possible, this chapter excludes findings from studies that rely on automated segmentation of the hippocampus using FreeSurfer 5.3. Studies segmented using this method are explicitly identified as such. Otherwise, all the studies cited in this article employed manual segmentation of hippocampal subfields.

Aging

A number of studies looked cross-sectionally at the effect of aging on hippocampal subfield volumes in neurologically healthy adults across the adult lifespan using manual segmentation. There are two subfields where volume reductions have been consistently associated with

increasing age. Firstly, many studies reported a negative relationship between age and CA1 volume (e.g., Mueller et al., 2007; Wisse, Biessels, & Geerlings, 2014, but see also La Joie et al., 2010). Studies that contrast regional volumes between younger adult (20–30) and older adult (65+) groups also report selective volume reductions in CA1 (e.g., Kerchner et al., 2013). Secondly, volume reductions in the dentate gyrus are associated with increased age in healthy older adults when the dentate gyrus is evaluated independently (Wisse, Biessels, Heringa, et al., 2014), instead of being combined with CA3. Some studies also reported age-related differences in subiculum volume (La Joie et al., 2010), but other studies have not. This discrepancy might be explained by differences in the relative position of the CA1/subiculum boundary across different segmentation protocols.

Alzheimer's Disease

In AD, high-resolution *in vivo* hippocampus subfield volumetry studies show a progressive pattern of volume loss, consistent with direct postmortem analysis of pathology and neuronal loss. The staging work of Braak and Braak (1991) reported that neurofibrillary tangles first appear in the hippocampus/adjacent entorhinal cortex, before appearing in the hippocampus in CA1, then spreading to the subiculum, and finally to the remaining CA subfields, affecting the dentate gyrus particularly late in the progression of the disease. Histological studies of neuronal loss demonstrate a similar progression. There is acute neuronal loss in CA1, even in the earlier stages of the disease (Bobinski et al., 1995). At more advanced stages, there is neuronal loss in the other CA subfields, and even in layers of the dentate gyrus.

This pattern of AD-related volume loss is mirrored in the imaging results. Nearly all studies contrasting healthy controls with AD patients, or with patients with amnesic mild cognitive impairment (MCI), who are at particularly risk for developing AD, report volume reductions in CA1 (e.g., La Joie et al., 2013; Mueller et al., 2007), as well as in the hippocampus/adjacent entorhinal cortex (e.g., Kerchner et al., 2013). A number of studies also report volume reductions of the subiculum in AD and MCI (e.g., La Joie et al., 2013; Mueller et al., 2007). Studies that differentiate patient groups into a less advanced MCI group and a more advanced AD group tend to show a progressive pattern of volume loss matching the histological studies. For instance, La Joie et al. (2013) reported CA1 and subiculum reductions in individuals with MCI and additional volume reductions in a combined CA3/DG region in their AD group. Scanning at 7T, Kerchner et al. (2013) and Wisse, Biessels, Heringa, et al. (2014) both report no volume reductions in MCI patients, but volume reductions in all subfields in AD patients. However, other studies have also reported that all subfields are reduced in volume in MCI and the volume reductions are larger with progression to AD.

Schizophrenia

Hippocampus subfield volumetry studies comparing schizophrenia patients with healthy controls suggest that all subfields might be affected, with an emphasis on CA1 volume changes early in the disease process. Cross-sectional studies using FreeSurfer 5.3 to segment the hippocampus have reported that that severity of positive symptoms in schizophrenia patients was related to volume reductions in CA1 and CA2/3, but other studies have reported that schizophrenia patients might also be affected by volume reductions in the dentate gyrus and subiculum as well. A more recent study (Ho et al., 2017)—using the FreeSurfer 6.0 segmentation

algorithm—that divided schizophrenia patients by the length of illness found that participants who had schizophrenia for a longer period of time (mean = 18 years) had volume reductions in all hippocampal subfields. However, a second patient group who developed schizophrenia more recently (mean = 7 years) only had volume reductions in CA1. Longitudinal follow-up of the latter group showed volume reduction in all subfields over time. Studies employing other automated segmentation methods provide similar results. For instance, a recent study using the ASHS protocol on high-resolution scans reported volume reductions in CA1 and the dentate gyrus in schizophrenia patients relative to controls (Ota et al., 2017).

Depression

Studies examining hippocampal subfield volume in individuals with MDD tend to report one of two different effects. Some studies report selective reduction in dentate gyrus volume in MDD patients compared with healthy controls (e.g., Travis et al., 2015). Dentate gyrus volume has also been linked to peripheral markers of oxidative stress associated with MDD. In contrast, other studies reported no subfield volume differences in individuals with depression compared with non-depressed controls. For instance, Ota et al. (2017) used the ASHS toolbox to examine subfield volume differences between 36 MDD patients (17 medicated, 19 unmedicated) and age-matched controls and found no differences in the volumes of any of the subfields in either patient group. One plausible explanation for the contradictory results reported by different studies arises from differences in segmentation protocols used by different groups. For instance, Travis et al. (2015) combined all the CA subfields, whereas Ota et al. (2017) split up those subfields. There are differing opinions about where the boundary between the dentate gyrus and CA3/4 lies, which may also explain observed differences.

Conclusion

High-resolution structural imaging, combined with the development of sophisticated segmentation protocols, has made it possible to examine how hippocampal subfields are affected *in vivo* by healthy aging and by neurological and psychiatric disease. This work has shown specific patterns of decline in different subfields due to aging and the progression of AD. However, studies looking at schizophrenia and MDD have yet to reach a consensus about how these diseases might affect individual hippocampus subfields differently. Taking the results of the studies reviewed here, schizophrenia may affect all hippocampal subfields, perhaps with a preference for CA1 in the earlier stages. MDD may be related to volume reductions in dentate gyrus and subiculum, but some studies also find no effects on any hippocampal subfield.

This chapter focused solely on the hippocampus as an anatomical structure and discussed how aging and disease might affect its constituent subfields. The importance of the hippocampus, however, rests in its involvement in cognition, primarily in terms of long-term memory and spatial representation. By looking at variation and change in the anatomy of different hippocampus subfields as a result of aging or disease, we can better understand how different hippocampal subfields might subserve distinct parts of the cognitive roles attributed to the hippocampus. For instance, a commonly accepted model of hippocampal subfield function (McClelland, McNaughton, & O'Reilly, 1995) proposes that the dentate gyrus is responsible

for parsing apart essential details of similar or overlapping stimuli (“pattern separation”), CA3 is responsible for storing and recalling episodic memory information from incomplete cues (“pattern completion”), and CA1 connects the hippocampus to neocortical regions that support the different aspects of a memory. These anatomical–behavioral dissociations suggest that changes that affect one hippocampus subfield would yield distinct behavioral consequences compared with changes that affect a different subfield (i.e., explaining why we might see differences between the cognitive symptoms of different diseases). Hippocampal subfield volumetric studies of aging and disease are important because they connect models of hippocampus function to clinical practice. This works both ways: our understanding of what the subfields are doing might be applicable to a better understanding of aging/disease processes (i.e., uncovering aspects of AD that have been overlooked), and clinical observations might inform our theories of hippocampus function.

Like any developing body of work, the experimental results of hippocampus subfield volumetry are sometimes unclear or even contradictory. Additional studies involving larger sample sizes and finer in-plane scan resolution may help us tease apart what is actually happening and to better understand how potential contradictory findings reported by different groups arose. These shortcomings of the current work are caused by technical as well as practical considerations. For instance, there are limitations to the resolution of structural MRI that limit the precision of previous results. The future development of new methods that can boost the resolution of our images would improve the accuracy of our results and might clarify our understanding of how disease processes actually affect the hippocampus. Further, few hippocampal subfield volumetry studies have been conducted with large sample sizes. Aside from the difficulty of recruiting and imaging patient populations, the process of manual segmentation required for accurate results is both time consuming and requires significant skill, acquired only after extensive training and practice. Differences between the segmentation protocols used might also account for some of the inconsistent results between different studies. The development of more accurate automated hippocampal segmentation methods and of a consensus protocol for hippocampus subfield segmentation will hopefully alleviate these issues.

Acknowledgments

We would like to thank Batool Rizvi for her assistance with the figures.

Author Biographies

Lok-Kin Yeung, PhD, is a postdoctoral research scientist (in the Taub Institute for Research on Alzheimer’s Disease and the Aging Brain) at Columbia University. His research focuses on elucidating the cognitive role of the hippocampus and the medial temporal lobes in memory and perception, particularly in the context of aging and dementia.

Adam M. Brickman, PhD, is an associate professor of neuropsychology (in Neurology, the Taub Institute for Research on Alzheimer’s Disease and the Aging Brain, and the Gertrude H. Sergievsky Center) at Columbia University. His research focuses on using advanced neuroimaging techniques to understand cognitive aging and dementia. He is particularly interested in white matter abnormalities and the intersection between vascular disease and Alzheimer’s disease.

References

- Bobinski, M., Wegiel, J., Wisniewski, H. M., Tarnawski, M., Reisberg, B., Mlodzik, B., ... Miller, D. C. (1995). Atrophy of hippocampal formation subdivisions correlates with stage and duration of Alzheimer disease. *Dementia*, 6(4), 205–210. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/7550600>
- Braak, H., & Braak, E. (1991). Neuropathological staging of Alzheimer-related changes. *Acta Neuropathologica*, 82(4), 239–259. doi:10.1007/BF00308809
- Duvernoy, H. M., Cattin, F., & Risold, P.-Y. (2013). *The human hippocampus: Functional anatomy, vascularization and serial sections with MRI* (4th ed.). New York, NY: Springer.
- Ho, N. F., Iglesias, J. E., Sum, M. Y., Kuswanto, C. N., Sitoh, Y. Y., De Souza, J., ... Holt, D. J. (2017). Progression from selective to general involvement of hippocampal subfields in schizophrenia. *Molecular Psychiatry*, 22(1), 142–152. doi:10.1038/mp.2016.4
- Iglesias, J. E., Augustinack, J. C., Nguyen, K., Player, C. M., Player, A., Wright, M., ... Van Leemput, K. (2015). A computational atlas of the hippocampal formation using ex vivo, ultra-high resolution MRI: Application to adaptive segmentation of in vivo MRI. *NeuroImage*, 115, 117–137. doi:10.1016/j.neuroimage.2015.04.042
- Kerchner, G. A., Bernstein, J. D., Fenesy, M. C., Deutsch, G. K., Saranathan, M., Zeineh, M. M., & Rutt, B. K. (2013). Shared vulnerability of two synaptically-connected medial temporal lobe areas to age and cognitive decline: A seven tesla magnetic resonance imaging study. *Journal of Neuroscience*, 33(42), 16666–16672. doi:10.1523/jneurosci.1915-13.2013
- La Joie, R., Fouquet, M., Mézenge, F., Landeau, B., Villain, N., Mevel, K., ... Chételat, G. (2010). Differential effect of age on hippocampal subfields assessed using a new high-resolution 3T MR sequence. *NeuroImage*, 53(2), 506–514. doi:10.1016/j.neuroimage.2010.06.024
- La Joie, R., Perrotin, A., de La Sayette, V., Egret, S., Dœuvre, L., Belliard, S., ... Chételat, G. (2013). Hippocampal subfield volumetry in mild cognitive impairment, Alzheimer's disease and semantic dementia. *NeuroImage: Clinical*, 3, 155–162. doi:10.1016/j.nicl.2013.08.007
- McClelland, J. L., McNaughton, B. L., & O'Reilly, R. C. (1995). Why there are complementary learning systems in the hippocampus and neocortex: Insights from the successes and failures of connectionist models of learning and memory. *Psychological Review*, 102(3), 419–457. doi:10.1037/0033-295X.102.3.419
- Mueller, S. G., Stables, L., Du, A. T., Schuff, N., Truran, D., Cashdollar, N., & Weiner, M. W. (2007). Measurement of hippocampal subfields and age-related changes with high resolution MRI at 4T. *Neurobiology of Aging*, 28(5), 719–726. doi:10.1016/j.neurobiolaging.2006.03.007
- Ota, M., Sato, N., Hidese, S., Teraishi, T., Maikusa, N., Matsuda, H., ... Kunugi, H. (2017). Structural differences in hippocampal subfields among schizophrenia patients, major depressive disorder patients, and healthy subjects. *Psychiatry Research: Neuroimaging*, 259(October 2016), 54–59. doi:10.1016/j.psychresns.2016.11.002
- Travis, S., Coupland, N. J., Silversone, P. H., Huang, Y., Fujiwara, E., Carter, R., ... Malykhin, N. V. (2015). Dentate gyrus volume and memory performance in major depressive disorder. *Journal of Affective Disorders*, 172, 159–164. doi:10.1016/j.jad.2014.09.048
- Wise, L. E. M., Biessels, G. J., & Geerlings, M. I. (2014). A critical appraisal of the hippocampal subfield segmentation package in FreeSurfer. *Frontiers in Aging Neuroscience*, 6(2), 127–134. doi:10.3389/fnagi.2014.00261
- Wise, L. E. M., Biessels, G. J., Heringa, S. M., Kuijff, H. J., Koek, D. H. L., Luijten, P. R., & Geerlings, M. I. (2014). Hippocampal subfield volumes at 7T in early Alzheimer's disease and normal aging. *Neurobiology of Aging*, 35(9), 2039–2045. doi:10.1016/j.neurobiolaging.2014.02.021
- Yushkevich, P. A., Amaral, R. S. C., Augustinack, J. C., Bender, A. R., Bernstein, J. D., Boccardi, M., ... Zeineh, M. M. (2015). Quantitative comparison of 21 protocols for labeling hippocampal subfields and parahippocampal subregions in in vivo MRI: Towards a harmonized segmentation protocol. *NeuroImage*, 111, 526–541. doi:10.1016/j.neuroimage.2015.01.004

Suggested Reading

- de Flores, R., La Joie, R., Landeau, B., Perrotin, A., Mézenge, F., de La Sayette, V., ... Chételat, G. (2015). Effects of age and Alzheimer's disease on hippocampal subfields: Comparison between manual and freesurfer volumetry. *Human Brain Mapping, 36*, 463–474.
- Pipitone, J., Park, M. T. M., Winterburn, J., Lett, T. A., Lerch, J. P., Pruessner, J. C., ... Chakravarty, M. M. (2014). Multi-atlas segmentation of the whole hippocampus and subfields using multiple automatically generated templates. *NeuroImage, 101*, 494–512. doi:10.1016/j.neuroimage.2014.04.054
- Wisse, L. E. M., Daugherty, A. M., Olsen, R. K., Berron, D., Carr, V. A., Stark, C. E. L., ... la Joie, R. (2017). A harmonized segmentation protocol for hippocampal and parahippocampal subregions: Why do we need one and what are the key goals? *Hippocampus, 27*, 3–11.
- Yushkevich, P. A., Pluta, J. B., Wang, H., Xie, L., Ding, S.-L., Gertje, E. C., ... Wolk, D. A. (2015). Automated volumetry and regional thickness analysis of hippocampal subfields and medial temporal cortical structures in mild cognitive impairment. *Human Brain Mapping, 36*, 258–287.

DTI Tractography Metrics: Fiber Bundle Length

Stephen Correia

*Department of Psychiatry and Human Behavior, Alpert Medical School,
Brown University, Providence, RI, USA*

Diffusion Tensor Imaging

The MRI technique of diffusion tensor imaging (DTI) measures the three-dimensional translational motion of water diffusion (i.e., Brownian motion) in biological tissue (Basser, Matteillo, & LeBihan, 1994a, 1994b; Mori & Zhang, 2006) or non-biological phantom (Hubbard, Zhou, Eichorn, & Parker, 2015; U.S. Patent No. 7,667,458 B2, 2010). The rate of diffusion of a water molecule in biological tissue depends on temperature and its local structural environment.

The translational movement (i.e., diffusion) of a water molecule in the brain varies by tissue type. The probabilistic spatial dispersion of a water molecule over a period of time in a structurally unconstrained setting such as within the lateral ventricles of the human brain is largely random or “isotropic” and can be visualized as a sphere. In contrast, water diffusion in cerebral white matter is constrained by axon membranes, microtubules, and axons sheathed in hydrophobic myelin (Beaulieu, 2002; Weise, 1985). Accordingly, the probabilistic displacement of a molecule in cerebral white matter is generally linear. That is, a water molecule is more likely to move along the axon linearly than across it. This is particularly the case in regions where axons are oriented in a coherent direction (e.g., corpus callosum). In general, axon membranes and fiber packing densities play prominent roles in diffusion anisotropy, whereas myelin appears to play a secondary role (Beaulieu, 2009; LeBihan et al., 2001). In cerebral gray matter, water diffusion is constrained by the less linear membranes of cell bodies and organelles and, accordingly, is more randomly dispersed (less anisotropic).

The foregoing is the context for how DTI provides information about the structural coherence (also referred to as “integrity”) of white matter. Highly coherent fiber bundles, such as the corpus callosum, are highly anisotropic, whereas isotropy typically increases in regions of

white matter bundles that are less coherent. Lower directional coherence can be due to normal anatomic variation such as crossing fibers, large axon diameters, and lower fiber packing density; or to pathological processes such as axon loss, demyelination, and intracellular or extracellular edema (Beaulieu, 2009).

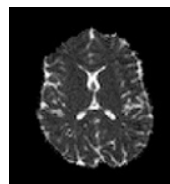
Derivation of DTI Scalar Metrics

Three-dimensional characterization of water motion requires measurement of water diffusion (i.e., diffusion encoding) in a minimum of six orthogonal directions (Buxton, 2002). This means that at least six entire brain volumes are collected, one for each direction. An additional volume is collected with no diffusion encoding to permit calculation of an apparent diffusion coefficient (ADC), based on the signal intensity differences between the diffusion-encoded and non-diffusion-encoded images. The ADC is a measure of water diffusion that takes into account tissue barriers such as cellular membranes and filaments (Mori & Zhang, 2006). After appropriate spatial registration across volumes, each image voxel will have six diffusion values, one for each direction. DTI acquisitions of brain typically involve many more diffusion encoding directions (e.g., >30). More diffusion encoding directions permits better characterization of the underlying white matter. The details of the MRI sequences required for such measurements vary across scanners and research settings and are beyond the scope of this chapter.

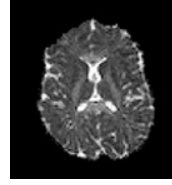
The ADC values in each image voxel can be expressed as a second-order tensor that can then be decomposed into three non-negative eigenvalues ($\lambda_1 > \lambda_2 > \lambda_3$) and their corresponding eigenvectors (ϵ_1 , ϵ_2 , and ϵ) that respectively capture the magnitude and direction of diffusion in each imaging voxel. The eigenvectors are in the frame of reference of the underlying tissue and are therefore independent of the scanner axes. The dominant fiber orientation within an image voxel (ϵ_1) is the orientation with the largest magnitude of diffusion (λ_1) (Basser et al., 1994a, 1994b; Buxton, 2002; Mori & Zhang, 2006).

The eigenvalues can then be mathematically combined into scalar metrics describing various aspects of diffusion in each imaging voxel. Commonly used DTI scalar metrics include mean diffusivity (MD), or the average magnitude of diffusion in each voxel; axial diffusivity (AD or DA) or rate of diffusion in the principal diffusion direction (i.e., λ_1); radial diffusivity (RD) or off-axis diffusivity (i.e., λ_2 , λ_3); and fractional anisotropy (FA) or the degree to which diffusion is directionally restricted in each image voxel. These scalar metrics have values between 0.0 and 1.0. Higher values of FA indicate greater directional restriction of water diffusion (e.g., greater white matter structural coherence), whereas higher MD values indicate greater isotropic diffusion possibly related to lower fiber coherence. MD, RD, and DA are in units of mm^2/s , whereas FA is dimensionless. These scalar metrics are typically displayed as grayscale images. Formulas and sample images for MD, DA, RD, and FA are provided below. Other scalar metrics such as relative anisotropy and volume ratio appear less frequently in published research and will not be discussed further here.

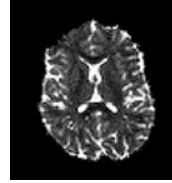
$$\text{MD} = (\lambda_1 + \lambda_2 + \lambda_3)/3$$



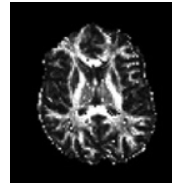
$$AD \text{ (or DA)} = \lambda_1$$



$$RD = (\lambda_2 + \lambda_3)/2$$



$$FA = \sqrt{\frac{1}{2} \frac{(\lambda_1 - \lambda_2)^2 + (\lambda_2 - \lambda_3)^2 + (\lambda_3 - \lambda_1)^2}{\lambda_1^2 + \lambda_2^2 + \lambda_3^2}}$$

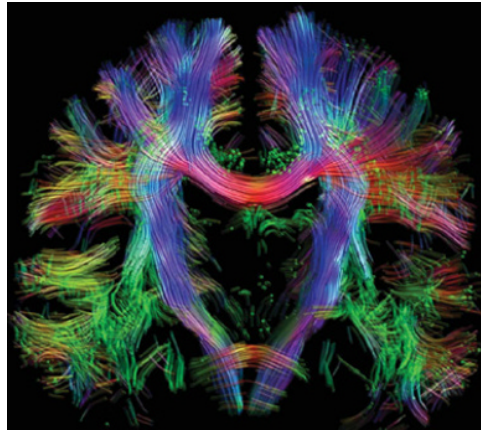


DTI is sensitive to multiple factors that impact the structural integrity of cerebral white matter. This includes developmental maturation (Vertes & Bullmore, 2015) and aging (Marner, Nyengaard, Tang, & Pakkenberg, 2003; McLaughlin et al., 2007; Tang, Nyengaard, Pakkenberg, & Gundersen, 1997) as well as acquired conditions causing white matter injury, edema, demyelination, and degeneration (e.g., traumatic brain injury, ischemia, multiple sclerosis, Alzheimer's disease, etc.). The scalar metrics listed above are sensitive to these and other pathologies causing alterations of white matter structural integrity although the individual metrics are not specific to any particular pathologic process. For example, Wallerian degeneration seen in Alzheimer's disease has been associated with increased diffusivity measures (DA, MD, and RD) and decreased fractional anisotropy (Alves et al., 2012). A similar pattern of change in FA, MD, and RD was found in widespread white matter regions in retired athletes with history of mild traumatic brain injuries (Tremblay et al., 2014). Increased diffusivity has been shown in inflammation (Logyngensky et al., 2010). Increased RD has been shown to be sensitive to myelin changes as in multiple sclerosis (Ontaneda et al., 2014). Myriad other human and animal studies show sensitivity of DTI scalar metrics to various developmental and pathological factors that impact white matter integrity.

DTI data can also be displayed using tractography. This method incorporates the scalar and vector information to provide indirect visual information about the orientation and path of white matter fibers as they course through the cerebrum. The resulting images are visualized *mathematical representations* (i.e., integral curves; a.k.a. streamtubes) of the underlying white matter anatomy based on the acquired diffusion signal.

Typical tractography color-coding convention is that red indicates fibers or tracts running in the left–right direction, blue indicates inferior–superior direction, and green indicates anterior–posterior (Pajevic & Pierpaoli, 1999), although this convention is not always followed.

Tractography can be accomplished based on the diffusion tensor (deterministic tractography, e.g., Mori, Crain, Chacko, & van Zijl, 1999; Zhang, Demiralp, & Laidlaw, 2003) or can be performed without tensor computation using probabilistic methods (probabilistic tractography,



Coronal section through cerebral tractography model.

(https://www.researchgate.net/figure/51021601_fig12_FIG-16-Whole-brain-nontensor-probabilistic-tractography-results-displayed-as-a-coronal)

Behrens, Berg, Jbabdi, Rushworth, & Woolrich, 2007; Behrens, Johansen-Berg, et al., 2003). In deterministic methods, the visualized fibers are actually integral curves that have been propagated through three-dimensional image space based on seed points. The curves follow the spatial orientation of the principal eigenvector. The algorithm is set to terminate curve propagation when FA falls below a prespecified value (often <0.2) chosen to reduce likelihood of fibers entering grey matter, ventricles, or extra-cerebral space. The curves are also terminated when curvature exceeds a predetermined value (typically $\geq 90^\circ$) to avoid image noise-driven errors that cause propagation of curves with anatomically implausible turns.

Probabilistic methods differ from tensor-based approaches. Probabilistic methods incorporate uncertainty into the fiber tracking process using a Bayesian approach that models the diffusion signal. Probabilistic tractography takes into account uncertainty in fiber tracking due to anomalies in the diffusion signal resulting from noise and artifact. The approach produces probability distributions of parameters in a model of the diffusion signal and uses these parameter distributions to produce a tractography fiber representation that reflects the likelihood of a connection between brain regions (Behrens, Woolrich, et al., 2003; Correia & Gongvatana, 2010).

Irrespective of the tractography model, individual tracks (e.g., forceps minor, uncinate fasciculus, corticospinal tract, etc.) can be selected manually using specialized software or can be segmented using prespecified white matter atlases. Once selected, the structural integrity of a tract can be described using summary scalar values (e.g., mean FA, MD, or RD summed across all voxels comprising all fibers of the tract) or by other values such as number of fibers in a bundle, distances between fibers, or FBL.

Correia et al., 2008, demonstrated the potential utility of FBL as a marker of white matter integrity. The rationale for FBL is that even subtle reductions in white matter integrity, such as those related to older age (Marner et al., 2003; Pakkenberg et al., 2003) or very mild cerebrovascular injury that falls below the threshold of visualization on conventional T2-weighted clinical imaging (Moseley, 2002), can cause local alterations in diffusion signal and anisotropy (e.g., reduced FA). Alterations of sufficient magnitude (e.g., $FA < 0.2$) should cause fiber propagation to terminate prematurely, thereby resulting in reduced fiber length (and possibly number) irrespective of the method used to generate the tractography model.

For example, a small lesion with $FA < 0.2$ in the center of the left cingulate bundle should cause fiber propagation to terminate at the boundaries of the lesion, whereas cingulate fibers that circumvent the lesion should propagate the length of the bundle without disruption. For this reason, mean left cingulate FBL should be lower compared with an individual without such lesion. In contrast, mean FA summed across all *propagated* left cingulate fibers could be comparable for both individuals. Accordingly, FBL provides information about white matter integrity that complements other DTI tractography metrics such as average white matter bundle FA or MD.

FBL is subject to the limitations impacting tractography generally, such as ambiguity and possible errors in fiber tracking in regions of fiber crossing. Probabilistic methods and higher dimensional DTI acquisitions involving high numbers of diffusion encoding directions (e.g., 512 directions) help to minimize such errors, thereby improving accuracy of FBL metrics (Yao et al., 2015).

A variety of FBL metrics can be computed (Correia et al., 2008) such as total length (length of each fiber in millimeters summed across all fibers in a white matter bundle, e.g., forceps major) and average FBL (total bundle length divided by the number of fibers in the bundle). Other metrics can be weighted for FA or normalized by white matter volume. Average FBL has now been incorporated into several software analysis packages.

Research Using FBL

Several studies have used FBL to characterize clinical entities. A subset of those studies is reviewed, briefly demonstrating the breadth of applicability of the metric.

Bolzenius et al. (2013) showed age-related declines in mean FBL in frontal, temporal, and whole-brain white matter in 72 healthy adults age 51–85 years (mean age = 63.26 ± 8.76 years). Moreover, they showed that elevated body mass index was associated with shorter mean FBL in the temporal lobes bilaterally independent of age. The authors interpreted the findings as evidence that elevated BMI is adversely associated with cerebral white matter changes.

Behrman-Lay et al. (2015) found shorter mean FBLs were associated with older age and with decreased executive function and cognitive processing speed in a sample of 73 healthy older adults (mean age = 63.56 ± 8.59 years). Specifically, FBL in parietal and occipital regions accounted for a significant portion of the variance in executive function; whereas FBL in frontal, temporal, and occipital regions accounted for a significant amount of variance in cognitive processing speed.

Baker et al. (2016) examined the association between FBL and cognitive reserve in a sample of 63 healthy older adults (mean age = 63.13 ± 8.23 years). Cognitive reserve was determined as estimated IQ, and a total score on a brief cognitive battery was used as a measure of current cognitive status. Shorter whole-brain FBL was related to both lower cognitive reserve and lower current cognitive status. Cognitive reserve moderated the relationship between current cognitive status and FBL in the hippocampal segment of the cingulum bundle. The association between FBL and cognitive status was attenuated in individuals with higher cognitive reserve. The authors concluded that higher cognitive reserve protects older adults against poor cognitive status and against age-related decline in FBL.

Salminen et al. (2013) used FBL to examine the influence of the epsilon 4 ($\epsilon 4$) isoform of the apolipoprotein E (ApoE) gene, a known genetic risk factor for Alzheimer's disease, on temporal lobe white matter integrity. They studied 64 healthy older adults including 24 participants with

at least one ApoE $\epsilon 4$ allele (mean age = 58.78; SD = 6.65) and 51 participants with no $\epsilon 4$ allele (mean age 64.71; SD = 8.60). They also examined memory status using a standard neuropsychological measure. Results showed significantly shorter FBL in the left uncinate fasciculus in $\epsilon 4$ carriers compared with non- $\epsilon 4$ carriers. Overall, results also showed that increasing age correlated with shorter temporal lobe and uncinate FBL and lower memory scores. The authors concluded that ApoE $\epsilon 4$ status alters FBL status in the uncinate fasciculus.

Nakata et al. (2009) used DTI to examine the integrity of the ventral cingulum bundle (VCB), the posterior portion of the cingulum bundle coursing inferolaterally into the infero-medial temporal lobe in patients with partial or complete agenesis of the corpus callosum. Compared with controls, individuals with complete or partial callosal agenesis showed reduced FA in the right VCB and reduced VCB volume bilaterally. Additionally, FBL was reduced bilaterally in the subgroup with complete callosal agenesis. The authors concluded the agenesis of the corpus callosum is associated with abnormalities in intrahemispheric pathways.

FBL has also been examined in animal models of traumatic brain injury and in humans with traumatic brain injury and aneurysmal subarachnoid hemorrhage (e.g., Harris, Verley, Gutman, & Sutton, 2016; Sener et al., 2016). It has also been examined in multiple sclerosis (Li et al., 2013), HIV infection (e.g., Tate et al., 2010), and development (e.g., Wierenga et al., 2016). These applications demonstrate the wide applicability of this metric.

In sum, FBL is a tractography-based metric that provides unique information about cerebral white matter integrity and complements other quantitative DTI metrics. FBL has clinical research utility in conditions known to impact white matter integrity in aging and development.

Author Biography

Dr. Stephen Correia is associate professor in the Department of Psychiatry and Human Behavior of Alpert Medical School at Brown University. He is the Neuropsychology Section Chief at the Providence Veterans Affairs Medical Center (PVAMC) and Director of the Neuroimaging Center in the Memory and Aging Program at Butler Hospital. Dr. Correia's research focuses on the use of diffusion tensor and other imaging modalities to study cerebral white matter integrity in aging, dementia, and in other neurodegenerative and neurobehavioral disorders. In addition, Dr. Correia has a prominent training role as the director of Brown's postdoctoral Clinical Neuropsychology Specialty Program.

References

- Alves, G. S., O'Dwyer, L., Jurcoane, A., Oertel-Knochel, V., Knochel, C., Prvulovic, D., ... Laks, J. (2012). Different patterns of white matter degeneration using multiple diffusion indices and volumetric data in mild cognitive impairment and Alzheimer patients. *PLoS One*, 7(12), e52859.
- Baker, L. M., Laidlaw, D. H., Cabeen, R., Conturo, T., Akbudak, E., Correia, S., ... Paul, R. (2016). Cognitive reserve moderates the relationship between neuropsychological performance and white matter fiber bundle length in healthy older adults. *Brain Imaging and Behavior*. doi:10.1007/s11682-016-9540-7
- Basser, P. J., Mattiello, J., & LeBihan, D. (1994a). Estimation of the effective self-diffusion tensor from the NMR spin echo. *Journal of Magnetic Resonance. Series B*, 103(3), 247–254.
- Basser, P. J., Mattiello, J., & LeBihan, D. (1994b). MR diffusion tensor spectroscopy and imaging. *Biophysical Journal*, 66(1), 259–267.

- Beaulieu, C. (2002). The basis of anisotropic water diffusion in the nervous system—A technical review. *NMR in Biomedicine*, 15(7–8), 435–455.
- Beaulieu, C. (2009). The biologic basis of diffusion anisotropy. In H. Johansen-Berg & T. E. J. Behrens (Eds.), *Diffusion MRI* (pp. 105–126). Academic Press.
- Behrens, T. E., Berg, H. J., Jbabdi, S., Rushworth, M. F., & Woolrich, M. W. (2007). Probabilistic diffusion tractography with multiple fibre orientations: What can we gain? *NeuroImage*, 34(1), 144–155.
- Behrens, T. E., Johansen-Berg, H., Woolrich, M. W., Smith, S. M., Wheeler-Kingshott, C. A. M., Boulby, P. A., ... Thompson, A. J. (2003). Non-invasive mapping of connections between human thalamus and cortex using diffusion imaging. *Nature neuroscience*, 6(7), 750–757.
- Behrens, T. E., Woolrich, M. W., Jenkinson, M., Johansen-Berg, H., Nunes, R. G., Clare, S., ... Smith, S. M. (2003). Characterization and propagation of uncertainty in diffusion-weighted MR imaging. *Magnetic Resonance in Medicine*, 50(5), 1077–1088.
- Behrman-Lay, A. M., Usher, C., Conturo, T. E., Correia, S., Laidlaw, D. H., Lane, E. M., ... Paul, R. H. (2015). Fiber bundle length and cognition: A length-based tractography magnetic resonance imaging study. *Biological Psychiatry*, 9(4), 765–775.
- Bolzenius, J. D., Laidlaw, D. H., Cabeen, R. P., Conturo, T. E., McMichael, A. R., Lane, E. M., ... Paul, R. H. (2013). Impact of body mass index on neuronal fiber bundle lengths among healthy older adults. *Brain Imaging and Behavior*, 7(3), 300–306.
- Buxton, R. (2002). Diffusion and the MR signal. In *Introduction to functional magnetic resonance imaging: Principles & techniques* (pp. 185–216).
- Correia, S., & Gongvatana, A. (2010). Diffusion-tensor imaging in behavioral medicine. In R. A. Cohen & L. H. Sweet (Eds.), *Brain imaging in behavioral medicine and neuropsychology*. New York, NY: Springer.
- Correia, S., Lee, S., Voorn, T., Tate, D., Paul, R., Zhang, S., ... Laidlaw, D. H. (2008). Quantitative tractography metrics of white matter integrity in diffusion-tensor MRI. *NeuroImage*, 42, 568–581.
- Harris, N. G., Verley, D. R., Gutman, B. A., & Sutton, R. L. (2016). Bi-directional changes in fractional anisotropy after experiment TBI: Disorganization and reorganization? *NeuroImage*, 133, 129–143.
- Hubbard, P. L., Zhou, F. L., Eichorn, S. J., & Parker, G. J. (2015). Biometric phantom for the validation of diffusion magnetic resonance imaging. *Magnetic Resonance in Medicine*, 73(1), 299–305.
- LeBihan, D., Mangin, J.-F., Poupon, C., Clark, C. A., Pappata, S., Molko, N., & Chabriet, H. (2001). Diffusion tensor imaging: Concepts and applications. *Journal of Magnetic Resonance Imaging*, 13, 534–546.
- Li, Y., Jewells, V., Kim, M., Chen, Y., Moon, A., Armao, D., ... Shen, D. (2013). Diffusion tensor imaging based network analysis detects alterations of neuroconnectivity in patients with clinically early relapsing-remitting multiple sclerosis. *Human Brain Mapping*, 34(12), 3376–3391.
- Logygensky, G. A., West, T., Stump, M., Holtzman, D. M., Inder, T. E., & Neil, J. (2010). In vivo MRI analysis of an inflammatory injury in the developing brain. *Brain, Behavior, and Immunology*, 24(5), 759–767.
- Marner, L., Nyengaard, J. R., Tang, Y., & Pakkenberg, B. (2003). Marked loss of myelinated nerve fibers in the human brain with age. *Journal of Comparative Neurology*, 462, 144–152.
- McLaughlin, C., Paul, R. H., Grieve, S. M., Williams, L. M., Laidlaw, D., DiCarlo, M., ... Gordon, E. (2007). Diffusion tensor imaging of the corpus callosum: A cross-sectional study across the lifespan. *International Journal of Developmental Neuroscience*, 25(4), 215–221.
- Mori, S., Crain, B. J., Chacko, V. P., & van Zijl, P. C. (1999). Three-dimensional tracking of axonal projections in the brain by magnetic resonance imaging. *Annals of Neurology*, 45(2), 265–269.
- Mori, S., & Zhang, J. (2006). Principles of diffusion tensor imaging and its applications to basic neuroscience research. *Neuron*, 51(5, 7), 527–539.
- Moseley, M. (2002). Diffusion tensor imaging and aging—A review. *NMR in Biomedicine*, 15(7–8), 553–560.

- Nakata, Y., Barkovich, A. J., Wahl, M., Strominger, Z., Jeremy, R. J., Wakahiro, M., ... Sherr, E. H. (2009). Diffusion abnormalities and reduced volume of the ventral cingulum bundle in agenesis of the corpus callosum: A 3T imaging study. *AJNR. American Journal of Neuroradiology*, 30(6), 1142–1148.
- Ontaneda, D., Sakaie, K., Lin, J., Wang, X., Lowe, M. J., Philips, M. D., & Fox, R. J. (2014). Identifying the start of multiple sclerosis: A serial DTI study. (2014). *Journal of Neuroimaging*, 24(6), 569–576.
- Pajevic, S., & Pierpaoli, C. (1999). Color schemes to represent the orientation of anisotropic tissues from diffusion tensor data: Application to white matter fiber tract mapping in the human brain. *Magnetic Resonance in Medicine*, 42(3), 526–540.
- Pakkenberg, B., Pelvig, D., Marner, L., Bundgaard, M. J., Gundersen, H. J., Nyengaard, J. R., & Regeur, L. (2003). Aging and the human cortex. *Experimental Gerontology*, 38(1-2), 95–99.
- Salminen, L. E., Schofield, P. R., Lane, E. M., Heaps, J. M., Pierce, K. D., Cabeen, R., ... Paul, R. (2013). Neuronal fiber bundle lengths in healthy adult carriers of the ApoE4 allele: A quantitative tractography DTI study. *Brain Imaging and Behavior*, 7(3), 274–281.
- Sener, S., Van Hecke, W., Feyen, B. F., Van der Steen, G., Pullens, P., Van de Hauwe, L., ... Maas, A. I. (2016). Diffusion tensor imaging: A possible biomarker in severe traumatic brain injury and aneurysmal subarachnoid hemorrhage? *Neurosurgery*, 79(6), 786–793.
- Tang, Y., Nyengaard, J. R., Pakkenberg, B., & Gundersen, H. J. G. (1997). Age-induced white matter changes in the human brain: A stereological investigation. *Neurobiology of Aging*, 18(6), 609–615.
- Tate, D. F., Conley, J., Paul, R. H., Coop, K., Zhang, S., Zhou, W., ... Tashima, K. (2010). Quantitative diffusion tensor imaging tractography metrics are associated with cognitive performance among HIV-infected patients. *Brain Imaging and Behavior*, 4(1), 68–79.
- Tremblay, S., Henry, L. C., Bedetti, C., Larson-Dupuis, C., Gagnon, J.-F., Evans, A. C., ... De Beaumont, L. (2014). Diffusion white matter tract abnormalities in clinically normal ageing athletes with a history of sports-related concussions. *Brain*, 137(11), 2997–3011.
- Vertes, P. E., & Bullmore, E. T. (2015). Annual research review: Growth connectomics—the organization and reorganization of brain networks during normal and abnormal development. *Journal of Child Psychology and Psychiatry*, 56(3), 299–320.
- Weise, M. J. (1985). Hydrophobic regions in myelin proteins characterized through analysis of “hydrophobic” profiles. *Journal of Neurochemistry*, 44(1), 163–170.
- Wierenga, L. M., van den Heuvel, M. P., van Dijk, S., Rijks, Y., de Reus, M. A., & Durston, S. (2016). The development of brain network architecture. *Human Brain Mapping*, 37(2), 717–729.
- Yao, Y., Yu, T., Liang, B., Xia, T., Huang, Q., & Zhuang, S. (2015). Effect of increasing diffusion gradient direction number on diffusion tensor imaging fiber tracking in the human brain. *Korean Journal of Radiology*, 16(2), 410–418.
- Yoo, D. S., Chang, Y. M., Kim, Y. J., & Kim, S. H. (2010). Phantom for diffusion tensor imaging. *U.S. Patent No. 7,667,458 B2*.
- Zhang, S., Demiralp, C., & Laidlaw, D. (2003). Visualizing diffusion tensor MR images using streamtubes and streamsurfaces. *IEEE Transactions on Visualization and Computer Graphics*, 9(4), 454–462.

The Use of Diffusional Kurtosis Imaging (DKI) to Define Pathological Processes in Stroke and Dementia

Rachael L. Deardorff¹, Jens H. Jensen², and
Joseph A. Helpert³

¹*Center for Biomedical Imaging, Department of Radiology and Radiological Science,
Medical University of South Carolina, Charleston, SC, USA*

²*Center for Biomedical Imaging, Department of Neuroscience, and Department of Radiology
and Radiological Science, Medical University of South Carolina, Charleston, SC, USA*

³*Center for Biomedical Imaging, Department of Neuroscience, Department of Neurology, and
Department of Radiology and Radiological Science, Medical University of South Carolina,
Charleston, SC, USA*

Diffusional Kurtosis Imaging

Molecular diffusion is the random movement of molecules due to thermal agitation. In an unrestricted medium, such as a glass of water, the distance a diffusing water molecule moves over a given time interval is described by a normal or Gaussian probability distribution. Diffusion of this type is referred to as Gaussian diffusion. However in many biological tissues including brain, the presence of cellular membranes, organelles, and other barriers to the water molecules' free movement causes the diffusion process to deviate from the Gaussian behavior of pure water. This deviation can be quantified by a standard statistical metric called the (excess) kurtosis (DeCarlo, 1997). When the kurtosis of the diffusion probability distribution differs significantly from zero, the diffusion dynamics is said to be non-Gaussian. The kurtosis provides a precise measure of the degree of non-Gaussianity and an indirect means of characterizing the tissue microstructure that comprise the diffusion barriers.

Diffusion magnetic resonance imaging (MRI) is a broad category of noninvasive MRI methods that are sensitive to water diffusion and widely applied in both human and animal MRI studies. One of these is diffusional kurtosis imaging (DKI), which as its name suggests is

distinguished by its ability to measure the kurtosis for water diffusion (Jensen & Helpert, 2010). Since the kurtosis is intimately linked to microstructure, this technique is effective in detecting subtle changes, due to disease or injury, which occur in a tissue on a cellular level. When utilizing DKI, one obtains three-dimensional images of an anatomical region of interest that spatially map the kurtosis to a resolution of a few millimeters.

For the brain, there is an added complication that water diffusion is anisotropic. This means that the diffusion probability distribution, and hence the kurtosis, varies with the direction in which it is measured. This is largely a consequence of the brain containing many elongated cellular projections, like axons and dendrites, which steer the water to preferentially diffuse along their axes. Hence the kurtosis becomes a function of orientation as well as spatial location. This orientational dependence of the kurtosis can be summarized by several specific kurtosis metrics. The most commonly used are the mean kurtosis (MK, the kurtosis averaged over all orientations), the axial kurtosis ($K_{\parallel}$, the kurtosis in the orientation for which the diffusion probability distribution is widest), and the radial kurtosis ($K_{\perp}$, the kurtosis averaged over all directions orthogonal to that for which the diffusion probability distribution is widest).

In addition to these kurtosis metrics, DKI also enables the measurement of several other diffusion metrics that are related to the diffusivity, which is simply the mean-square width of

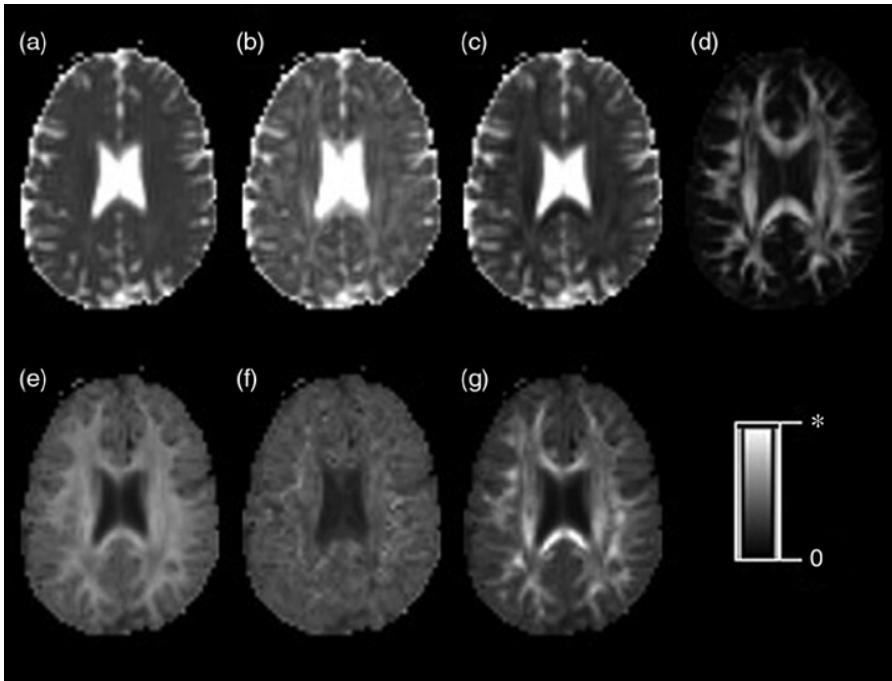


Figure 1 Diffusional kurtosis images of a healthy brain. Each image corresponds to the same anatomical slice. The parameters shown are the mean diffusivity (a), axial diffusivity (b), radial diffusivity (c), fractional anisotropy (d), mean kurtosis (e), axial kurtosis (f), and radial kurtosis (g). The differing contrasts reflect the distinct diffusion properties that each parameter quantifies. The parameters for panels (a), (b), and (c) have units of $\mu\text{m}^2/\text{ms}$, while (d), (e), (f), and (g) are dimensionless. The asterisk (*) by the scale bar indicates the upper limit. This upper limit has a value of 3 for (a), (b), (c), and (g), a value of 2 for (e) and (f), and a value of 1 for (d).

the diffusion probability distribution divided by twice the observation time. Similar to the kurtosis, the diffusivity depends on orientation, allowing one to define a mean diffusivity (MD), axial diffusivity ($D_{\parallel}$), and radial diffusivity ($D_{\perp}$). These diffusivity metrics can also be measured with an alternative diffusion MRI approach known as diffusion tensor imaging (DTI), which is a forerunner to DKI but does not quantify the kurtosis. DTI is one of the oldest and most commonly used diffusion MRI methods (Mori & Zhang, 2006). However, it has important limitations, which has spurred the recent development of more advanced techniques such as DKI.

With this battery of diffusion metrics (i.e., MK, $K_{\parallel}$, $K_{\perp}$, MD, $D_{\parallel}$, and $D_{\perp}$), DKI provides a fairly comprehensive assessment of the diffusion dynamics. Some examples of images corresponding to these metrics are shown in Figure 1. Each metric quantifies a different aspect of diffusion, and the ensemble of metrics is interpreted as a group in order to build up a full picture of this complex molecular process.

Because of the close connection between diffusion and tissue microstructure, DKI enables the *in vivo* detection of microstructural changes associated with pathology, something that is otherwise usually accomplished by microscopic examination of *ex vivo* tissue specimens. As a consequence, DKI is being actively applied to the study of a variety of diseases, particularly in brain (Steven, Zhuo, & Melhem, 2014). However, this work is still at an early stage, with more research being needed to determine the ultimate clinical utility of DKI. Here we review some of this work related to stroke and dementia, two important diseases for which MRI is often used in both clinical and research studies.

Stroke

Ischemic stroke occurs when blood vessels in the brain are blocked by a clot, thereby depriving the downstream tissue of oxygen. This results in a cascade of changes in cell metabolism and structure that can eventually lead to cell death, if blood flow is not quickly restored. It was observed many years ago with diffusion MRI that the water diffusivity decreases sharply following ischemic stroke (Knight et al., 1991; Kucharczyk, Mintorovitch, Asgari, & Moseley, 1991). For this reason diffusion MRI is routinely used clinically for stroke diagnosis (Fung, Roccatagliata, Gonzalez, & Schaefer, 2011). Recently, it has been found with DKI that ischemic stroke greatly increases the kurtosis in the affected areas (Hui, Fieremans, et al., 2012; Jensen et al., 2011). Current research is aimed at determining the meaning and practical value of this finding.

Interestingly, multiple clinical and animal studies have demonstrated that the kurtosis and diffusivity metrics provide complimentary characterization of the damaged regions in ischemic stroke, with the spatiotemporal dynamics of MD and MK being markedly different (Hui, Du, Huang, Shen, & Duong, 2012). In particular, the size of the lesion identified with MK is sometimes smaller than that revealed by the MD changes (Cheung, Wang, Lo, & Sun, 2012). This has led to the hypothesis that the MK may be better in predicting the more seriously damaged tissue (Grinberg, Ciobanu, Farrher, & Shah, 2012). If physicians were able to predict the brain regions most likely to recover, treatment and rehabilitation planning may be more effective. In addition, it has been observed that the kurtosis changes persist longer after the onset of stroke than do those associated with the diffusivity, which could provide clues that might help guide therapy (Weber et al., 2015).

Perhaps the most notable contribution of DKI to stroke research is that it lends compelling support to a recent theory (Budde & Frank, 2010) for the biophysical mechanism behind the

diffusion changes associated with ischemic stroke, which has been a long-standing puzzle in the field. According to this theory, the beading of axons and dendrites is the key process behind the sensitivity of diffusion to stroke. By combining DKI data with a simple model of white matter structure, it was then shown that the model parameters changed after stroke precisely as predicted by Budde and Frank's theory (Hui, Fieremans, et al., 2012). Solving this old riddle may inspire new ideas for improved stroke treatments.

Dementia

The most common cause of dementia is Alzheimer's disease (AD), which is chronic and incurable. It is currently the sixth leading cause of death in the United States. Prior to being diagnosed with AD, some subjects are classified as having mild cognitive impairment (MCI), which means that they have a measurable decline in certain cognitive abilities but not so much as to significantly impact their daily activities. Approximately 10–15% of MCI subjects per year will progress to AD, and MCI is often regarded a precursory or prodromal form of AD. There is great interest in studying the brain changes that occur during MCI, because it is at this early stage of the disease process that potential therapies are believed to have the best chance of being effective.

Recently, several studies have indicated that DKI may be a useful tool for quantifying the microstructural brain changes associated with AD and MCI (Benitez et al., 2014; Falangola et al., 2013; Fieremans et al., 2013; Gong, Wong, Chan, Leung, & Chu, 2013; Struyfs et al., 2015; Vanhoutte et al., 2013; D. Wang, Guo, Liu, Li, & Wang, 2015; Yuan et al., 2016). For example, it has been reported that DKI can distinguish MCI from age-matched controls with a high degree of sensitivity and specificity. In one study, the most accurate parameters were specific combinations of the diffusion metrics that are expected to be especially sensitive to water diffusion taking place in white matter spaces external to the axons (Fieremans et al., 2013). This suggests that subtle structural changes in the extra-axonal white matter occur early in the progression of AD pathology, an observation consistent with at least one theory for the genesis of AD that posits myelin breakdown as playing a central role in AD development (Bartzokis, 2011). Myelin forms sheaths that surround many axons, and a disruption of myelin integrity can plausibly cause the type of diffusion parameter changes detected with DKI. However, it is important to note that the etiology of AD is currently quite controversial, and there is no broad consensus regarding which biological mechanisms are most important.

Looking forward, one possible application of DKI to AD would be to help monitor drug trials. At present, the drugs used for AD improve some symptoms but do not slow down disease progression. DKI and other diffusion MRI methods may eventually prove useful in testing whether new drug therapies are more effective in preserving brain tissue, although work in this direction is still very preliminary. The advantage of using MRI over alternative imaging methods, such as positron emission tomography, is that MRI does not use ionizing radiation and is more widely available.

Besides AD, there is also a limited amount of research applying DKI to other disorders with associated dementia, including Parkinson's and Huntington's diseases (Blockx, De Groof, et al., 2012; Blockx, Verhoye, et al., 2012; Kamagata et al., 2013, 2014; J. J. Wang et al., 2011). Again DKI seems to be quite sensitive to changes in brain microstructure. This fact, together with the relative ease with which DKI can be implemented on clinical MRI scanners, makes DKI a promising tool for yielding new insights and better treatments for this devastating class of maladies.

Future Directions

DKI has yielded encouraging results in several research studies of a variety of diseases, including stroke and dementia. However, several improvements would facilitate its wider use in clinical settings. First, the sensitivity and specificity of DKI-derived diffusion metrics to various pathological processes could be better documented. Second, the DKI image acquisition protocols could be better optimized to accommodate clinical work flows. Third, convenient software tools could be developed that allow the diffusion metrics to be accurately quantified from DKI data without the need of specialized training. DKI is currently a useful research tool that is growing in popularity. As the development of this image method progresses, it may one day become a standard imaging modality available on most clinical MRI systems.

Author Biographies

Rachael L. Deardorff received a MS in biomedical sciences from Medical University of South Carolina and a BS in chemical engineering from Trine University. She is currently a neuroimaging research program coordinator at Indiana University School of Medicine focusing on magnetic resonance image analysis.

Jens H. Jensen received a PhD in physics from Princeton University in 1987 and is currently a Professor of Radiology at the Medical University of South Carolina. His research focuses on the development of advanced magnetic resonance imaging methods for biomedical applications.

Joseph A. Helpert received a MA in chemistry from The University of North Carolina at Chapel Hill and a PhD in medical physics from Oakland University. He currently holds endowed chairs in Brain Imaging and Neurodegenerative Disease at the Medical University of South Carolina. His research focuses on the application of advanced magnetic resonance imaging methods to study brain disease.

References

- Bartzokis, G. (2011). Alzheimer's disease as homeostatic responses to age-related myelin breakdown. *Neurobiology of Aging*, 32(8), 1341–1371. doi:10.1016/j.neurobiolaging.2009.08.007
- Benitez, A., Fieremans, E., Jensen, J. H., Falangola, M. F., Tabesh, A., Ferris, S. H., & Helpert, J. A. (2014). White matter tract integrity metrics reflect the vulnerability of late-myelinating tracts in Alzheimer's disease. *NeuroImage: Clinical*, 4, 64–71. doi:10.1016/j.nicl.2013.11.001
- Blockx, I., De Groof, G., Verhoye, M., Van Audekerke, J., Raber, K., Poot, D., ... Van der Linden, A. (2012). Microstructural changes observed with DKI in a transgenic Huntington rat model: Evidence for abnormal neurodevelopment. *NeuroImage*, 59(2), 957–967. doi:10.1016/j.neuroimage.2011.08.062
- Blockx, I., Verhoye, M., Van Audekerke, J., Bergwerf, I., Kane, J. X., Delgado, Y. P. R., ... Van der Linden, A. (2012). Identification and characterization of Huntington related pathology: An in vivo DKI imaging study. *NeuroImage*, 63(2), 653–662. doi:10.1016/j.neuroimage.2012.06.032
- Budde, M. D., & Frank, J. A. (2010). Neurite beading is sufficient to decrease the apparent diffusion coefficient after ischemic stroke. *Proceedings of the National Academy of Sciences*, 107(32), 14472–14477. doi:10.1073/pnas.1004841107
- Cheung, J. S., Wang, E., Lo, E. H., & Sun, P. Z. (2012). Stratification of heterogeneous diffusion MRI ischemic lesion with kurtosis imaging: Evaluation of mean diffusion and kurtosis MRI mismatch in an animal model of transient focal ischemia. *Stroke*, 43(8), 2252–2254. doi:10.1161/STROKEAHA.112.661926

- DeCarlo, L. T. (1997). On the meaning and use of kurtosis. *Psychological Methods*, 2(3), 292–307.
- Falangola, M. F., Jensen, J. H., Tabesh, A., Hu, C., Deardorff, R. L., Babb, J. S., ... Helpert, J. A. (2013). Non-Gaussian diffusion MRI assessment of brain microstructure in mild cognitive impairment and Alzheimer's disease. *Magnetic Resonance Imaging*, 31(6), 840–846. doi:10.1016/j.mri.2013.02.008
- Fieremans, E., Benitez, A., Jensen, J. H., Falangola, M. F., Tabesh, A., Deardorff, R. L., ... Helpert, J. A. (2013). Novel white matter tract integrity metrics sensitive to Alzheimer disease progression. *AJNR. American Journal of Neuroradiology*, 34(11), 2105–2112. doi:10.3174/ajnr.A3553
- Fung, S. H., Roccatagliata, L., Gonzalez, R. G., & Schaefer, P. W. (2011). MR diffusion imaging in ischemic stroke. *Neuroimaging Clinics of North America*, 21(2), 345–377, xi. doi:10.1016/j.nic.2011.03.001
- Gong, N. J., Wong, C. S., Chan, C. C., Leung, L. M., & Chu, Y. C. (2013). Correlations between microstructural alterations and severity of cognitive deficiency in Alzheimer's disease and mild cognitive impairment: A diffusional kurtosis imaging study. *Magnetic Resonance Imaging*, 31(5), 688–694. doi:10.1016/j.mri.2012.10.027
- Grinberg, F., Ciobanu, L., Farrher, E., & Shah, N. J. (2012). Diffusion kurtosis imaging and log-normal distribution function imaging enhance the visualisation of lesions in animal stroke models. *NMR in Biomedicine*, 25(11), 1295–1304. doi:10.1002/nbm.2802
- Hui, E. S., Du, F., Huang, S., Shen, Q., & Duong, T. Q. (2012). Spatiotemporal dynamics of diffusional kurtosis, mean diffusivity and perfusion changes in experimental stroke. *Brain Research*, 1451, 100–109. doi:10.1016/j.brainres.2012.02.044
- Hui, E. S., Fieremans, E., Jensen, J. H., Tabesh, A., Feng, W., Bonilha, L., ... Helpert, J. A. (2012). Stroke assessment with diffusional kurtosis imaging. *Stroke*, 43(11), 2968–2973. doi:10.1161/STROKEAHA.112.657742
- Jensen, J. H., Falangola, M. F., Hu, C., Tabesh, A., Rapalino, O., Lo, C., & Helpert, J. A. (2011). Preliminary observations of increased diffusional kurtosis in human brain following recent cerebral infarction. *NMR in Biomedicine*, 24(5), 452–457. doi:10.1002/nbm.1610
- Jensen, J. H., & Helpert, J. A. (2010). MRI quantification of non-Gaussian water diffusion by kurtosis analysis. *NMR in Biomedicine*, 23(7), 698–710. doi:10.1002/nbm.1518
- Kamagata, K., Tomiyama, H., Hatano, T., Motoi, Y., Abe, O., Shimoji, K., ... Aoki, S. (2014). A preliminary diffusional kurtosis imaging study of Parkinson disease: Comparison with conventional diffusion tensor imaging. *Neuroradiology*, 56(3), 251–258. doi:10.1007/s00234-014-1327-1
- Kamagata, K., Tomiyama, H., Motoi, Y., Kano, M., Abe, O., Ito, K., ... Hattori, N. (2013). Diffusional kurtosis imaging of cingulate fibers in Parkinson disease: Comparison with conventional diffusion tensor imaging. *Magnetic Resonance Imaging*, 31(9), 1501–1506. doi:10.1016/j.mri.2013.06.009
- Knight, R. A., Ordidge, R. J., Helpert, J. A., Chopp, M., Rodolosi, L. C., & Peck, D. (1991). Temporal evolution of ischemic damage in rat brain measured by proton nuclear magnetic resonance imaging. *Stroke*, 22(6), 802–808.
- Kucharczyk, J., Mintorovitch, J., Asgari, H. S., & Moseley, M. (1991). Diffusion/perfusion MR imaging of acute cerebral ischemia. *Magnetic Resonance in Medicine*, 19(2), 311–315.
- Mori, S., & Zhang, J. (2006). Principles of diffusion tensor imaging and its applications to basic neuroscience research. *Neuron*, 51(5), 527–539. doi:10.1016/j.neuron.2006.08.012
- Steven, A. J., Zhuo, J., & Melhem, E. R. (2014). Diffusion kurtosis imaging: An emerging technique for evaluating the microstructural environment of the brain. *AJR. American Journal of Roentgenology*, 202(1), W26–W33. doi:10.2214/AJR.13.11365
- Struyfs, H., Van Hecke, W., Veraart, J., Sijbers, J., Slaets, S., De Belder, M., ... Engelborghs, S. (2015). Diffusion kurtosis imaging: A possible MRI biomarker for AD diagnosis? *Journal of Alzheimer's Disease*, 48(4), 937–948. doi:10.3233/JAD-150253
- Vanhoutte, G., Pereson, S., Delgado, Y. P. R., Guns, P. J., Asselbergh, B., Veraart, J., ... Van der Linden, A. (2013). Diffusion kurtosis imaging to detect amyloidosis in an APP/PS1 mouse model for Alzheimer's disease. *Magnetic Resonance in Medicine*, 69(4), 1115–1121. doi:10.1002/mrm.24680

- Wang, D., Guo, Z. H., Liu, X. H., Li, Y. H., & Wang, H. (2015). Examination of hippocampal differences between Alzheimer disease, amnesic mild cognitive impairment and normal aging: Diffusion kurtosis. *Current Alzheimer Research*, 12(1), 80–87.
- Wang, J. J., Lin, W. Y., Lu, C. S., Weng, Y. H., Ng, S. H., Wang, C. H., ... Wai, Y. Y. (2011). Parkinson disease: Diagnostic utility of diffusion kurtosis imaging. *Radiology*, 261(1), 210–217. doi:10.1148/radiol.11102277
- Weber, R. A., Hui, E. S., Jensen, J. H., Nie, X., Falangola, M. F., Helpert, J. A., & Adkins, D. L. (2015). Diffusional kurtosis and diffusion tensor imaging reveal different time-sensitive stroke-induced microstructural changes. *Stroke*, 46(2), 545–550. doi:10.1161/STROKEAHA.114.006782
- Yuan, L., Sun, M., Chen, Y., Long, M., Zhao, X., Yin, J., ... Ni, H. (2016). Non-Gaussian diffusion alterations on diffusion kurtosis imaging in patients with early Alzheimer's disease. *Neuroscience Letters*, 616, 11–18. doi:10.1016/j.neulet.2016.01.021

Functional Magnetic Resonance Imaging: fMRI

Tricia Z. King

The Neuroscience Institute, Georgia State University, Atlanta, GA, USA

Magnetic resonance imaging (MRI) is a noninvasive neuroimaging technique that utilizes a standard MRI scanner. Depending on the changing magnetic pulse sequences employed, images are created that are sensitive to tissue properties and tissue types. While structural MRI (e.g., neuroanatomy, volumetrics) and diffusion tensor imaging (e.g., tractography or fractional anisotropy of white matter) use specific pulse sequences to identify neuroanatomical structure, functional magnetic resonance imaging (fMRI) pulse sequences reveal short-term physiological changes associated with active functioning of the brain. The blood oxygenation level-dependent (BOLD) signal reflects a delayed hemodynamic response to neural activity; however, the exact mechanism of the coupling between the brain's activity and the vascular response is not well understood. More specifically, increases or decreases in BOLD response when a participant performs a task are compared with BOLD response during a contrasting task to provide information on changes in oxygen level in voxels over time. These changes in deoxygenated hemoglobin are in response to the metabolic requirements of active neurons during different task processes that involve the extraction of oxygen by active neurons as well as changes in the blood flow and volume. BOLD contrast is the difference in signal images as a function of the amount of deoxygenated hemoglobin. BOLD contrast values are then used as an indirect indicator of neuronal activity.

Within a task-based fMRI experiment, it is critical that the two conditions are as similar as possible to each other and only differ based on the one key property (i.e., the independent variable). If the conditions differ in more than one way, then these additional differences are confounding variables that offer alternative plausible hypotheses, in addition to the original hypothesis for which the study was designed. For that reason, it is critical that comparisons aim to hold as many extraneous variables as possible constant across tasks.

Task-based fMRI studies primarily employ either block or event-related designs. However some designs combine features of each (e.g., mixed design). In a block design, each condition

is presented continuously over a period of time (i.e., block), and then alternating conditions occur across the run. In contrast in an event-related design, stimuli are presented one at a time with variable interstimulus intervals, rather than repeated across a block. Therefore, an event-related design isolates individual trial events in which trial types are randomly intermixed in rapid succession. Event-related fMRI paradigms are sensitive to transient changes to specific events. Block design is also sensitive to transient signal change, but this transient change is averaged over the course of a block. Moreover, block design is most sensitive to sustained changes in activity. Mixed block/event-related fMRI designs have also been employed to characterize these complementary BOLD signal components (Peterson & Dubois, 2012).

Working Memory: N-Back Paradigm

One commonly used block design paradigm is the n-back. It has been used internationally and across a wide age range to better understand working memory. Working memory is a cognitive skill that enables temporary storage and manipulation of remembered information (Baddeley, 1986). Many researchers have established that working memory is a fundamental cognitive process that supports a range of abilities from selective attention to complex decision making and more distal skills of intelligence and academic achievement.

The n-back task requires active monitoring, updating, and manipulation of information. A variety of stimuli have been employed across various sensory modalities using this paradigm. Participants must monitor a series of stimuli and respond yes or no if the stimuli were seen n -trials before. The parametric design typically includes varying loads of 1-, 2-, and 3-back blocks. Depending on the instructions for the respective block, the participant endorses when the stimuli repeat itself n -stimuli before. For example, a letter n-back presents a series of letters (e.g., X, S, S, T, S, T...). For a 1-back block, the participant would endorse yes for the third letter because S repeated itself 1 letter before. During a 2-back block with these same stimuli, the participant would endorse yes for the fifth and sixth letter because the respective letter occurred two stimuli before. During a 3-back block, the participant would endorse yes for the fifth letter because S occurred 3 stimuli back (second stimuli). Researchers interested in examining vigilance within the same paradigm may add 0-back and crosshair blocks. For the 0-back the participant would endorse yes only when a prespecified letter appears. A higher “ n ” value (i.e., 2- and 3-back) represents a higher cognitive load due to higher monitoring, updating, and manipulating demands and thus working memory. In contrast, an “ n ” value of 0 or 1 is conceptualized as a measure of vigilance as it requires primarily monitoring. The most common n-back BOLD contrast employed is 2-back versus 0-back to examine working memory.

Clinical Relevance

Owen, McMillan, Laird, and Bullmore (2005) conducted a meta-analysis of studies with healthy adult participants without neurological or psychiatric conditions and identified a highly consistent frontal and parietal system activation across various stimuli. Given this highly reliable frontoparietal network activated by n-back contrasts, researchers have employed the n-back paradigm to investigate working memory in a wide range of clinical populations. Researchers investigate the BOLD contrast for the clinical population and how it activates similar to the control group (i.e., conjunction analyses) as well as how activations may differ between groups with the BOLD contrast. However, when group differences in BOLD activation (e.g., 2-back versus 0-back) have been identified, it is critical to determine whether BOLD

activation differences are due to (a) higher activations on the 2-back task in the clinical group compared with the control group, with identical 0-back activations; (b) lower activations on the 0-back in the clinical group compared with the control group, with identical activations on the 2-back; or (c) a combination of higher 2-back activation and lower 0-back activation in the clinical group compared with the control group. In addition, establishing the relationship between the percent of BOLD change in an identified ROI with n-back behavioral performance (e.g., accuracy) and with clinical measures of working memory (e.g., performance on clinical measures and self or informant ratings) is also important to consider when researchers are interpreting the results of their fMRI analyses.

For example, in adult survivors of childhood brain tumors and demographically matched controls, conjunction analyses (2-back versus 0-back) revealed a similar distributed frontoparietal network with comparable activations in the right superior parietal lobe, right paracingulate, right insular cortex, left middle frontal gyrus, thalamus, and right lateral cerebellum (King, Na, & Mao, 2015). This finding highlighted the common distributed frontoparietal network employed by both groups. However, the main goal of this research was to investigate if group differences exist. In fact, adult survivors of childhood cerebellar brain tumors had significantly greater BOLD activation in the left superior/middle frontal gyri and left parietal lobe (2-back versus 0-back contrast). In addition, the differences during the 2-back working memory task were due to higher activations in survivors relative to controls, as there were no BOLD differences between groups on the 0-back versus crosshair contrast. Furthermore, the left superior/middle frontal activation was specifically negatively associated with 2-back and 3-back performance as well as clinical measures of working memory, and it was not associated with lower load n-back performance or clinical attention measures. In summary, these findings highlighted that as working memory load increased, and performance declined, it was specifically associated with increased frontal regions of BOLD response and not overall cognitive abilities. This increased activation that is associated with poorer performance likely reflects recruitment of additional cognitive resources for the more challenging task and not compensation or brain reorganization (see Hillary, 2008).

Another example is using the n-back to examine working memory of adolescents with congenital heart disease (King et al., 2017), another population known to have executive function difficulties. The clinical and demographically matched control groups both activated similar frontoparietal working memory networks when using conjunction analyses. Yet, participants with congenital heart disease demonstrated significant differences within the left precuneus and right inferior frontal gyrus relative to controls. These differences appeared to be due to less task-induced deactivation in the congenital heart disease group during working memory and greater task-induced deactivation in the control group. Furthermore, the performance on clinical measures and informant ratings reflected significantly greater working memory and executive function differences. The frontal regions identified by significant group differences were negatively associated with performance suggesting that greater deactivation was associated with improved working memory performance in participants with congenital heart disease. These findings, similar to the interpretation of findings with adult survivors of childhood brain tumor, are consistent with reallocation of processing resources during challenging tasks.

Future Directions

Future directions in fMRI are beginning to include multiple methods of neuroscience and sophisticated neuroimaging analyses. For example, combining fMRI data to include one or more of the following (effective connectivity modeling, resting state functional connectivity,

EEG time course methods, genetics, and/or structural MRI studies (e.g., DTI or MRI volumetric studies)) is appearing more frequently in the literature given the complementary information each distinct approach provides. In addition, more research is examining BOLD activation changes following intervention or over the course of recovery from brain injury. For example, healthy individuals who participated in an intensive working memory training of a dual n-back paradigm demonstrated brain plasticity (i.e., reduced activation selectively for 2- and 3-back conditions) and behavioral accuracy improvements that are not demonstrated in a group of individuals who received a different cognitive training experience (Thompson, Waskom, & Gabrieli, 2016). Furthermore, the two frontoparietal networks of executive control and dorsal attention were similar pre-training in showing load-dependent activation. However, post-training, these two networks were dissociated, and the executive control network no longer evidenced the relationship with WM load. Furthermore, training-specific increases of functional connectivity of the executive control network and the dorsal attention network correlated with improved behavioral performance. Together, these findings highlight the changes in traditional fMRI BOLD activation magnitudes as well as altered network connectivity in response to intensive working memory training. These diverse and complementary methods used together help to highlight the complex and dynamic nature of brain functions and the exciting developments in neuroimaging research that are designed to better understand brain–behavior relationships.

See also

Functional Anatomy

Structural Imaging

Functional Imaging: Functional Connectivity

Emerging Methodologies in Clinical Research: Brain Mapping

Author Biography

Tricia Z. King, PhD, is professor of psychology at the Neuroscience Institute at Georgia State University. She received her PhD from the University of Florida and completed her Clinical Neuropsychology Postdoctoral Fellowship at Brown University Medical School. Her research focuses on optimizing long-term outcomes and discovering what happens to the developing brain and corresponding abilities across the lifespan (e.g., pediatric brain tumor, congenital heart disease). Multiple complementary neuroimaging techniques are employed to investigate the neural mechanisms underlying cognitive and socioemotional abilities of individuals and the role these play in daily life functioning.

References

- Baddeley A. (1986). Oxford psychology series, No. 11. Working memory. New York, NY, USA.
- Hillary, F. (2008). Neuroimaging of working memory dysfunction and the dilemma of brain reorganization hypotheses. *Journal of the International Neuropsychological Society*, 14, 526–534.
- King, T. Z., Na, S., & Mao, H. (2015). Neural underpinnings of working memory in adult survivors of childhood brain tumors. *Journal of the International Neuropsychological Society*, 21(7), 494–505.

- King, T. Z., Smith, K. M., Burns, T. G., Sun, B., Shin, J., Jones, R. A., ... Mahle, W. T. (2017). fMRI investigation of working memory in adolescents with surgically treated congenital heart disease. *Applied Neuropsychology: Child*, 6(1), 7–21.
- Owen, A. M., KM, M. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping*, 25(1), 46–59.
- Peterson, S. E., & Dubis, J. W. (2012). The mixed block/event-related design. *NeuroImage*, 62, 1177–1184.
- Thompson, T. W., Waskom, M. L., & Gabrieli, J. D. (2016). Intensive working memory training produces functional changes in large-scale frontoparietal networks. *Journal of Cognitive Neuroscience*, 28, 575–588.

Resting-State Functional Magnetic Resonance Imaging and Neuropsychiatric Conditions

Carissa L. Philippi¹, Tasheia Floyd², and Gregory Dahl¹

¹*Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA*

²*Washington University School of Medicine, St. Louis, MO, USA*

Introduction

Over the past decade, there has been growing interest in neuroimaging methods that characterize functional and structural connectivity between neighboring brain regions, as well as within and between large-scale neural networks. A particularly promising neuroimaging technique that can be used to examine functional connectivity is resting-state functional magnetic resonance imaging (rs-fMRI). rs-fMRI measures correlations in spontaneous low-frequency fluctuations in the blood oxygen level-dependent (BOLD) response while participants are at rest, thus depicting the functional network architecture of the brain (Power, Schlaggar, & Petersen, 2014 for review). rs-fMRI has been effectively applied to nonclinical as well as clinical populations, in an effort to identify dysfunction in resting-state functional connectivity (rsFC) in large-scale neural circuits across a variety of neurological and psychiatric conditions, such as Alzheimer's disease, depression, and schizophrenia (Greicius, 2008). Because of this capability, it has been suggested that rsFC may serve as a neurobiological marker of pathology to distinguish between various neuropsychiatric disorders (Finn & Constable, 2016; Menon, 2011; Williams, 2017). In this chapter, we present (a) a brief overview of rs-fMRI and analytic techniques to examine rsFC, (b) a review of literature on rsFC in several neuropsychiatric conditions, and (c) the advantages and limitations of using rs-fMRI as an outcome measure to predict treatment response and to assess the usefulness of interventions in clinical studies at the single-subject level. Finally, we highlight future directions for rs-fMRI research in neuropsychiatric populations, including dimensional approaches.

Resting-State fMRI: Methods and Analytic Techniques

rs-fMRI Background

In contrast to task-based fMRI, which measures changes in the BOLD signal evoked in response to specific stimuli (e.g., emotional faces), rs-fMRI measures spontaneous low-frequency fluctuations (<0.1 Hz) in the BOLD signal during periods of relaxed wakefulness (i.e., at rest). During a typical rs-fMRI scan, participants are instructed to keep their thoughts unfocused while remaining alert as they passively view a fixation cross or close their eyes (Power et al., 2014 for review). These different rs-fMRI conditions are generally considered comparable, though research suggests that participants may be more likely to fall asleep in eyes-closed conditions (Power et al., 2014). The average duration of a rs-fMRI scan is about 5–7 minutes.

Biswal, Zerrin Yetkin, Haughton, and Hyde (1995) were the first researchers to perform rsFC using fMRI. In this seminal rs-fMRI study, significant rsFC was observed between homologous brain regions in the sensorimotor cortex as well as other regions of the motor system (Biswal et al., 1995). These results suggested that neural systems involving specific functions, such as movement, could be isolated using functional connectivity methods during rs-fMRI. Subsequent rsFC research has identified numerous resting-state networks associated with sensory, motor, cognitive, and affective processes in healthy populations including sensory (e.g., visual), subcortical (e.g., striatal), and cortical (e.g., default mode, central executive, salience) networks (Power et al., 2014). The rsFC research reviewed later in the chapter will focus on alterations in those cortical, subcortical, and/or limbic resting-state networks consistently associated with neuropsychiatric conditions. In the next section, we briefly review common preprocessing and rsFC connectivity methods.

rs-fMRI Preprocessing and rsFC Methods

As in task-based fMRI, typical preprocessing steps for rs-fMRI data include removing the first several volumes, despiking, slice-time correction, alignment of the rs-fMRI scan with the anatomical image, and spatial smoothing (e.g., Yeo et al., 2011). To remove nonneuronal noise from the rs-fMRI signal, additional preprocessing steps are often performed including nuisance regression, motion correction and/or censoring, and bandpass filtering to isolate the low-frequency BOLD signal (~ 0.009 – 0.1 Hz) (Power et al., 2014 for review). For nuisance regression, signals associated with the ventricles, white matter, and physiological noise (e.g., cardiac and respiratory signal) are typically regressed from the rs-fMRI data (Power et al., 2014). In some cases, global signal regression (i.e., regressing the signal averaged across the whole brain) is also performed to remove noise in the data related to motion or physiological artifacts (Power et al., 2014). To account for individual differences in motion that can alter rsFC, most researchers perform regression for motion across the scan and censor the rs-fMRI data for excessive framewise motion (i.e., volume-to-volume movement >0.2 mm) (Power, Schlaggar, & Petersen, 2015 for review). However, several controversies remain in rs-fMRI preprocessing, such as whether the global signal should be included (Power et al., 2014).

Following preprocessing, functional connectivity analyses are performed on the rs-fMRI data using any of numerous different methodological approaches that have been developed to compute rsFC. For the purposes of this chapter, the rsFC methods discussed below will be those techniques used most frequently in neuropsychiatric populations: seed-based connectivity and independent component analysis (ICA). The first of the rsFC methods is seed-based connectivity, where the average BOLD time course within a seed region of interest (ROI) (e.g., posterior

cingulate cortex) is correlated with the time courses of all other voxels in the brain (e.g., Biswal et al., 1995). One main advantage of seed-based connectivity is ease of interpretation, while a major disadvantage is the potential bias in a priori selection of seed ROIs.

In contrast to seed-based analyses, ICA is a data-driven approach that does not require a priori selection of seed ROIs. In ICA, multivariate statistics are used to isolate maximally independent spatial components, which correspond to individual resting-state networks, such as the default mode network (DMN) (Calhoun, Liu, & Adali, 2009). Each voxel within a spatial component represents the correlation between the BOLD time course in that voxel and the BOLD time course for that component. To identify the known resting-state networks in ICA, a semiautomated method can be used to calculate the maximum overlap between each spatial component and existing resting-state network masks (e.g., Yeo et al., 2011), an approach implemented in the GIFT toolbox (Calhoun et al., 2009). Besides identifying resting-state networks, ICA can also be useful for isolating independent components associated with nonneuronal signals, such as movement or physiological noise (Power et al., 2014). Other rsFC methods, including graph-theoretical and hierarchical clustering algorithms, are reviewed elsewhere (Power et al., 2014).

Compared with task-based fMRI, rs-fMRI has two main advantages in examining large-scale network functioning in neuropsychiatric populations. First, rs-fMRI scans can be acquired relatively quickly, which reduces the total scan duration. Second, rs-fMRI does not require that participants perform a task, which may be ideal for patient populations with cognitive impairments, such as individuals with Alzheimer's disease (Greicius, 2008). In the following sections, we provide a targeted review of rs-fMRI findings in neuropsychiatric conditions including major depression, posttraumatic stress disorder (PTSD), and traumatic brain injury.

rs-fMRI in Neuropsychiatric Conditions

Neuropsychiatric conditions are increasingly being understood as disorders of neural circuits (Menon, 2011; Williams, 2017). A growing body of neuroimaging research has identified rsFC alterations in distinct cortical (e.g., DMN; frontoparietal/central executive network [CEN]; cingulo-opercular/salience network [SN]) and subcortical or limbic (e.g., affective network [AN]) networks across different psychiatric and neurological disorders (Greicius, 2008; Lanius, Frewen, Tursich, Jetly, & McKinnon, 2015; Menon, 2011; Williams, 2017). Briefly, to describe these networks, the DMN consists of the medial prefrontal (mPFC), posterior cingulate/retrosplenial (PCC), and inferior parietal cortices and is associated with an internal focus of attention to self-related thoughts including mind wandering, autobiographical memory, and self-reflection (Buckner, Andrews-Hanna, & Schacter, 2008 for review). The CEN consists of the dorsolateral prefrontal (dlPFC) and lateral superior parietal cortices and is associated with externally directed attention (Corbetta & Shulman, 2002; Fox et al., 2005; Seeley et al., 2007) and may function to initiate top-down attentional control during cognitive tasks (Corbetta & Shulman, 2002). The SN, consisting of anterior insula/frontal operculum, dorsal anterior cingulate/medial prefrontal cortex, and amygdala, is implicated in detecting and integrating salient external stimuli and internal states (Seeley et al., 2007). Thus, alterations in these cortical networks may underlie specific cognitive, affective, and self-reflective symptoms in neurological and psychiatric disorders (Lanius et al., 2015; Menon, 2011; Williams, 2017). Therefore, in our review of rsFC in neuropsychiatric conditions, we focus on changes to the DMN, CEN, and SN, with some mention of subcortical and/or limbic networks.

Major Depressive Disorder

Major depressive disorder (MDD) is one of the most prevalent psychiatric disorders and is associated with a variety of affective and cognitive symptoms, such as dysphoria, anhedonia, and rumination. Neuroscientific research has associated MDD with abnormalities in several subcortical, limbic, and cortical brain regions (Drevets, Price, & Furey, 2008; Mayberg, 2003), with a growing recognition that dysfunction in large-scale neural networks likely plays a critical role in the pathophysiology of the disorder (Drevets et al., 2008; Mayberg, 2003). Using rs-fMRI, considerable progress has been made in identifying network dysfunction in MDD, including in the DMN, CEN, and SN.

Patients with MDD consistently exhibit enhanced rsFC within mPFC regions of the DMN (i.e., ventromedial prefrontal cortex, dorsomedial prefrontal cortex, pregenual ACC) for both seed-based and ICA methods (Mulders, van Eijndhoven, Schene, Beckmann, & Tendolkar, 2015 for review). A recent meta-analysis of 27 seed-based rsFC studies in MDD also provided confirmatory evidence for DMN hyperconnectivity in this population, specifically in the medial prefrontal cortex and the right hippocampus extending into middle temporal gyrus (Kaiser, Andrews-Hanna, Wager, & Pizzagalli, 2015). Research suggests that heightened rsFC within the DMN in MDD may be associated with increased negative self-related thought, such as the rumination typical to MDD (Philippi & Koenigs, 2014 for review).

In MDD, seed-based rsFC analyses using dlPFC seeds of the CEN have consistently shown diminished connectivity with posterior parietal regions of the CEN (Kaiser et al., 2015; Mulders et al., 2015). Kaiser et al. (2015) propose that hypoconnectivity within frontoparietal CEN regions may contribute to cognitive and emotional symptoms in MDD, such as impaired attention and emotion regulation. However, further research is warranted, as ICA studies are inconsistent in this regard (Mulders et al., 2015). In MDD, findings for the SN have been mixed, with evidence for both increased and decreased connectivity of the SN (Kaiser et al., 2015; Mulders et al., 2015). For the AN, MDD patients often exhibit hypoconnectivity, including decreased rsFC between the sgACC and nucleus accumbens of the AN (Kaiser et al., 2015).

Besides within network alterations in rsFC, MDD patients frequently exhibit enhanced connectivity between the DMN and CEN, due to either reduced negative correlations or enhanced positive correlations between these networks in MDD (Kaiser et al., 2015). The DMN and CEN are typically negatively correlated at rest (Fox et al., 2005). Thus, one possible interpretation of these findings in MDD is that regions of CEN are over-recruited to accommodate the elevated focus on self-related thoughts occurring in this condition. In support of this hypothesis, regions within the DMN and CEN are both active during periods of mind wandering (Fox, Spreng, Ellamil, Andrews-Hanna, & Christoff, 2015). Interestingly, neuromodulatory treatments for MDD, including transcranial magnetic stimulation, target stimulation of the dlPFC within the CEN and may exert their therapeutic effects in part by normalizing network interactions between the CEN and DMN (Fox, Buckner, White, Greicius, & Pascual-Leone, 2012). In sum, using a variety of rsFC and meta-analytic methods, research is beginning to identify common dysfunction within and between large-scale neural circuits throughout the brain in MDD. Comparatively fewer rsFC studies have been conducted in PTSD, which is addressed in the next section.

Posttraumatic Stress Disorder

PTSD is a psychiatric condition that may develop after exposure to a traumatic event and is associated with clusters of cognitive and affective symptoms including reexperiencing, hyperarousal, avoidance of trauma-related stimuli, and both negative cognitions and mood

(American Psychiatric Association, 2013). Using rs-fMRI, researchers are beginning to characterize large-scale network dysfunction for cortical, subcortical, and limbic networks in PTSD (Lanius et al., 2015), with primarily case-control analyses (e.g., PTSD versus trauma-exposed groups) and some dimensional analyses (e.g., PTSD symptom severity).

Individuals with PTSD consistently exhibit diminished rsFC within the DMN (e.g., Bluhm et al., 2009; Shang et al., 2014; Sripada et al., 2012), which may be related in part to dissociative symptoms of PTSD (Tursich et al., 2015). By contrast, using a dimensional approach, PTSD severity was associated with greater rsFC within the DMN (Lanius et al., 2010). While seemingly inconsistent, these findings may be at least partially explained by differences in characteristics of the control group (e.g., trauma exposed versus non-trauma exposed). Nevertheless, DMN connectivity may be a particularly robust biomarker of PTSD. A preliminary study, for example, found that rsFC within the DMN 2 days after a motor vehicle accident predicted the onset of PTSD (Qin et al., 2012). Compared with the DMN, few rs-fMRI studies to date have focused on the frontoparietal CEN in PTSD, with one study reporting decreased rsFC within the CEN in PTSD using ICA (Shang et al., 2014). The SN has been associated with both elevated and diminished rsFC in PTSD, primarily for insula and amygdala regions of the SN (Lanius et al., 2015 for review). Based on the findings of elevated SN, Sripada et al. (2012) proposed that the hyperarousal symptoms of PTSD may be specifically related to enhanced rsFC of the SN and more generally may reflect increased attention to salient external/internal stimuli in PTSD. Conversely, a study using ICA to calculate rsFC found that increased hyperarousal was associated with reduced rsFC with the posterior insula of the SN in individuals with PTSD related to childhood trauma (Tursich et al., 2015). Thus, further research is necessary to determine the precise relationship between hyperarousal symptoms and SN rsFC in PTSD.

Besides aberrant within network rsFC, there is also evidence for disrupted interactions between networks, in particular the SN and DMN (e.g., Sripada et al., 2012). In the context of altered SN rsFC in PTSD, abnormal coupling between the SN and DMN may also interfere with the ability to flexibly shift away from external or salient stimuli (Sripada et al., 2012). To test this hypothesis, future research could examine dynamic rsFC using a time window analysis to determine the relative dominance of SN and DMN in PTSD.

A summary of the rsFC findings to date in PTSD suggests that there is evidence for alterations within and between the DMN, CEN, and SN. Given that there are inconsistencies in the direction of the changes in rsFC in PTSD (i.e., increased or decreased), future meta-analyses of rs-fMRI studies for seed-based and ICA methods in PTSD will be required to determine whether specific participant variables contribute to rsFC results in PTSD. Additionally, a dimensional approach may be used to examine whether individual differences in symptom clusters in PTSD, such as hyperarousal or reexperiencing, are associated with overlapping and/or distinct rsFC network variations. Nevertheless, this research suggests that rsFC may represent a viable biomarker that could be used to predict diagnosis of PTSD and to develop targeted treatments, a topic we will consider further in our concluding sections.

Mild Traumatic Brain Injury

Mild traumatic brain injury (mTBI) is often associated with a variety of somatic, cognitive, and emotional symptoms, such as headaches and fatigue, attention and memory impairments, and changes in mood (e.g., symptoms of depression; Levin & Diaz-Arrastia, 2015). Research examining network dysfunction in mTBI has been steadily increasing in recent years using various rsFC approaches, with studies predominantly concentrating on the DMN. Several studies have reported decreased rsFC within the DMN in subacute/acute mTBI, for both mPFC and PCC DMN regions, using seed-based or ICA analyses (Johnson et al., 2012;

Mayer, Mannell, Ling, Gasparovic, & Yeo, 2011; Palacios et al., 2017; Stevens et al., 2012). Increased rsFC within the DMN has also been found in mTBI, though usually when individuals are in the chronic phase of recovery (>6 months' post-injury) (e.g., Palacios et al., 2013; Sharp et al., 2011). Given that reduced DMN connectivity has been reported more frequently for subacute/acute mTBI, it is possible that time since injury could impact rsFC in the DMN. Another potential explanation of differential rsFC of the DMN across studies in mTBI could be due to individual differences in depression symptoms, as depression severity has been consistently associated with increased rsFC within the DMN (Kaiser et al., 2015). However, additional research will be crucial to understand what subject-specific factors, including chronicity and depression, may contribute to rsFC of the DMN after mTBI.

In comparison to the DMN, results for rsFC within the CEN and SN in mTBI have been more inconsistent, with reports of both increased and decreased rsFC in mTBI for both seed-based and ICA methods (Palacios et al., 2017; Shumskaya, Andriessen, Norris, & Vos, 2012; Slobounov et al., 2011; Stevens et al., 2012). While fewer studies have focused on subcortical or limbic connectivity in mTBI, there is some evidence for altered rsFC within hippocampal, striatal, and amygdala networks in mTBI (Shumskaya et al., 2012; Slobounov et al., 2011; Stevens et al., 2012). Analogous to altered between-network rsFC in MDD, studies have consistently demonstrated enhanced rsFC between DMN regions and the lateral prefrontal cortex of the CEN in mTBI (e.g., Johnson et al., 2012; Mayer et al., 2011; Sharp et al., 2011).

Despite some contradictory findings, research to date has identified rsFC alterations across multiple cortical and subcortical/limbic networks in mTBI. Researchers have further proposed that such complex alterations in rsFC in mTBI, in particular for the DMN and SN, may predict specific cognitive and affective symptom profiles in individuals with mTBI (Sharp, Scott, & Leech, 2014). Nevertheless, further research is warranted to understand the relationship between mTBI and large-scale neural network connectivity as it relates to specific physical, cognitive, and emotional symptoms and stages of recovery after brain injury.

Together, the reviewed findings across MDD, PTSD, and mTBI underscore the potential for rsFC analysis of large-scale neural networks to be used as a novel tool for diagnosis, individually targeted treatment plans, and a clinical outcome measure following treatment; these topics will be discussed in the next section.

rsFC as an Outcome Measure for Clinical Studies

Clinical neuroscientific research is increasingly using rsFC as an outcome measure to determine the treatment efficacy of psychotherapeutic, pharmacological, and brain stimulation treatments for neuropsychiatric conditions, with particular emphasis on MDD. Review of rs-fMRI studies in MDD suggests that response to treatment regardless of the type, including brain stimulation methods, may restore aberrant rsFC, in particular within DMN and cortical/limbic networks (Gudayol-Ferré, Però-Cebollero, González-Garrido, & Guàrdia-Olmos, 2015). Fewer studies have measured changes in rsFC after treatment in PTSD, but preliminary work indicates that effective treatment response, such as for alpha neurofeedback, may also normalize rsFC in PTSD, especially within the DMN and SN (Lanius et al., 2015). In PTSD, normalization of the DMN and SN may be associated with specific changes in self-related processes and hyperarousal, respectively. However, future research will be needed to determine the specific relationships between network modulation

after treatment and symptom clusters in individuals with PTSD (Lanius et al., 2015). To our knowledge, few studies have investigated changes in rsFC after treatment for mTBI. Further research in mTBI is warranted as preliminary studies suggest that behavioral and brain stimulation interventions in mTBI improve cognitive symptoms, which would likely modulate large-scale rsFC networks involved in attention and cognitive control, such as the CEN (Barbey et al., 2015).

Challenges and Limitations

While the findings from clinical treatment studies are promising, there are several challenges and limitations to consider in rsFC clinical outcome research for neuropsychiatric conditions. First, it can be difficult to interpret the functional significance of hyper- and hypoconnectivity in rs-fMRI, either within a single network or without reference to other clinical or behavioral measures. Potential solutions to these problems would include characterizing rsFC within and between multiple networks in one study, combining rs-fMRI with task-based fMRI or other imaging modalities (e.g., diffusion tensor imaging), and collecting relevant clinical and behavioral measures. Second, concerns have been raised about individual variability in rsFC, which may lessen the reliability of rsFC measures used to predict clinical outcomes (Finn & Constable, 2016). Though, it seems that this may not be an issue, as recent studies have shown that whole-brain rsFC can accurately discriminate among healthy individuals (Finn & Constable, 2016). Neuroimaging researchers are also working to increase the reliability and validity of rsFC through multicenter projects and large data sharing initiatives, which will further facilitate the use of rsFC metrics as biomarkers at the single-subject level for neuropsychiatric conditions (Finn & Constable, 2016). Thus, despite these current limitations and challenges, progress is being made in identifying rsFC biomarkers to improve personalized treatment and prevention of neuropsychiatric conditions.

Future Directions

Below we highlight key areas of future rsFC research and their potential impact on clinical practice:

- Mapping specific profiles of cognitive, affective, and somatic symptoms onto specific neural circuit dysfunction across different neuropsychiatric conditions. This dimensional approach could help clinicians develop targeted treatments to restore network dysfunction (Lanius et al., 2015; Williams, 2017).
- Applying machine learning algorithms to rsFC data gathered from populations at risk for developing neuropsychiatric conditions, such as schizophrenia or anxiety. This may lead to insights that help predict the onset of a neuropsychiatric condition and/or demarcate the transition from subclinical to clinical symptomatology (Finn & Constable, 2016).
- Developing novel methodological approaches of rs-fMRI to further characterize alterations in rsFC in neuropsychiatric conditions, such as dynamic rsFC (i.e., moment-to-moment variations in rsFC). This may help further differentiate between disorders by enhancing knowledge of large-scale network dysfunction on a shorter time scale, such as when individuals switch between networks.

Author Biographies

Carissa L. Philippi is an assistant professor in the Department of Psychological Sciences at the University of Missouri–St. Louis (UMSL). Her research takes a dimensional approach to understanding the behavioral (e.g., negative self-related thought) and neural correlates of neuropsychiatric conditions, such as depression. For example, in a recent study, she identified associations between subclinical depression severity and resting-state functional connectivity of the anterior cingulate cortex that mirrored neural circuit dysfunction in major depression.

Tasheia Floyd is a second year doctoral student in the Behavioral Neuroscience Program at the UMSL. She graduated from UMSL with her Bachelor of Arts in Psychology in May 2015. Her research interests include examining the neurobiological, behavioral, and emotional consequences of spontaneous imagery and thought (e.g., mind wandering) in healthy and clinical adult and pediatric populations.

Gregory Dahl is a graduate research assistant in the Behavioral Neuroscience Program at UMSL. He completed his undergraduate work in psychology and philosophy in May of 2015 and began working with client data/statistics for a not-for-profit mental health clinic. Upon entry to graduate school, he began working with Professor Philippi on the cognitive and affective neurobiology of self. His research interests include the neurobiology of memory and the cognitive/behavioral/environmental factors that can affect memory.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Barbey, A. K., Belli, A., Logan, A., Rubin, R., Zamroziewicz, M., & Operskalski, J. T. (2015). Network topology and dynamics in traumatic brain injury. *Current Opinion in Behavioral Sciences*, 4, 92–102. doi:10.1016/j.cobeha.2015.04.002
- Biswal, B., Zerrin Yetkin, F., Haughton, V. M., & Hyde, J. S. (1995). Functional connectivity in the motor cortex of resting human brain using echo-planar mri. *Magnetic Resonance in Medicine*, 34(4), 537–541. doi:10.1002/mrm.1910340409
- Bluhm, R. L., Williamson, P. C., Osuch, E. A., Frewen, P. A., Stevens, T. K., Boksman, K., ... Lanius, R. A. (2009). Alterations in default network connectivity in posttraumatic stress disorder related to early-life trauma. *Journal of Psychiatry & Neuroscience*, 34(3), 187–194.
- Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network. *Annals of the New York Academy of Sciences*, 1124(1), 1–38. doi:10.1196/annals.1440.011
- Calhoun, V., Liu, J., & Adali, T. (2009). A review of group ICA for fMRI data and ICA for joint inference of imaging, genetic, and ERP data. *NeuroImage*, 45(1), S163–S172.
- Corbetta, M., & Shulman, G. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews. Neuroscience*, 3(3), 201–215. doi:10.1038/nrn755
- Drevets, W. C., Price, J. L., & Furey, M. L. (2008). Brain structural and functional abnormalities in mood disorders: Implications for neurocircuitry models of depression. *Brain Structure & Function*, 213(1–2), 93–118. doi:10.1007/s00429-008-0189-x
- Finn, E. S., & Constable, R. T. (2016). Individual variation in functional brain connectivity: Implications for personalized approaches to psychiatric disease. *Dialogues in Clinical Neuroscience*, 18(3), 277–287.
- Fox, K. C. R., Spreng, R. N., Ellamil, M., Andrews-Hanna, J. R., & Christoff, K. (2015). The wandering brain: Meta-analysis of functional neuroimaging studies of mind-wandering and related spontaneous thought processes. *NeuroImage*, 111, 611–621. doi:10.1016/j.neuroimage.2015.02.039

- Fox, M. D., Buckner, R. L., White, M. P., Greicius, M. D., & Pascual-Leone, A. (2012). Efficacy of transcranial magnetic stimulation targets for depression is related to intrinsic functional connectivity with the subgenual cingulate. *Biological Psychiatry*, 72(7), 595–603. doi:10.1016/j.biopsych.2012.04.028
- Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., Van Essen, D. C., & Raichle, M. E. (2005). The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proceedings of the National Academy of Sciences of the United States of America*, 102(27), 9673–9678. doi:10.1073/pnas.0504136102
- Greicius, M. (2008). Resting-state functional connectivity in neuropsychiatric disorders. *Current Opinion in Neurology*, 21(4), 424–430. doi:10.1097/WCO.0b013e328306f2c5
- Gudayol-Ferré, E., Peró-Cebollero, M., González-Garrido, A. A., & Guàrdia-Olmos, J. (2015). Changes in brain connectivity related to the treatment of depression measured through fMRI: A systematic review. *Frontiers in Human Neuroscience*, 9(582). doi:10.3389/fnhum.2015.00582
- Johnson, B., Zhang, K., Gay, M., Horovitz, S., Hallett, M., Sebastianelli, W., & Slobounov, S. (2012). Alteration of brain default network in subacute phase of injury in concussed individuals: Resting-state fMRI study. *NeuroImage*, 59(1), 511–518. doi:10.1016/j.neuroimage.2011.07.081
- Kaiser, R. H., Andrews-Hanna, J. R., Wager, T. D., & Pizzagalli, D. A. (2015). Large-scale network dysfunction in major depressive disorder: A meta-analysis of resting-state functional connectivity. *JAMA Psychiatry*. doi:10.1001/jamapsychiatry.2015.0071
- Lanius, R. A., Bluhm, R. L., Coupland, N. J., Hegadoren, K. M., Rowe, B., Théberge, J., ... Brimson, M. (2010). Default mode network connectivity as a predictor of post-traumatic stress disorder symptom severity in acutely traumatized subjects. *Acta Psychiatrica Scandinavica*, 121(1), 33–40. doi:10.1111/j.1600-0447.2009.01391.x
- Lanius, R. A., Frewen, P. A., Tursich, M., Jetly, R., & McKinnon, M. C. (2015). Restoring large-scale brain networks in PTSD and related disorders: A proposal for neuroscientifically-informed treatment interventions. *European Journal of Psychotraumatology*, 6. doi:10.3402/ejpt.v6.27313
- Levin, H. S., & Diaz-Arrastia, R. R. (2015). Diagnosis, prognosis, and clinical management of mild traumatic brain injury. *The Lancet Neurology*, 14(5), 506–517. doi:10.1016/S1474-4422(15)00002-2
- Mayberg, H. S. (2003). Positron emission tomography imaging in depression: A neural systems perspective. *Neuroimaging Clinics of North America*, 13(4), 805–815.
- Mayer, A. R., Mannell, M. V., Ling, J., Gasparovic, C., & Yeo, R. A. (2011). Functional connectivity in mild traumatic brain injury. *Human Brain Mapping*, 32(11), 1825–1835. doi:10.1002/hbm.21151
- Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Sciences*, 15(10), 483–506. doi:10.1016/j.tics.2011.08.003
- Mulders, P. C., van Eijndhoven, P. F., Schene, A. H., Beckmann, C. F., & Tendolkar, I. (2015). Resting-state functional connectivity in major depressive disorder: A review. *Neuroscience & Biobehavioral Reviews*, 56, 330–344. doi:10.1016/j.neubiorev.2015.07.014
- Palacios, E. M., Sala-Llonch, R., Junque, C., Roig, T., Tormos, J. M., Bargallo, N., & Vendrell, P. (2013). Resting-state functional magnetic resonance imaging activity and connectivity and cognitive outcome in traumatic brain injury. *JAMA Neurology*, 70(7), 845–851. doi:10.1001/jamaneurol.2013.38
- Palacios, E. M., Yuh, E. L., Chang, Y.-S., Yue, J. K., Schnyer, D. M., Okonkwo, D. O., ... Mukherjee, P. (2017). Resting-state functional connectivity alterations associated with six-month outcomes in mild traumatic brain injury. *Journal of Neurotrauma*, 34(8), 1546–1557. doi:10.1089/neu.2016.4752
- Philippi, C. L., & Koenigs, M. (2014). The neuropsychology of self-reflection in psychiatric illness. *Journal of Psychiatric Research*, 54, 55–63.
- Power, J. D., Schlaggar, B. L., & Petersen, S. E. (2014). Studying brain organization via spontaneous fMRI signal. *Neuron*, 84(4), 681–696.
- Power, J. D., Schlaggar, B. L., & Petersen, S. E. (2015). Recent progress and outstanding issues in motion correction in resting state fMRI. *NeuroImage*, 105, 536–551. doi:10.1016/j.neuroimage.2014.10.044

- Qin, L. D., Wang, Z., Sun, Y. W., Wan, J. Q., Su, S. S., Zhou, Y., & Xu, J. R. (2012). A preliminary study of alterations in default network connectivity in post-traumatic stress disorder patients following recent trauma. *Brain Research*, 1484, 50–56. doi:10.1016/j.brainres.2012.09.029
- Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., ... Greicius, M. D. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *The Journal of Neuroscience*, 27(9), 2349–2356. doi:10.1523/jneurosci.5587-06.2007
- Shang, J., Lui, S., Meng, Y., Zhu, H., Qiu, C., Gong, Q., ... Zhang, W. (2014). Alterations in low-level perceptual networks related to clinical severity in PTSD after an earthquake: A resting-state fMRI study. *PLoS One*, 9(5), e96834. doi:10.1371/journal.pone.0096834
- Sharp, D. J., Beckmann, C. F., Greenwood, R., Kinnunen, K. M., Bonnelle, V., De Boissezon, X., ... Leech, R. (2011). Default mode network functional and structural connectivity after traumatic brain injury. *Brain*, 134(8), 2233–2247. doi:10.1093/brain/awr175
- Sharp, D. J., Scott, G., & Leech, R. (2014). Network dysfunction after traumatic brain injury. *Nature Reviews. Neurology*, 10(3), 156–166. doi:10.1038/nrneurol.2014.15 Reterived from <http://www.nature.com/nrneurol/journal/v10/n3/abs/nrneurol.2014.15.html#supplementary-information>
- Shumskaya, E., Andriessen, T. M. J. C., Norris, D. G., & Vos, P. E. (2012). Abnormal whole-brain functional networks in homogeneous acute mild traumatic brain injury. *Neurology*, 79(2), 175–182. doi:10.1212/WNL.0b013e31825f04fb
- Slobounov, S. M., Gay, M., Zhang, K., Johnson, B., Pennell, D., Sebastianelli, W., ... Hallett, M. (2011). Alteration of brain functional network at rest and in response to YMCA physical stress test in concussed athletes: RsfMRI study. *NeuroImage*, 55(4), 1716–1727. doi:10.1016/j.neuroimage.2011.01.024
- Sripada, R. K., King, A. P., Welsh, R. C., Garfinkel, S. N., Wang, X., Sripada, C. S., & Liberzon, I. (2012). Neural dysregulation in posttraumatic stress disorder: Evidence for disrupted equilibrium between salience and default mode brain networks. *Psychosomatic Medicine*, 74(9), 904–911. doi:10.1097/PSY.0b013e318273bf33
- Stevens, M. C., Lovejoy, D., Kim, J., Oakes, H., Kureshi, I., & Witt, S. T. (2012). Multiple resting state network functional connectivity abnormalities in mild traumatic brain injury. *Brain Imaging and Behavior*, 6(2), 293–318. doi:10.1007/s11682-012-9157-4
- Tursich, M., Ros, T., Frewen, P. A., Kluetsch, R. C., Calhoun, V. D., & Lanius, R. A. (2015). Distinct intrinsic network connectivity patterns of post-traumatic stress disorder symptom clusters. *Acta Psychiatrica Scandinavica*, 132(1), 29–38. doi:10.1111/acps.12387
- Williams, L. M. (2017). Defining biotypes for depression and anxiety based on large-scale circuit dysfunction: A theoretical review of the evidence and future directions for clinical translation. *Depression and Anxiety*, 34(1), 9–24. doi:10.1002/da.22556
- Yeo, B. T. T., Krienen, F. M., Sepulcre, J., Sabuncu, M. R., Lashkari, D., Hollinshead, M., ... Buckner, R. L. (2011). *The organization of the human cerebral cortex estimated by intrinsic functional connectivity*. *Journal of Neurophysiology*, 106(3), 1125–1165. <https://doi.org/10.1152/jn.00338.2011>

The Event-Related Potential (ERP) Method and Applications in Clinical Research

Amanda C. Eaton^{1,2}, Rebecca A. Meza^{1,2}, Kirk Ballew^{1,2,3},
and Suzanne E. Welcome^{1,2}

¹*Department of Communication, Fontbonne University, St. Louis, MO, USA*

²*Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA*

³*Department of Communication, University of Illinois at Urbana-Champaign College of
Liberal Arts and Sciences, Urbana, IL, USA*

The electroencephalogram (EEG) is a noninvasive and relatively inexpensive technique that records differences in electrical potential from scalp electrodes. The resulting waveform represents time (x -axis) and potential amplitude changes (y -axis). Event-related potentials (ERPs) record voltage fluctuations in the ongoing EEG in response to time-locked stimuli that can be associated with particular cognitive operations. ERPs are sensitive to small voltage changes with precise temporal resolution, making it one of the most widely used methods for studying behavior and cognition.

Clinically, ERPs have been used to examine a variety of disorders. ERP is widely used to test the integrity of both visual and auditory systems. Abnormalities in visual and somatosensory ERPs have been observed in multiple sclerosis, showing promise in detecting degeneration more subtle than MRI can detect (Giesser et al., 1987). ERP markers of Alzheimer's disease are observable during the early stages of disease progression (Polich, 2003). As discussed below, ERP abnormalities have been shown in diverse neurological conditions, and many attempts have been made to relate electrophysiological responses to behavioral disorders. In subsequent sections, this chapter will describe ERP data collection and analysis, associations between ERP components and cognitive processes, and applications in clinical research.

ERP Data Collection and Analysis

Acquisition of the EEG Waveform

In order to record electrical changes on the scalp surface, highly conductive silver chloride electrodes are applied. Voltage changes reflect postsynaptic potentials, and thus, ERPs indirectly measure neurotransmission of large populations of neurons, called “neurogenerators,” that are oriented in the same direction on the order of milliseconds. Roughly 6–10 neurogenerators are simultaneously active at any given time point (Luck, 2005). Waveforms from a given electrode are not exclusive to the cortical area directly below the electrode, rather the weighted sum of all the cortical activity beneath the electrode. Spatial resolution is affected by the number of electrodes in the electrode array, which typically range from 16 to 250 units, such that the greater the array, the easier it is to identify underlying neurogenerators. However, spatial resolution is relatively poor compared with the technique’s outstanding temporal resolution. ERP waveforms can reveal temporal dynamics of specific cognitive behaviors with detailed precision and without requiring participants to provide a motor or verbal response (Key, Dove, & Maguire, 2005).

Post-acquisition Data Processing

The result of data acquisition is a trace of voltage changes over time at each electrode site. In order to perform analyses, these data must be processed. In most experimental paradigms, amplitudes and latencies are averaged over several trials within a subject. Averaging across multiple time windows, called “epochs,” reveals ERPs that would not otherwise be visible on single trials due to trial-to-trial variability and individual differences. Grand averages combining waveforms of a group are reported in most studies. Each subject contributes to statistical differences between groups/conditions. While this simplifies data processing, it also can discount significant variability among subjects and introduce distortions. Latency jitter, differences in cognitive strategies, and differences in cortical folding patterns resulting in opposite polarity for a particular component could arise. Therefore, grand averages are most useful when the sample size of each group is large enough to withstand individual differences without losing the effect of the cognitive operation under investigation. In addition to waveforms, topographic maps plot data at selected time periods from ERP sets to represent areas of relative cortical negative/positive polarity.

Components

ERP components are segments of the waveform, typically associated with particular cognitive operations, which are defined by (a) latency (time point at which they occur in response to the stimuli), (b) polarity (all components are dipoles with positive and negative peaks at some place in the head), (c) scalp location (components can be grouped into “families” but are standalone components of distinct functions that occur at the same time and general location), and (d) amplitude (the strength of the amplitude typically reflects the sensitivity to experimental conditions and indicate the brain has reacted to a stimulus) (Luck, 2005). For example, the N400 component is a negative (N) peak in a waveform occurring around 400 ms post-stimulus presentation and reflects cognitive operations of word finding.

Components are not simply peaks in the waveforms as their names suggest, nor do they always occur at the same time or polarity. Waveforms reflect the sum of negative and positive

amplitudes. Consequently, polarities can appear greater or smaller, and latencies appear longer or shorter in the waveform. Therefore, the duration and amplitude of peaks in a waveform may differ from the duration and amplitude of the components themselves, which could lead to erroneous conclusions about what caused the observed waveform effects (Luck, 2005).

Associations Between ERP Components and Cognitive Processes

ERPs have been extensively used to investigate the neural underpinnings of a variety of cognitive processes. The timing of responses is thought to provide insight into the neural communication and information processing being done. Different cognitive domains tend to focus on different ERP components.

Attention

In order to encode and respond to a sensory stimulus, the brain must first attend to it. Depending on the sensory domain of the stimulus, various attentional mechanisms are elicited at varying latencies post-stimulus onset. ERPs are robust measures for revealing the temporal order of attentional processes because ERP components occur at characteristic times throughout the waveform. By correlating components to cognitive operations of attentional processes, researchers can construct more accurate descriptions of sensory perception.

While visual attention processes generally occur between 80 and 600 ms after stimulus onset, auditory attention processes typically occur even earlier, with latencies of 50–400 ms (Key et al., 2005). Most studies suggest that the visual system first orients to the spatial location of the stimulus, beginning around 80 ms after onset (Hillyard & Anllo-Vento, 1998). In the auditory system, the P1 component (P for *positive* and 1 for *first peak in the waveform*) peaks with a latency of around 50 ms, as the brain actively suppresses irrelevant auditory signals (Key et al., 2005). In both sensory domains, modulations of the P1 and N1 components amplify the sensory signal and reduce the noise of irrelevant stimuli, making the attended stimulus more salient (Lange, 2012). Nonspatial features of visual stimuli (e.g., color, motion, shape) are processed within 100–150 ms, whereas auditory features are processed around 200 ms after onset (Key et al., 2005). Around 150–300 ms, the visual features are integrated, allowing for object recognition. P2 and N2 amplitudes correspond to attention to task-relevant stimuli and inhibition of task-irrelevant stimuli (Chernyshev, Lazarev, Byzgalov, & Novikov, 2015). With a latency of about 300 ms, the P3a component indicates a reorienting of attentional resources as distracting stimuli vie for attention (Horváth, 2014). The P3b amplitude, also occurring approximately 300 ms after stimulus onset, indicates how visual features are encoded into working memory as a meaningful whole (Acqua et al., 2015).

Memory

ERP techniques can be useful to distinguish memory subtypes such as short-term, working, and long-term memory. ERP is also used to investigate emotional memory and false memories. The P300 component is the most commonly studied memory component and is believed to reflect context integration for working memory. ERPs are also sensitive to differences in recognition memory tasks (Rugg & Curran, 2007).

Language

Language integrates a myriad of cognitions including hallmark components of communication—expression (speech and writing), reception (reading and auditory comprehension), and social feedback—as well as attention, memory, and executive function. ERP can capture fine distinctions between these domains and provide key insights into the detailed interaction of these processes. Specifically, ERP has been used extensively to study semantics (word meaning), syntax (word order and grammatical rules), and phonology (speech sounds and the rules that govern sound combinations in a language).

One of the first successful applications of ERP to measure language processing occurred almost four decades ago when Kutas and Hillyard (1980) discovered the N400 component. The N400 is associated with accessing word meaning and is sensitive to semantic violations and incongruences. As words in sentences are encoded, semantic expectations are developed through top-down processing of contextual information. This results in increased ease of lexical access and decreased N400 amplitudes as words fit into plausible sentences. However, when words in a sentence violate the expectation, large N400 amplitudes are observed. The amplitude of the N400 can be modulated by manipulating the plausibility of the sentence stimuli. For example, the sentence “Joe washed dishes with soap and *sugar*” contains a sentence final word (sugar) that violates the expectation (water) and disrupts the meaning of the overall sentence. Differences in N400 latencies and amplitudes have been used for detecting reading and language impairments due to poor semantic processing (Kutas & Hillyard, 1980).

Similar to semantic violations, syntactic violations elicit changes in ERP components. In contrast to the N400 elicited for semantic violations, an asyntactic sentence like “The husband is startled by the emotional rather response of his wife” elicits positive peak around 500 ms after the word “rather” is presented.

Phonetic distinctions for speech can be slight and difficult to distinguish from other auditory processes. Using the ERP technique, researchers are able to detect processing changes through phonological manipulations in order to identify breakdowns in speech/reading comprehension and processing speed.

Applications in Clinical Research

Psychiatric Disorders

ERPs are a powerful tool for discerning breakdowns in neural processes associated with psychiatric dysfunction. Researchers have studied disturbances in the N1, N2, P2, P3, and N400 components in order to characterize deficits associated with schizophrenia. Researchers have also made progress in correctly diagnosing schizophrenia by using ERP data. In one study, machine learning algorithms were able to diagnose schizophrenia with an accuracy of 72.4% using only the N1 and P3 components (Neuhaus, Popescu, Bates, Goldberg, & Malhotra, 2012).

Several studies report reduced amplitudes and increased latencies for a range of ERP components in patients with bipolar disorder (BD) (Andersson, Barder, Hellvin, Løvdahl, & Malt, 2008). For example, patients with bipolar II disorder (BDII) were found to have reduced frontal mismatch negativity (MMN) amplitudes and longer MMN latencies when performing an auditory oddball task, suggesting that patients with BDII have deficits in change detection or pre-attentive stimulus discrimination (Andersson et al., 2008).

Patients with major depressive disorder (MDD) typically exhibit reduced ERP amplitudes for a variety of cognitive tasks, mirroring the decreased interest in activities and fatigue that are

common in MDD. However, patients with MDD exhibit increased ERP amplitudes when they engage in negative behaviors. For example, when asked if a list of positive and negative words applied to them, individuals with MDD had increased P1 amplitudes for negative words (Auerbach, Stanton, Proudfit, & Pizzagalli, 2015).

ERPs have been used to characterize a range of anxiety disorders, including generalized anxiety disorder, panic disorder (PD), obsessive–compulsive disorder (OCD), and posttraumatic stress disorder. Patients with OCD exhibit increased P3 amplitudes for novel auditory stimuli, consistent with attentional biases toward potentially threatening stimuli (Ischebeck, Endrass, Simon, & Kathmann, 2011). By understanding the electrophysiological underpinnings of psychiatric disorders, researchers can better characterize the disorders that could lead to the development of superior diagnostic and treatment practices.

Future Directions

ERP methods remain a strong tool for assessing brain function. Due to stability and reproducibility, ERPs are useful in assessing neuroplastic and functional changes that occur in normal course of recovery from neurological insults following stroke or traumatic brain injury. Additionally, ERPs could be a tool for monitoring changes induced by behavioral or pharmacological treatments (Polich, 2003). Despite the popularity of neuroimaging techniques, integrating temporal precision of electrophysiological measures with spatial localization of hemodynamic methods such as fMRI allows further specification of the brain processes.

ERPs are utilized for brain–computer interface systems (BCIs), which translate electrical signals in the brain to an output device (e.g., button press, keyboard, robotic arm) for communication and controlled movement. These devices are in high demand for patients with severe motor neuron disease, paralysis, and locked-in syndrome. P300-based BCIs are among the most common BCI systems and have been used for individuals with severe physical disabilities due to amyotrophic lateral sclerosis (McCane et al., 2015). Another type of BCI uses the N200 component to select letters and spell out messages. This type of technology could present limitless possibilities for individuals with severe communication and motor deficits.

Author Biographies

Amanda C. Eaton is an assistant professor in communication disorders at Fontbonne University in St. Louis, Missouri. Her research predominantly focuses on aphasia pathophysiology and recovery, electrophysiology and right hemisphere language processes, and cognitive rehabilitation. Amanda worked as a speech language pathologist in adult acute and subacute care and currently teaches courses in dysphagia, motor speech disorders, voice pathology, research methods, and neurogenic communication disorders.

Rebecca A. Meza earned her master of arts degree in psychology, with an emphasis in behavioral neuroscience, from the University of Missouri–St. Louis. Rebecca is interested in applying cognitive neuroscience toward adapting and improving the education system for all learners. Her passion comes from her extensive teaching and tutoring experience; she has worked with students of various ages and economic backgrounds, as well as internationally where she was a Peace Corps Volunteer in West Africa. Rebecca is currently working in the Early Emotional Development Program at the Washington University Medical Campus in St. Louis under the supervision of Dr. Joan Luby.

Kirk Ballew received his Bachelor of Arts in Psychology from Webster University in 2013. He is currently enrolled in a PhD program at the University of Illinois in Urbana-Champaign, where he is studying attention and perception. He is a member of Psi Chi (the international honor society in psychology) and a graduate student affiliate member of the Association for Psychological Science.

Suzanne E. Welcome is an assistant professor in the Department of Psychological Sciences at the University of Missouri–St Louis. Her research interests include the neural substrates of language and reading. More specifically, her work examines individual differences in the cognitive and neural underpinnings of reading.

References

- Acqua, R. D., Dux, P. E., Wyble, B., Doro, M., Sessa, P., Meconi, F., & Jolicoeur, P. (2015). The attentional blink impairs detection and delays encoding of visual information: Evidence from human electrophysiology. *Journal of Cognitive Neuroscience*, 27(4), 720–735.
- Andersson, S., Barder, H. E., Hellvin, T., Løvdaahl, H., & Malt, U. F. (2008). Neuropsychological and electrophysiological indices of neurocognitive dysfunction in bipolar II disorder. *Bipolar Disorders*, 10, 888–899.
- Auerbach, R. P., Stanton, C. H., Proudfit, G. H., & Pizzagalli, D. A. (2015). Self-referential processing in depressed adolescents: A high-density event-related potential study. *Journal of Abnormal Psychology*, 124, 233–245.
- Chernyshev, B. V., Lazarev, I. E., Bryzgalov, D. V., & Novikov, N. A. (2015). Spontaneous attentional performance lapses during the auditory condensation task: An ERP study. *Psychology & Neuroscience*, 8(1), 4.
- Giesser, B. S., Kurtzberg, D., Vaughan, H. G., Arezzo, J. C., Aisen, M. L., Smith, C. R., ... Scheinberg, L. C. (1987). Trimodal evoked potentials compared with magnetic resonance imaging in the diagnosis of multiple sclerosis. *Archives of Neurology*, 44(3), 281–284.
- Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related brain potentials in the study of visual selective attention. *Proceedings of the National Academy of Sciences*, 95(3), 781–787.
- Horváth, J. (2014). Sensory ERP effects in auditory distraction: Did we miss the main event? *Psychological Research*, 78, 339–348.
- Ischebeck, M., Endrass, T., Simon, D., & Kathmann, N. (2011). Auditory novel processing is enhanced in obsessive-compulsive disorder. *Depression and Anxiety*, 28, 915–923.
- Key, A. P. F., Dove, G. O., & Maguire, M. J. (2005). Linking brainwaves to the brain: An ERP primer. *Developmental Neuropsychology*, 27(2), 183–215.
- Kutas, M., & Hillyard, S. A. (1980). Reading senseless sentences: Brain potentials reflect semantic incongruity. *Science*, 207(4427), 203–205.
- Lange, K. (2012). The N1 effect of temporal attention is independent of sound location and intensity: Implications for possible mechanisms of temporal attention. *Psychophysiology*, 49, 1468–1480.
- Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: MIT Press.
- McCane, L. M., Heckman, S. M., McFarland, D. J., Townsend, D., Mak, J. N., Sellers, E. W., ... Vaughan, T. M. (2015). P300-based brain-computer interface (BCI) event-related potentials (ERPs): People with amyotrophic lateral sclerosis (ALS) vs. age-matched controls. *Clinical Neurophysiology*, 126, 2124–2131.
- Neuhaus, A. H., Popescu, F. C., Bates, J. A., Goldberg, T. E., & Malhotra, A. K. (2012). Single-subject classification of schizophrenia using event-related potentials obtained during auditory and visual oddball paradigms. *European Archives of Psychiatry and Clinical Neuroscience*, 263, 241–247.
- Polich, J. (2003). Event related potentials and everyday drugs. *Brain and Cognition*, 53, 45.
- Rugg, M. D., & Curran, T. (2007). Event-related potentials and recognition memory. *Trends in Cognitive Sciences*, 11(6), 251–257.

Suggested Reading

- Bledowski, C., Kadosh, K. C., Wibral, M., Rahm, B., Bittner, R. A., Hoechstetter, K., ... Linden, D. E. J. (2006). Mental chronometry of working memory retrieval: A combined functional magnetic resonance imaging and event-related potentials approach. *The Journal of Neuroscience*, 26(3), 821–829.
- Dehaene-Lambertz, G., Dupoux, E., & Gout, A. (2000). Electrophysiological correlates of phonological processing: A cross-linguistic study. *Journal of Cognitive Neuroscience*, 12(4), 635–647.
- Hong, B., Guo, F., Liu, T., Goa, X., & Gao, S. (2009). N200-Speller using motion-onset visual response. *Clinical Neurophysiology*, 120, 1658–1666.
- Johnson, J. S., & Olshausen, B. A. (2003). Timecourse of neural signatures of object recognition. *Journal of Vision*, 3(7), 499–512.

Magnetic Resonance Spectroscopy (MRS) in Psychiatric Diseases

Alexander P. Lin¹, Sai Merugumala¹, Huijun Liao¹, and Napapon Sailasuta²

¹*Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA*

²*Centre for Addiction and Mental Health, Toronto, ON, Canada*

Introduction

Research in neuroscience has slowly been trying to bridge the gap between understanding the disease processes. Many of the concepts in cognitive psychology and psychiatry are being rewritten as development and application of new technologies further our understanding of the brain processes. A particularly productive field of research has involved medical imaging that utilizes techniques such as proton magnetic resonance spectroscopy (¹H MRS), a non-invasive neurochemical assay that can directly measure neuronal activity, biochemical systems, and metabolic processes utilized by neurons. In this section, each brain chemical that can be directly quantified using ¹H MRS method is covered as well as examples of their roles in psychiatric diseases.

Physics of MRS

¹H MRS is a method typically employed by the organic chemist to define chemical structures; MRS can be applied to the human body, or more specifically to the human brain, providing quantitative and noninvasive studies of brain biochemistry. The physics behind the technique is similar to that utilized by the nuclear magnetic resonance (NMR): briefly, for a head scan, the patient slides inside the radiofrequency (RF) volume head coil that situates inside the bore of the magnet where there is a homogeneous static magnetic field such that free nuclei are oriented parallel and antiparallel to the magnetic field, according to their spin state. The nuclei are then excited by electromagnetic radiation in the form of a pulse sequence via a transmitter.

The nuclei absorb the energy, altering the nuclear spin. As the nuclei precess or ‘relax’ back to their original state, energy is released and detected by the receiver in the RF coil as a free induction decay, which is promptly fast Fourier transformed into the resulting spectra.

Each chemical resonates at established frequencies that upon Fourier transform results in peaks at specific locations, or chemical shifts, along the x -axis. The chemical shift of each chemical is governed by the structure of the chemical, in particular, the grouping of the hydrogen atoms as single or multiple peaks (singlets and multiplets) and proximity to other hydrogen-containing groups (j -coupling). These chemical shifts are often expressed as “parts per million,” which can be confusing as it does not relate to the concentration of the chemical, but historically has been used to express the frequency. The use of this nomenclature is so that it is independent of the field strength of the magnet used. The concentration of the metabolite is expressed along the y -axis as the height of the peak such that higher concentrations result in higher peaks and vice versa. Quantitation of these peak heights can provide an objective measure of brain biochemistry that is similar to a blood test or lab analysis, which can then be used for diagnosis or disease characterization. More importantly, however, the role of each of the chemicals is tied to metabolic and physiological processes within the brain that directly relate to brain processes. The remainder of this chapter will be devoted to the major metabolites that are detected by MRS, along with their findings in psychiatric diseases.

MRS Methods

There are several data acquisition methods available for human application and can be found in several review articles (Alger, 2010). Single-voxel double spin echo MRS (SV PRESS MRS) is by far the method of choice for most human study due to the flexibility of placing the voxel of interest (VOI) anywhere in the brain where there is reasonable magnet homogeneity (Soher, Hurd, Sailasuta, & Barker, 1996). As a simple rule, short echo time of 30–35 ms is commonly used where brain metabolites with short and long T2 relaxation times are observed and can be used for initial disease diagnosis. With a minor modification of the standard PRESS MRS (Bottomley, 1987), there are TE-averaged SV MRS (Hurd et al., 2004), two-dimensional (2D) MRS (Thomas, Binesh, Yue, & DeBruhl, 2001), GluCEST (Cai et al., 2012), and MEGA-PRESS (Mescher, Merkle, Kirsch, Garwood, & Gruetter, 1998). Each of these methods offers MRS detection of metabolites that have low concentrations and strongly overlapped with the metabolites with higher concentrations such as gamma-aminobutyric acid (GABA) and glutathione (GSH). Long echo time(>130ms) multivoxel approach (MRSI) offers an ability to acquire MRS data from multiple voxels at the same time but suffers numerous drawbacks (Tran et al., 2000) such as the inability to detect short T2 relaxation times such as myoinositol, glutamate, and glutathione.

MRS and Brain Chemical

N-Acetylaspartate

N-acetylaspartate (NAA) is an amino acid derivative considered a putative “marker” of viable neurons, axons, and dendrites. Studies demonstrated that NAA was distributed throughout the neuron and axon and its concentration correlated with the number of neurons measured (Urenjak, Williams, Gadian, & Noble, 1992). The ability to quantifiably measure neuron populations allows MRS to provide a diagnostic tool that no other radiological technique can

match: an ability to literally “count” the number of active brain cells using a completely noninvasive and quantitative technique by simply measuring the peak height or area of the NAA resonance in the MRS spectra. This can be utilized in practical ways. For example, every metabolite has a “normal” concentration that generates a pattern of peaks that is the same from person to person unless there is an underlying pathology. Diagnosis with MRS can, therefore, be made by either comparing the numeric values of metabolite concentrations or by recognizing abnormal patterns of peaks in the spectra. With either method, it is the increase or decrease in the concentrations of the metabolites that are diagnostic for neurodegenerative pathology as has been shown across a broad variety of diseases. However, it should also be noted that NAA is a cerebral metabolite that participates in some metabolic processes, and therefore the interpretation of NAA as solely a neuronal marker is somewhat of an oversimplification.

NAA in schizophrenia

A recent meta-review of spectroscopy papers in schizophrenia revealed 103 papers alone that focused on MRS that included a total of 2,067 subjects and 2,115 controls, of which the great majority of the studies focused on treated chronically ill patients (Kraguljac et al., 2012). All papers utilized either the single-voxel (SV) or multivoxel (MRSI) methods. Decreased levels of NAA were found all across the brain including structures such as the hippocampus, thalamus, and frontal and temporal lobes. While the temporal lobes showed the greatest decreases in NAA, the variability of measurements in the temporal lobe was also greater than any other area of the brain. The reason for this variability is due to the susceptibility artifacts that occur due to the air and bone tissue interfaces within this brain region that result in distortions and signal loss. The basal ganglia and frontal lobe showed the most consistent decreases in NAA. While whole-brain NAA measures have not been applied to schizophrenia, these global changes in NAA throughout the schizophrenic brain would indicate that this method would be sensitive to these changes.

NAA in dementia

Given the relationship between NAA and neuronal viability, NAA is a key biomarker for dementia. Dozens of papers have shown decreases in NAA in both cortical and white matter regions of the brain including the posterior cingulate gyrus, temporal lobe, and the occipital lobe (Graff-Radford & Kantarci, 2013). In combination with myoinositol (mI), a putative glial marker, NAA provides up to 90% sensitivity and 95% specificity for distinguishing Alzheimer’s disease (AD) from healthy subjects. In a recent study, the ratio of NAA/mI predicted progression to mild cognitive impairment (MCI) or dementia in 214 subjects, which supports previous findings of reduced NAA in MCI patients. Furthermore, MRS is valuable in differentiating between different types of dementia such as Lewy body, frontal lobe, and vascular dementia. While the temporal lobe is often thought to be the ideal location for detecting AD, it is the posterior cingulate gyrus that has been shown to be the most sensitive to AD. While there may be a neuropathological role for the posterior cingulate cortex in AD, as has been shown in PET studies, this region of the brain is also one of the most homogeneous parts of the brain that results in spectra of superior technical quality, which would also contribute to the diagnostic sensitivity of MRS to AD and other dementias.

NAA in depression/bipolar disorder

Changes in NAA in bipolar disorder (BD) and major depression have also been studied extensively (Capizzano, Jorge, Acion, & Robinson, 2007). Several studies have shown reductions in

NAA/Cr in the temporal lobes as a result of BD. Some studies have also shown reductions in NAA/Cr in the frontal lobe in patients suffering from major depression; however there are also studies that were not able to replicate these results. This may be due to the fact that a majority of the studies used a ratio to Cr despite the fact that some studies have shown that Cr is altered as a result of major depression.

NAA in HIV

¹H MRS has been able to characterize the chemical pathology of HIV-related brain change at various disease stages. Changes in NAA have been found in HIV-positive individuals with numerous HIV-related brain pathologies (Chang et al., 2004). These studies indicate that NAA levels are reduced in both neurologically asymptomatic and impaired HIV-positive patients. This change is likely to be related to neuronal loss and neurocognitive dysfunction. However, NAA is unchanged in acute HIV infection when MRS examinations were performed approximately 14 days after initial infection with elevated choline to creatine ratio (Cho/Cr) (Sailasuta et al., 2012) implicating cellular inflammation in the absence of measurable neuronal injury.

Glutamate and Glutamine

Glutamate (Glu) is an amino acid with several important roles in the brain. First, it is the most abundant excitatory neurotransmitter in the human brain; it plays a major role in neurotransmission where it is released from presynaptic cells and then binds to postsynaptic receptors, thus inducing activation. As a result, Glu has many impacts on neurological and psychiatric diseases. In particular, dysfunction reflected in excessive glutamate Glu release or reduced uptake can lead to an accumulation of Glu, which results in excitotoxicity. This second mechanism not only is key to understanding the underlying pathophysiology of different brain disorders but also provides a potential pathway for disease treatment as there now exist numerous glutamate Glu agonist and antagonist pharmaceutical interventions that can be used to modulate glutamate Glu levels and thus provide potential treatments. Finally, Glu is a key compound in brain metabolism via the citric acid cycle and therefore also tightly coupled to brain energetics.

Metabolically, glutamate (Glu) is stored as glutamine (Gln) in the glia, and the balanced cycling between these two neurochemicals is essential for normal functioning of brain cells. Glu and Gln are compartmentalized in neurons and glia, respectively, and this chemical inter-conversion reflects an important aspect of metabolic interaction between these two types of cells. *In vivo* studies have revealed that the neuronal/glial Glu/Gln cycle is highly dynamic in the human brain and is the major pathway of both neuronal Glu repletion and astroglial Gln synthesis. Gln is released from astrocytes, accrued by neurons, and converted to Glu by the neuron-specific enzyme phosphate-activated glutaminase. Gln is the main precursor for neuronal Glu and GABA, but Glu can also be synthesized *de novo* from tricarboxylic acid (TCA) cycle intermediates.

Glutamate in Schizophrenia

It is thought that schizophrenia is related to a dysfunction in *N*-methyl-D-aspartate (NMDA) receptors, the major subtype of Glu receptors. Evidence from studies of NMDA receptor agonists such as phencyclidine and ketamine has shown decreased Glu levels as well as the

psychotic symptoms observed in schizophrenia (Javitt & Zukin, 1991). A recent meta-review (Marsman et al., 2013) examined 28 publications for a total of 647 patients with schizophrenia compared with 608 healthy controls appears to support this hypothesis by showing evidence of reduced Glu. While several brain regions were explored including the medial frontal region, hippocampus, and thalamus, only significant differences were found in the medial frontal region with reduced Glu. Group-by-age associations showed that Glu decreased at a faster rate with age in patients with schizophrenia compared with controls. While there have been few longitudinal studies of Glu using MRS, the literature appears to reflect that first-episode schizophrenics show increased glutamate, whereas, in the chronic stage of disease, Glu is decreased. This provides the basis for the second hypothesis of excitotoxicity of Glu in schizophrenia. Proponents argue that initially Glu levels are increased leading to excitotoxicity, which in turn leads to neuronal death as reflected by decreases in NAA. In the chronic stages of the diseases, the neuronal loss would also be reflected in decreases in Glu.

Glutamate in Dementia

Several proton spectroscopy studies have demonstrated decreases in Glu in Alzheimer's dementia using traditional MRS methods (Ross, Bluml, Danielson, & Kanamori, 1997) as well as others such as GluCEST (Cai et al., 2012). Perhaps of greatest interest is that some studies in dementia patients have shown how Glu levels can be reversed using pharmaceutical interventions such as galantamine, a cholinesterase inhibitor, which showed that Glu increased after treatment in AD patients.

The role of Glu in AD has also been explored in detail using ^{13}C MRS. The initial study in AD utilized $[1-^{13}\text{C}]$ glucose in patients clinically diagnosed with AD as well as age-matched controls. The results of the study showed reduced Glu neurotransmission as measured by examining the time course of enrichment of Glu via the TCA cycle as well as relative enrichment of Glu to Gln. The time course measurements reflect the neuroenergetics of the brain and effectively measure TCA cycle rates. In AD patients, these rates were found to be reduced. The measure of Glu and Gln enrichment is of particular interest as it is reflective of Glu neurotransmission in itself. For example, if the glial TCA cycle is operating faster, then an enrichment pattern with relatively increased Gln signal versus Glu signal would be expected. In this study, Glu/Gln ratios were found to be significantly decreased, reflective of decreased neurotransmission. More interestingly, when correlated with NAA measures as a surrogate marker for neuronal integrity, significant correlates were found with Glu/Gln ratios, further supporting the argument that this measure may be reflective of Glu neurotransmission in and of itself. As NAA or the number of neurons decreased, Glu/Gln decreased, thus demonstrating that Glu neurotransmission may be decreased as a result in the reduction of the number of functioning brain cells. Sailasuta et al. also found that using $[1-^{13}\text{C}]$ acetate infusion that is preferential to glial glutamate metabolism, the third turn of the TCA cycle, which produces bicarbonate, is significantly slower in AD patients when compared with controls (Sailasuta, Harris, Tran, & Ross, 2011).

Glutamate in Anxiety Disorders

Several studies have demonstrated that Glu is increased in the anterior cingulate cortex as a result of general and social anxiety disorder (Pollack, Jensen, Simon, Kaufman, & Renshaw, 2008) when compared with healthy controls. Excess Glu in anxiety is not only found in group

differences and correlations with anxiety severity. One study showed that when anxiety is induced using cholecystokinin tetrapeptide, Glu levels in the anterior cingulate markedly increase within 2–10 min of the challenge. Furthermore, when patients with anxiety are treated with medications, such as levetiracetam, Glu levels appear to decrease as a result of the treatment.

Glutamate in Depression

A recent meta-review (Luykx et al., 2012) examined 16 publications of Glu in major depressive disorders (MDD) using MRS for a total of 281 patients and 301 controls. The anterior cingulate cortex and the prefrontal cortex were the two primary brain regions examined by most studies. The result of meta-analysis showed that Glu and a combination of Glu and Gln (Glx) were found to be significantly decreased in the anterior cingulate in MDD subjects. However the literature remains mixed. For example, 2D COSY was used to study levels of metabolites in the dorsolateral prefrontal white matter regions to study MDD in the elderly. The study concluded that the depressed subjects had lower levels of NAA and higher levels of Glu/Gln, mI, and phosphoethanolamine (Binesh, Kumar, Hwang, Mintz, & Thomas, 2004).

Perhaps the strongest evidence of the importance of using MRS to measure Glu in MDD comes from neuropharmacological literature where MRS has been used extensively to measure the effects of medication on brain metabolites. A recent review identified 15 articles where MRS was used to assess MDD treatments, of which six specifically studied Glu and/or Glx. These findings showed several studies where Glx levels were initially decreased in MDD subjects, but after treatments such as electroconvulsive therapy and repetitive transcranial magnetic stimulation, Glx levels increased. These studies give rise to the hypothesis that Glu has a role in neuroplasticity (Sanacora, Treccani, & Popoli, 2012), thus clearly demonstrating that Glx is an important component of MDD and that MRS is an effective means by which one can monitor therapeutic interventions.

Glutamate in Bipolar Disorder

Given the role of Glu in psychosis and depression, there is also a body of literature that has explored the use of MRS in BD. Similar hypotheses of Glu neurotoxicity and neuroplasticity arise in the literature for Glu MRS of BD as discussed in a recent review (Gigante et al., 2012), where 17 studies were identified. In most studies, the frontal lobe was the focus of MRS in regions such as the anterior cingulate cortex and the dorsal lateral prefrontal cortex. The meta-analysis showed elevated levels of Glx in BD. It is particularly interesting that this remained the case regardless of whether the patients were medicated. This is in contrast to MDD studies where Glx levels were decreased and modified by treatment.

Glutamate in HIV

Chronic HIV-infected individuals with dementia had lower levels of Glx and Glx/Cr in the frontal white matter, which was associated with neurocognitive performance. With the development of TE-averaged PRESS, where the strongly overlapped Glu resonance can be directly quantified from other metabolites, elevated Glu was reported in the frontal white region of chronic HIV-infected adults (Sailasuta, Shriner, & Ross, 2009) and in perinatally infected youth but lower in the parietal gray matter of HIV-infected individuals with cognitive deficits (Ernst, Jiang, Nakama, Buchthal, & Chang, 2010).

γ -Aminobutyric Acid

The counterpart to Glu is GABA, the major inhibitory neurotransmitter in the brain. Interestingly, in the developing brain, GABA is initially excitatory and later shifts to its inhibitory role as glutamatergic functions develop. The balance between excitation and inhibition is reflected not only in the roles of these two neurotransmitters but also in their metabolism as Glu is a metabolic precursor of GABA. GABA is of particular interest given the availability of drugs that modulate the binding of GABA receptors, such as benzodiazepines, which have helped elucidate the role of GABA across a broad range of psychiatric conditions such as anxiety, memory loss, depression, and pain. Similar to Glu, GABA is difficult to detect using MRS due to its overlap with the resonances of Glu and Gln, therefore requiring specific sequences to measure. Further, the concentration of GABA in the brain ranges from 1.3 to 1.9 mM and therefore is difficult to detect accurately and consistently given its low concentration. A novel metabolite editing method (MEGA-PRESS) has been used successfully to quantify GABA in several brain disorders.

GABA in Schizophrenia

Alterations in GABA neurotransmission underlying the pathophysiology of schizophrenia are evidenced by reduced GABA synthesis as reflected by decreased activity GAD67 in parvalbumin-staining cortical neurons (Gonzalez-Burgos, Hashimoto, & Lewis, 2010). Additionally, GABA_A receptors may be upregulated, possibly reflecting a compensatory response to reduced GABA levels (Jarskog, Miyamoto, & Lieberman, 2007). Due to the relatively recent advances in MRS that have allowed for GABA measurements, MRS studies of GABA in schizophrenia are rapidly evolving. Using MEGA-PRESS, a study of chronic schizophrenics reported GABA to be 10% lower than healthy controls and also shown to correlate with the orientation-specific surround suppression, which is essentially a quantitation of the level of visual inhibition that is thought to be regulated by GABAergic pathways. A more recent study found a similar reduction of GABA in chronic schizophrenia in the anterior cingulate that also correlated with poor attention performance. In a study in early course schizophrenia, decreased GABA levels in the basal ganglia were also found. In contrast, however, another study found increases in GABA/Cr ratios in chronic schizophrenia. These discrepancies may have results from the use of Cr as studies have shown that Cr is reduced in schizophrenia, thus possibly elevating the ratio if the reduction is greater than the reduction in GABA. It is also clear that there are significant medication effects on the GABA measures as another study in schizophrenia showed that the measured GABA concentration in the anterior cingulate was significantly negatively correlated with the dose of antipsychotics. In this study, exclusion of patients on these compounds abolished the statistically significant effects.

GABA in Anxiety

There have only been a handful of studies that have used MRS for measuring GABA in anxiety. Comparing patients with panic disorder and healthy controls, MRS studies have found that GABA is reduced in the occipital cortex, anterior cingulate cortex, and medial prefrontal cortex (Goddard et al., 2001). Interestingly, upon administration of benzodiazepine, GABA levels in the occipital cortex did not change. Other studies however have not found differences in GABA levels in patients with panic disorder in the dorsal prefrontal or ventrolateral prefrontal

regions. Similarly, there were no significant differences in patients with social anxiety disorder (Pollack et al., 2008). It should be noted however that measuring GABA is challenging and can often result in large variability in the measurement, which would result in the lack of significant findings but should not necessarily be interpreted as a lack of change in GABA.

GABA in Depression

A recent review cited 14 papers that utilized MRS to measure GABA levels in MDD where there are general findings of decreased GABA in the occipital cortex, anterior cingulate, and dorsal prefrontal cortex but not in the frontal white matter (Maddock & Buonocore, 2012). Furthermore, the review highlights four studies that examine the effect of treatment on GABA levels and show that treatments of selective serotonin reuptake inhibitors and electroconvulsive therapy both increased levels of GABA in the occipital cortex, although one study of cognitive behavioral therapy did not show increased levels.

GABA in Bipolar Disorder

Two studies have shown that patients with BD exhibit lower GABA in the anterior cingulate, parieto-occipital cortex, and occipital cortex (Brady et al., 2013). A CSI study, however, did not show any significant differences between patients with BD and healthy controls (Kaufman et al., 2009). It is likely that the low SNR of multivoxel MRS approach, as described earlier, resulted in the lack of statistical differences between the two cohorts.

GABA in HIV

Given the complexity of measuring brain GABA using conventional spectroscopy methods due to overlap with other metabolites, there are limited studies that directly measure GABA in HIV-infected individuals. Using a 2D correlated spectroscopy, which can disambiguate the GABA signal, Banakar et al. reported reduced GABA/Cr in chronic HIV-infected children. GABA systems play important roles in HIV infection with several studies documented inhibition of the GABAergic neurotransmission in HIV infection (Feligioni et al., 2003).

Glutathione

The primary role of GSH is as an antioxidant, providing the brain's defense system against damaging reactive oxygen species (ROS) that arise from oxidative stress such as super oxide, nitric oxide, hydrogen peroxide, and free radicals (Dringen, 2000). The damage caused by ROS include DNA modification, lipid peroxidation, and protein modification from which the brain is especially vulnerable due to the large quantity of ROS produced there, availability of lipids, and iron deposition that catalyzes ROS reactions. Oxidative stress is also strongly associated with neuroinflammatory processes (Agostinho, Cunha, & Oliveira, 2010). As a result, compromises in GSH metabolism have been found to play an important role in the pathogenesis across a wide variety of diseases such as neurodegenerative disorders as well as psychiatric disorders.

GSH is synthesized intracellularly by first combining Glu and cysteine to form γ -glutamyl-cysteine via synthetase enzyme followed by addition of glycine as catalyzed by GSH synthetase. Due to the limited supply of Glu, cysteine, and glycine, in different cell types, GSH is synthesized

different between neurons, astrocytes, and glial cells, thus providing an interesting specificity to GSH measures to processes such as neuroinflammation. The thiol group on GSH is a reducing agent in that it can donate an electron to unstable molecules such as ROS. When ROS are present, GSH will reduce ROS by catalysis of glutathione peroxidase (GPx) to form glutathione disulfide (GSSG). GSSG can then regenerate GSH using glutathione reductase. GSSG resonates at a different frequency on the MRS spectrum (Satoh & Yoshioka, 2006), and therefore reductions in GSH concentrations as measured by MRS would provide a putative measure of brain oxidative stress and neuroinflammation. However, it is important to note that defects in GSH metabolism can also reduce concentrations of GSH and disruptions to GSH synthesis or GSH peroxidation can also reflect oxidative stress.

Glutathione in Schizophrenia

A review identified four studies that measure GSH in patients with schizophrenia (Matsuzawa & Hashimoto, 2011). The first study used double quantum coherence (DQC) approach to measure GSH in the medial prefrontal cortex in 14 patients including schizophrenia and schizophreniform disorder and compared them with 14 controls. GSH was found to be significantly lower, which was further confirmed by a decrease in CSF GSH levels. The second study utilized MEGA-PRESS and STEAM in the medial prefrontal cortex in 11 patients with schizophrenia and 9 controls, and although they found decreased GSH, the difference was not statistically significant between the two groups. The third study also utilized MEGA-PRESS in the medial prefrontal cortex, although they too did not find significant group differences between schizophrenic ($n = 20$) and controls ($n = 16$) and significant negative correlations between GSH measures and negative symptoms scores. Finally, the fourth study utilized LCModel with conventional PRESS (TE = 30 ms) in the bilateral temporal cortex in first-episode psychosis ($n = 30$), of which 13 were neuroleptic naïve and had various diagnoses of schizophrenia, schizo psychosis, schizoaffective disorder, and depression with psychotic symptoms. Their findings are surprisingly contrary to previous studies where they show increased GSH in the schizophrenia group when compared with controls. It is important to note however the authors used the criteria of <50% CRLB that indicates that metabolite concentrations could range from zero to twice the estimated concentration. According to the LCModel guidelines, this would imply that the metabolite is undetectable. Coupled with findings of interference of Glu with GSH LCModel measures, it is likely that these results are not reliable.

Glutathione in Dementia

Two recent studies have utilized MRS to measure GSH in dementia. The first study utilized MEGA-PRESS and measured GSH in healthy young and old controls, 11 patients diagnosed with MCI, and 14 patients that suffered from AD (Mandal, Tripathi, & Sugunan, 2012). They explored several different brain regions including the frontal cortex, parietal cortex, hippocampus, and cerebellum. While the study found higher GSH in the young controls versus the elderly controls and patients, there did not seem to be any significant differences between the elderly controls and MCI or AD patients. The second study (Duffy et al., 2014), however, examined a much larger group of subjects (54 MCI and 41 healthy controls) using PRESS (TE = 35 ms) MRS and LCModel in the anterior and posterior cingulate gyri. They found elevated GSH levels in MCI patients when compared with age-matched healthy subjects in both brain regions. Furthermore, they also found the anterior

and posterior cingulate GSH levels negatively correlated with executive and memory function, respectively. The authors hypothesize that the increased GSH may be a neuroprotective effect that arises from the upregulation of GSH in response to oxidative stress onset by MCI.

Glutathione in Bipolar Disorder

Fifty-three patients with BD (13 bipolar I, 25 bipolar II, 15 bipolar spectrum) and 51 age- and gender-matched controls were studied with PRESS (TE = 30 ms) in the anterior cingulate cortex and GSH measured using LCModel (Lagopoulos et al., 2013). In this case, CRLB was reported to be less than 20%. There were no significant differences found in the comparison of bipolar patients and controls, nor were there any significant correlations with clinical scores of depression or mania. The authors conclude that oxidative stress does not play a role in BD. It is interesting that an earlier paper from the same group utilizing the same methods but in a larger cohort ($n = 88$; mixed psychiatric disease including depression, bipolar, and psychosis) found that GSH was accounted for by the 40% of the clusters when cluster analysis was applied (Hermens, Lagopoulos, Naismith, Tobias-Webb, & Hickie, 2012). One of the clusters exhibited higher GSH; however, as a mix of different psychoses, it is difficult to determine the underlying pathophysiology, which might explain this finding.

Conclusion

MRS is a powerful imaging tool that can provide noninvasive, quantitative, and objective measures of several brain disorders. Highly complementary to other imaging tools, MRS can be used in conjunction to better understand the different brain functions such as neurodegeneration (NAA), excitotoxicity (Glu), inhibition (GABA), neuroinflammation (GSH), mitochondrial function (Cr), membrane turnover and demyelination (Cho), and gliosis (mI). In schizophrenia, reduced NAA, increased Gln, and variable Glu (dependent on chronicity, reduced GABA, GSH, and Cr) have been the major findings providing insight into the neuronal function and the influence of the major neurotransmitters upon the disease. In dementia, the primary findings are reduced NAA and Glu and increased mI, which are not only diagnostic for AD but also in predicting progression of dementia as well. Reduced NAA, Glu, and GABA are characteristic of depression. Those results provide an interesting contrast to BD where NAA and GABA are also reduced but Cho and Glu are increased and GSH does not appear to change. Anxiety disorder is probably the least explored by MRS but have shown increased Glu but decreased GABA, thereby demonstrating an imbalance between the major excitatory and inhibitory neurotransmitters in the brain. Numerous studies were reported in HIV infection before the introduction of highly antiretroviral therapy (ART) as well as after ART. In chronic HIV infection neuronal loss (NAA) is evidenced with elevated mI, and Cho confirmed neuronal and glial dysfunction. However, in the treatment naïve early infection (acute), NAA is unchanged indicating the virus inability to penetrate blood–brain barrier. As MRS clinical research is rapidly evolve through the various pulse sequences and post-processing methods that are more and more rapidly becoming available to neuroscientists, psychiatrists, psychologists, and physicians, MRS may eventually cross over into the realm of clinical diagnosis where it would be a valuable tool to provide mechanistic and physiological insight into psychiatric diseases much as genetics and pathophysiology have provided for other diseases.

Author Biographies

Alexander P. Lin, PhD, is the director of the Center for Clinical Spectroscopy in the Department of Radiology at Brigham and Women's Hospital and assistant professor of Radiology at Harvard Medical School. Dr. Lin's research has focused on translating magnetic resonance spectroscopy to the clinic including a strong research focus on traumatic brain injury. Dr. Lin is also an investigator at the Psychiatric Neuroimaging Laboratory at BWH, visiting research associate at the Center of MR Research at the University of Illinois Chicago, and is affiliated with Massachusetts General Hospital and Boston Children's Hospital.

Sai Merugumala, MS, is a clinical research staff working in Dr. Lin's laboratory. He is responsible for day-to-day MRS data processing, coordinate with local collaborators in MRI scanning.

Huijun Liao, BS RT, is a clinical nurse working with Dr. Lin. She is responsible for patients scheduling for MRI scan session and assisting in data processing.

Napapon Sailasuta, PhD, is an independent senior scientist at the Centre for Addiction and Mental Health, University of Toronto, Canada. She is also an associate professor at the Department of Tropical Medicine, University of Hawaii at Manoa. Her clinical research focuses on application of novel MRI and MRS methods to characterize brain abnormalities in psychiatry, infectious disease, and addiction.

References

- Agostinho, P., Cunha, R. A., & Oliveira, C. (2010). Neuroinflammation, oxidative stress and the pathogenesis of Alzheimer's disease. *Current Pharmaceutical Design*, 16(25), 2766–2778.
- Alger, J. R. (2010). Quantitative proton magnetic resonance spectroscopy and spectroscopic imaging of the brain: A didactic review. *Topics in Magnetic Resonance Imaging*, 21(2), 115–128.
- Binesh, N., Kumar, A., Hwang, S., Mintz, J., & Thomas, M. A. (2004). Neurochemistry of late-life major depression: A pilot two-dimensional MR spectroscopic study. *Journal of Magnetic Resonance Imaging*, 20(6), 1039–1045.
- Bottomley, P. A. (1987). Spatial localization in NMR spectroscopy in vivo. *Annals of the New York Academy of Sciences*, 508, 333–348.
- Brady, R. O., Jr., JM, M. C., Prescot, A. P., Jensen, J. E., Cooper, A. J., Cohen, B. M., ... Ongur, D. (2013). Brain gamma-aminobutyric acid (GABA) abnormalities in bipolar disorder. *Bipolar Disorders*, 15(4), 434–439.
- Cai, K., Haris, M., Singh, A., Kogan, F., Greenberg, J. H., Hariharan, H., ... Reddy, R. (2012). Magnetic resonance imaging of glutamate. *Nature Medicine*, 18(2), 302–306.
- Capizzano, A. A., Jorge, R. E., Acion, L. C., & Robinson, R. G. (2007). In vivo proton magnetic resonance spectroscopy in patients with mood disorders: A technically oriented review. *Journal of Magnetic Resonance Imaging*, 26(6), 1378–1389.
- Chang, L., Lee, P. L., Yiannoutsos, C. T., Ernst, T., Marra, C. M., Richards, T., ... Navia, B. A. (2004). A multicenter in vivo proton-MRS study of HIV-associated dementia and its relationship to age. *NeuroImage*, 23(4), 1336–1347.
- Dringen, R. (2000). Glutathione metabolism and oxidative stress in neurodegeneration. *European Journal of Biochemistry*, 267(16), 4903.
- Duffy, S. L., Lagopoulos, J., Hickie, I. B., Diamond, K., Graeber, M. B., Lewis, S. J., & Naismith, S. L. (2014). Glutathione relates to neuropsychological functioning in mild cognitive impairment. *Alzheimer's & Dementia*, 10(1), 67–75.
- Ernst, T., Jiang, C. S., Nakama, H., Buchthal, S., & Chang, L. (2010). Lower brain glutamate is associated with cognitive deficits in HIV patients: A new mechanism for HIV-associated neurocognitive disorder. *Journal of Magnetic Resonance Imaging*, 32(5), 1045–1053.

- Feligioni, M., Raiteri, L., Pattarini, R., Grilli, M., Bruzzone, S., Cavazzani, P., ... Pittaluga, A. (2003). The human immunodeficiency virus-1 protein Tat and its discrete fragments evoke selective release of acetylcholine from human and rat cerebrocortical terminals through species-specific mechanisms. *The Journal of Neuroscience*, 23(17), 6810–6818.
- Gigante, A. D., Bond, D. J., Lafer, B., Lam, R. W., Young, L. T., & Yatham, L. N. (2012). Brain glutamate levels measured by magnetic resonance spectroscopy in patients with bipolar disorder: A meta-analysis. *Bipolar Disorders*, 14(5), 478–487.
- Goddard, A. W., Mason, G. F., Almai, A., Rothman, D. L., Behar, K. L., Petroff, O. A., ... Krystal, J. H. (2001). Reductions in occipital cortex GABA levels in panic disorder detected with 1h-magnetic resonance spectroscopy. *Archives of General Psychiatry*, 58(6), 556–561.
- Gonzalez-Burgos, G., Hashimoto, T., & Lewis, D. A. (2010). Alterations of cortical GABA neurons and network oscillations in schizophrenia. *Current Psychiatry Reports*, 12(4), 335–344.
- Graff-Radford, J., & Kantarci, K. (2013). Magnetic resonance spectroscopy in Alzheimer's disease. *Neuropsychiatric Disease and Treatment*, 9, 687–696.
- Hermens, D. F., Lagopoulos, J., Naismith, S. L., Tobias-Webb, J., & Hickie, I. B. (2012). Distinct neurometabolic profiles are evident in the anterior cingulate of young people with major psychiatric disorders. *Translational Psychiatry*, 2, e110.
- Hurd, R., Sailasuta, N., Srinivasan, R., Vigneron, D. B., Pelletier, D., & Nelson, S. J. (2004). Measurement of brain glutamate using TE-averaged PRESS at 3T. *Magnetic Resonance in Medicine*, 51(3), 435–440.
- Jarskog, L. F., Miyamoto, S., & Lieberman, J. A. (2007). Schizophrenia: New pathological insights and therapies. *Annual Review of Medicine*, 58, 49–61.
- Javitt, D. C., & Zukin, S. R. (1991). Recent advances in the phencyclidine model of schizophrenia. *The American Journal of Psychiatry*, 148(10), 1301–1308.
- Kaufman, R. E., Ostacher, M. J., Marks, E. H., Simon, N. M., Sachs, G. S., Jensen, J. E., ... Pollack, M. H. (2009). Brain GABA levels in patients with bipolar disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 33(3), 427–434.
- Kraguljac, N. V., Reid, M., White, D., Jones, R., den Hollander, J., Lowman, D., & Lahti, A. C. (2012). Neurometabolites in schizophrenia and bipolar disorder—A systematic review and meta-analysis. *Psychiatry Research*, 203(2–3), 111–125.
- Lagopoulos, J., Hermens, D. F., Tobias-Webb, J., Duffy, S., Naismith, S. L., White, D., ... Hickie, I. B. (2013). In vivo glutathione levels in young persons with bipolar disorder: A magnetic resonance spectroscopy study. *Journal of Psychiatric Research*, 47(3), 412–417.
- Luykx, J. J., Laban, K. G., van den Heuvel, M. P., Boks, M. P., Mandl, R. C., Kahn, R. S., & Bakker, S. C. (2012). Region and state specific glutamate downregulation in major depressive disorder: A meta-analysis of (1)H-MRS findings. *Neuroscience and Biobehavioral Reviews*, 36(1), 198–205.
- Maddock, R. J., & Buonocore, M. H. (2012). MR spectroscopic studies of the brain in psychiatric disorders. *Current Topics in Behavioral Neurosciences*, 11, 199–251.
- Mandal, P. K., Tripathi, M., & Sugunan, S. (2012). Brain oxidative stress: Detection and mapping of anti-oxidant marker 'Glutathione' in different brain regions of healthy male/female, MCI and Alzheimer patients using non-invasive magnetic resonance spectroscopy. *Biochemical and Biophysical Research Communications*, 417(1), 43–48.
- Marsman, A., van den Heuvel, M. P., Klomp, D. W., Kahn, R. S., Luijten, P. R., & Hulshoff Pol, H. E. (2013). Glutamate in schizophrenia: A focused review and meta-analysis of (1)H-MRS studies. *Schizophrenia Bulletin*, 39(1), 120–129.
- Matsuzawa, D., & Hashimoto, K. (2011). Magnetic resonance spectroscopy study of the antioxidant defense system in schizophrenia. *Antioxidants & Redox Signaling*, 15(7), 2057–2065.
- Mescher, M., Merkle, H., Kirsch, J., Garwood, M., & Gruetter, R. (1998). Simultaneous in vivo spectral editing and water suppression. *NMR in Biomedicine*, 11(6), 266–272.
- Pollack, M. H., Jensen, J. E., Simon, N. M., Kaufman, R. E., & Renshaw, P. F. (2008). High-field MRS study of GABA, glutamate and glutamine in social anxiety disorder: Response to treatment with levetiracetam. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 32(3), 739–743.

- Ross, B., Bluml, S., Danielson, E., & Kanamori, K. (1997). Molecular Probing and imaging by magnetic resonance spectroscopy: Possibilities for imaging gene therapy. In W. Semmler & M. Schwaiger (Eds.), *Impact of molecular biology and new technical developments in diagnostic imaging* (pp. 43–65). Berlin, Germany: Springer.
- Sailasuta, N., Harris, K., Tran, T., & Ross, B. (2011). Minimally invasive biomarker confirms glial activation present in Alzheimer's disease: A preliminary study. *Neuropsychiatric Disease and Treatment*, 7, 495–499.
- Sailasuta, N., Ross W, Ananworanich J, Chalermchai T, DeGruttola V, Lerdlum S, Pothisri M, Busovaca E, Ratto-Kim S, Jagodzinski L, Spudich S, Michael N, Kim JH, Valcour V, teams RSp. Change in brain magnetic resonance spectroscopy after treatment during acute HIV infection. *PLoS One*, 2012. 7(11): p. e49272.
- Sailasuta, N., Shriner, K., & Ross, B. (2009). Evidence of reduced glutamate in the frontal lobe of HIV-seropositive patients. *NMR in Biomedicine*, 22(3), 326–331.
- Sanacora, G., Treccani, G., & Popoli, M. (2012). Towards a glutamate hypothesis of depression: An emerging frontier of neuropsychopharmacology for mood disorders. *Neuropharmacology*, 62(1), 63–77.
- Satoh, T., & Yoshioka, Y. (2006). Contribution of reduced and oxidized glutathione to signals detected by magnetic resonance spectroscopy as indicators of local brain redox state. *Neuroscience Research*, 55(1), 34–39.
- Soher, B. J., Hurd, R. E., Sailasuta, N., & Barker, P. B. (1996). Quantitation of automated single-voxel proton MRS using cerebral water as an internal reference. *Magnetic Resonance in Medicine*, 36(3), 335–339.
- Thomas, M.A., Binesh N., Yue K., DeBruhl N. Volume-localized two-dimensional correlated magnetic resonance spectroscopy of human breast cancer. *Journal of Magnetic Resonance Imaging*, 2001. 14(2): p. 181–6.
- Tran, T. K., Vigneron, D. B., Sailasuta, N., Tropp, J., Le Roux, P., Kurhanewicz, J., ... Hurd, R. (2000). Very selective suppression pulses for clinical MRSI studies of brain and prostate cancer. *Magnetic Resonance in Medicine*, 43(1), 23–33.
- Urenjak, J., Williams, S. R., Gadian, D. G., & Noble, M. (1992). Specific expression of N-acetylaspartate in neurons, oligodendrocyte-type-2 astrocyte progenitors, and immature oligodendrocytes in vitro. *Journal of Neurochemistry*, 59(1), 55–61.

Suggested Reading

- Baker, E. H., Basso, G., Barker, P. B., Smith, M. A., Bonekamp, D., & Horská, A. (2008). Regional apparent metabolite concentrations in young adult brain measured by (1)H MR spectroscopy at 3 Tesla. *Journal of Magnetic Resonance Imaging*, 27(3), 489–499.
- Mikkelsen, M., Barker, P. B., Bhattacharyya, P. K., Brix, M. K., Buur, P. F., Cecil, K. M., ... RAE, E. (2017). Big GABA: Edited MR spectroscopy at 24 research sites. *NeuroImage*, 159, 32–45.
- Oz, G., Alger, J. R., Barker, P. B., Bartha, R., Bizzi, A., Boesch, C., ... MRS Consensus Group. (2014). Clinical proton MR spectroscopy in central nervous system disorders. *Radiology*, 270(3), 658–679.
- Xu, V., Chan, H., Lin, A. P., Sailasuta, N., Valencerina, S., Tran, T., ... Ross, B. D. (2008). MR spectroscopy in diagnosis and neurological decision-making. *Seminars in Neurology*, 28(4), 407–422.
- Zhu, H., & Barker, P. B. (2011). MR spectroscopy and spectroscopic imaging of the brain. *Methods in Molecular Biology*, 711, 203–226.

Artificial Intelligence in Medicine: Can Computers Aid the Diagnosis of Alzheimer's Disease?

Christian Salvatore^{1,2,*}, Petronilla Battista^{3,*}, and
Isabella Castiglioni⁴

¹*Scuola Universitaria Superiore IUSS Pavia, Piazza della Vittoria, Pavia, Italy*

²*DeepTrace Technologies S.R.L., Via Conservatorio, Milan, Italy*

³*Istituti Clinici Scientifici Maugeri IRCCS, Pavia, Institute of Bari, Italy*

⁴*Dipartimento di Fisica "G. Occhialini", Università degli Studi di Milano-Bicocca – Piazza
della Scienza, Milan, Italy*

Manuscript

The impressive amount of research in the field of neurodegenerative diseases that has characterized the last two decades has been directed in particular toward the establishment of accurate measures able to make an early diagnosis of the diseases. In particular, the greatest effort and the most promising findings have been collected for the early detection of Alzheimer's disease (AD).

AD is the most common form of dementia, in which memory problems are typically the first symptoms. The underlying pathophysiological mechanisms start several years before the clinical manifestation of the disease. Therefore, revised diagnostic criteria emphasized the role of the *in vivo* detection of biomarkers for the identification of different stages of AD (Albert et al., 2011; McKhann et al., 2011). There is a great promise that biomarker assessment will lead to an accurate diagnosis of AD. Two main groups of validated biomarkers have been categorized: (a) amyloid-beta deposition/plaque formation and (b) tau-protein neurodegeneration. Neuroimaging and cerebrospinal fluid measures have been proposed as useful tools for the detection of the underpinning pathophysiological process of AD. Among neuroimaging

*Equally contributed

techniques, magnetic resonance imaging (MRI) allows detecting the evolution of distinct patterns from early to late AD. Many recent studies showed that the atrophy of the hippocampal area as well as of the entorhinal cortex are considered to be indicators of the regional neuronal injury (Apostolova et al., 2010; Martin, Smith, Collins, Schmitt, & Gold, 2010).

Furthermore, behavioral data have been shown to provide an important contribution for the early and differential diagnosis of AD. Some cognitive scores collected during the assessment of patients could represent a useful marker of the clinical onset of AD. In particular, it has been pointed out that low performance at free and cued selective reminding test (FCSRT), even in presence of retrieval facilitation with cueing, can be a valid clinical marker of typical AD (Dubois et al., 2014). Low free recall at FCSRT could also represent a predictor of conversion to AD in MCI patients (Dubois et al., 2016). Other studies (e.g., Della Sala, Parra, Fabi, Luzzi, & Abrahams, 2012) showed memory binding as a successful predictor of conversion from mild cognitive impairment (MCI) to AD. Although many neuropsychological measures may appear promising, the definition of the best cognitive test is still controversial.

Recently, it has been proposed that multimodal markers can improve the sensitivity and specificity of diagnosis. In this scenario, it clearly emerges the importance to identify the best combination of biomarkers able to accurately detect the early stage of the disease or to predict the prognostic outcome in symptomatic patients at elevated risk.

Advances in the field of artificial intelligence have led to new approaches useful for the analysis of medical data. Among these approaches, multivariate analysis and machine learning (ML) have shown promising results.

Multivariate analysis is the analysis of data based on the principle of multivariate statistics. In contrast with univariate analysis, it involves the study of more than one statistical variable at a time. In this sense, multivariate analysis is able to extract information from distributed patterns of data, without requiring a priori knowledge of where it may be coded (i.e., of which variables of interest should be considered). A great consequence of this point is that this approach allows highlighting differences that might otherwise remain undetected, because—in general—no a priori choice of variables is necessary in this case.

ML is a subfield of computer science that includes all advanced computer methods able to learn from *experience*. Therefore, ML also comprehends multivariate techniques that are able to learn a specific task gathering information from multiple variables. There are two great advantages of ML techniques: the first is that they are able to deal with large amounts of data in a relatively short time, which is particularly useful in problems that require the use of a large number of variables or samples (e.g., in medicine), and the second is that ML methods are able to generate predictive models (learned from experience) that can be used to make predictions on new (unseen) samples. Specifically, this last characteristic represents the fundament of ML methods, which are—by their own nature—able to perform automatic single-subject classification, in contrast with traditional statistical techniques. The ability of learning from a given (finite) dataset on how to classify samples not belonging to that dataset is called *generalization ability*. Finally, another interesting characteristic of ML algorithms is that they can be used to find which features most influence the generation of the predictive model. For example, in a simple binary classification task, ML can be used to determine which features are the most important for the discrimination of two classes.

Because of these characteristics, multivariate ML techniques are becoming one of the main research topics in data science, given their applicability to different fields and, in particular, to medicine, where there is strong need of tools able to analyze big data in an objective way, to make accurate predictions about patients' status, and to automatically extract potential markers of diseases.

As a matter of fact, although we are still in a *research* phase, ML techniques have been widely and successfully applied in medicine to the diagnosis of AD. A paper by Klöppel et al. (2008) showed that computers—intended as *intelligent* algorithms—are suitable for the diagnosis of AD. Indeed, in this study, a particular ML algorithm, called support vector machine (SVM), classified AD using neuroimaging data with performances comparable with or higher than well-trained neuroradiologists, thus suggesting a primary role for computerized diagnostic methods in clinical practice.

According to the field of applicability, there are several studies showing promising results in the classification of AD patients. Concerning the neuroimaging field, MRI coupled to ML has been reported to be successful in performing the automatic classification of AD and MCI patients (e.g., Nanni, Salvatore, Cerasa, & Castiglioni, 2016; Salvatore, Cerasa, et al., 2015). A previous systematic review investigated the use of ML for the classification of AD through structural MRI (Salvatore, Battista, & Castiglioni, 2015), reporting median specificity and sensitivity for the conversion from MCI to AD of 66 and 75%, respectively. There are only few papers investigating the application of ML to positron emission tomography (PET). Pagani et al. (2015) found a specificity and sensitivity of 92 and 91% for the prediction of conversion to AD, respectively. Overall, these studies demonstrated that ML algorithms applied to biomedical imaging might aid in the diagnosis of AD, but these conclusions should be confirmed in samples coming from dementia clinics.

Advanced ML methods have been successfully applied also to biological studies of AD. Craig-Schapiro et al. (2011) revealed that CSF calbindin, in combination with CSF A β 42 and age, predicts the risk of developing AD with 80% of sensitivity and specificity. Other results (Yang, Tan, & Qiu, 2012) coming from ML applied to biological data and neuroimaging indicate that abnormal values of CSF A β 42 coupled with atrophy as measured using MR imaging precede clinical symptoms of AD with a sensitivity and specificity of 88 and 82%, respectively.

ML methods have also found their fruitful application in the automatic analysis of behavioral/neuropsychological data collected from AD patients. In particular, Weakley, Williams, Schmitter-Edgecombe, & Cook (2015) showed that neuropsychological measures alone are able to achieve good performance for the diagnosis of AD, above 80%. ML algorithms have been applied to the identification of the best neuropsychological measures for the diagnosis of AD (e.g., Battista, Salvatore, & Castiglioni, 2017; Cui et al., 2011, 2012). Specifically, decline of episodic memory and executive functions appear to be the most accurate measures. Fraser and colleagues (Fraser, Meltzer, & Rudzicz, 2015) found that ML algorithms coupled with measures of picture description tasks are able to distinguish AD from the healthy condition. Results showed that the mean length of sentences, the number of produced words and verbs, the frequency, and the familiarity of words are consistent measures of AD. ML coupled with linguistic markers is object of study also for the automatic diagnosis of pathologies involving language impairment as a first symptom, such as primary progressive aphasia (PPA) (Garrard, Rentoumi, Gesierich, Miller, & Gorno-Tempini, 2014).

In the last few years, a growing interest in the field of biomarker assessment through artificial intelligence techniques has been directed toward the combination of multiple sources of clinical information coming from structural, functional, metabolic, molecular neuroimaging, neurophysiology, neuro-biochemistry of CSF, and neurogenic sources (Cavedo et al., 2014). The use of multimodality markers is getting more consensus in the literature, showing that combining features from different modalities leads to higher diagnostic accuracy compared to the results of each modality alone (e.g., Dukart, Sambataro, & Bertolino, 2015), since this approach provides complementary information for the diagnostic process.

Although several biomarkers have been identified from the recent literature, these markers have to be considered with high attention. The definition of the ML algorithm and of the

validation procedures associated with the accuracy of different measures can vary across studies, as well as the definition of the gold standards for the diagnosis and of the performance-evaluation metrics. Therefore, in order to improve the diagnostic power of ML techniques, it would be desirable to decrease the heterogeneity among studies, or—in other words—to make them comparable. This could provide a powerful and noninvasive tool not only for the diagnosis of AD but also for screening the efficacy of new drug treatments without the need for large and expensive clinical trials.

Finally, a new subfield of artificial intelligence is recently emerging from the computer-vision community (Krizhevsky, Sutskever, & Hinton, 2012). This subfield, known as deep learning, represents the most advanced frontier of ML, especially in terms of performance. Deep learning techniques are particularly useful for the automatic classification of images, for which they were originally designed, and they have been proven to outperform previous ML algorithms in most tasks (Razavian, Azizpour, Sullivan, & Carlsson, 2014). However, in order for deep learning algorithms to be trained preventing overfitting problems, a huge amount of data are needed. Namely, this translates in the need for datasets of thousands or millions of images. This is the reason why this technique has not been applied yet to the diagnosis of AD through biomedical imaging. The perspective of higher performance than those obtained by current ML methods is thus a strong motivation for the clinical community to collect big sets of medical data and to make them available in public repositories.

Author Biographies

Christian Salvatore is a senior researcher at the Scuola Universitaria Superiore IUSS, Pavia, Italy. He has a scientific background in the field of physics applied to medicine and diagnostic imaging. His research concerns the development of artificial intelligence methods for the quantification, extraction and classification of information acquired through imaging techniques. He is the co-founder and CEO of DeepTrace Technologies, a spin-off of Scuola Universitaria Superiore IUSS, which operates in the artificial intelligence field. His research work has been recognized with important awards such as the FORBES 30 UNDER 30 Award.

Petronilla Battista is a neuropsychologist in the Department of Neurological Rehabilitation at the Maugeri Institute of Bari, Italy. She completed her PhD at the Scuola Universitaria Superiore IUSS, Pavia, Italy, and obtained an Atlantic fellow for Equity in Brain Health at Global Brain Health Institute, University of California San Francisco (UCSF). Her research interests are focused on the application of artificial intelligence to neuropsychological data for the diagnosis of neurodegenerative diseases.

Isabella Castiglioni, physicist, is a Full Professor at the University of Milano Bicocca. Her current activities aim at the implementation and development of advanced methods for the elaboration, analysis, and classification of medical data, including biological and neuroimaging data. Her activities deal with the exchange and integration of information between basic and applied research and clinical diagnostic and therapeutic technologies.

References

- Albert, M. S., DeKosky, S. T., Dickson, D., Dubois, B., Feldman, H. H., Fox, N. C., ... Phelps, C. H. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging and Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 7(3), 270–279. doi:10.1016/j.jalz.2011.03.008

- Apostolova, L. G., Mosconi, L., Thompson, P. M., Green, A. E., Hwang, K. S., Ramirez, A., ... de Leon, M. J. (2010). Subregional hippocampal atrophy predicts Alzheimer's dementia in the cognitively normal. *Neurobiology of Aging*, 31(7), 1077–1088. doi:10.1016/j.neurobiolaging.2008.08.008
- Battista, P., Salvatore, C., & Castiglioni, I. (2017). Optimizing neuropsychological assessments for cognitive, behavioral, and functional impairment classification: A machine learning study. *Behavioural Neurology*, 2017, 1850909. doi:10.1155/2017/1850909
- Cavedo, E., Lista, S., Khachaturian, Z., Aisen, P., Amouyel, P., Herholz, K., ... Hampel, H. (2014). The road ahead to cure Alzheimer's disease: Development of biological markers and neuroimaging methods for prevention trials across all stages and target populations. *The Journal of Prevention of Alzheimer's Disease*, 1(3), 181–202. doi:10.14283/jpad.2014.32
- Craig-Schapiro, R., Kuhn, M., Xiong, C., Pickering, E. H., Liu, J., Misko, T. P., ... Holtzman, D. M. (2011). Multiplexed immunoassay panel identifies novel CSF biomarkers for Alzheimer's disease diagnosis and prognosis. *PLoS One*, 6(4). doi:10.1371/journal.pone.0018850
- Cui, Y., Liu, B., Luo, S., Zhen, X., Fan, M., Liu, T., ... Jin, J. S. (2011). Identification of conversion from mild cognitive impairment to Alzheimer's disease using multivariate predictors. *PLoS One*, 6(7). doi:10.1371/journal.pone.0021896
- Cui, Y., Wen, W., Lipnicki, D. M., Beg, M. F., Jin, J. S., Luo, S., ... Sachdev, P. S. (2012). Automated detection of amnesic mild cognitive impairment in community-dwelling elderly adults: A combined spatial atrophy and white matter alteration approach. *NeuroImage*, 59(2), 1209–1217. doi:10.1016/j.neuroimage.2011.08.013
- Della Sala, S., Parra, M. A., Fabi, K., Luzzi, S., & Abrahams, S. (2012). Short-term memory binding is impaired in AD but not in non-AD dementias. *Neuropsychologia*, 50(5), 833–840. doi:10.1016/j.neuropsychologia.2012.01.018
- Dubois, B., Feldman, H. H., Jacova, C., Hampel, H., Molinuevo, J. L., Blennow, K., ... Cummings, J. L. (2014). Advancing research diagnostic criteria for Alzheimer's disease: The IWG-2 criteria. *The Lancet Neurology*. doi:10.1016/S1474-4422(14)70090-0
- Dubois, B., Hampel, H., Feldman, H. H., Scheltens, P., Aisen, P., Andrieu, S., ... Jack, C. R. (2016). Preclinical Alzheimer's disease: Definition, natural history, and diagnostic criteria. *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*. doi:10.1016/j.jalz.2016.02.002
- Dukart, J., Sambataro, F., & Bertolino, A. (2015). Accurate prediction of conversion to Alzheimer's disease using imaging, genetic, and neuropsychological biomarkers. *Journal of Alzheimer's Disease: JAD*, 49(4), 1143–1159. doi:10.3233/JAD-150570
- Fraser, K. C., Meltzer, J. A., & Rudzicz, F. (2015). Linguistic features identify Alzheimer's disease in narrative speech. *Journal of Alzheimer's Disease: JAD*, 49(2), 407–422. doi:10.3233/JAD-150520
- Garrard, P., Rentoumi, V., Gesierich, B., Miller, B., & Gorno-Tempini, M. L. (2014). Machine learning approaches to diagnosis and laterality effects in semantic dementia discourse. *Cortex*, 55(1), 122–129. doi:10.1016/j.cortex.2013.05.008
- Klöppel, S., Stonnington, C. M., Barnes, J., Chen, F., Chu, C., Good, C. D., ... Frackowiak, R. S. J. (2008). Accuracy of dementia diagnosis—A direct comparison between radiologists and a computerized method. *Brain*, 131(11), 2969–2974. doi:10.1093/brain/awn239
- Krizhevsky, A., Sutskever, I., & Hinton, G. E. (2012). ImageNet classification with deep convolutional neural networks. *Advances in Neural Information Processing Systems*, 1–9. doi:10.1016/j.protcy.2014.09.007
- Martin, S. B., Smith, C. D., Collins, H. R., Schmitt, F. A., & Gold, B. T. (2010). Evidence that volume of anterior medial temporal lobe is reduced in seniors destined for mild cognitive impairment. *Neurobiology of Aging*, 31(7), 1099–1106. doi:10.1016/j.neurobiolaging.2008.08.010
- McKhann, G. M., Knopman, D. S., Chertkow, H., Hyman, B. T., Jack, C. R., Kawas, C. H., ... Phelps, C. H. (2011). The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*. doi:10.1016/j.jalz.2011.03.005

- Nanni, L., Salvatore, C., Cerasa, A., & Castiglioni, I. (2016). Combining multiple approaches for the early diagnosis of Alzheimer's disease. *Pattern Recognition Letters*, 84, 259–266. doi:10.1016/j.patrec.2016.10.010
- Pagani, M., De Carli, F., Morbelli, S., Öberg, J., Chincarini, A., Frisoni, G. B., ... Nobili, F. (2015). Volume of interest-based [18F]fluorodeoxyglucose PET discriminates MCI converting to Alzheimer's disease from healthy controls. A European Alzheimer's Disease Consortium (EADC) study. *NeuroImage. Clinical*, 7, 34–42. doi:10.1016/j.nicl.2014.11.007
- Razavian, A. S., Azizpour, H., Sullivan, J., & Carlsson, S. (2014). CNN features off-the-shelf: An astounding baseline for recognition. *IEEE Computer Society Conference on Computer Vision and Pattern Recognition Workshops* (pp. 512–519). doi:10.1109/CVPRW.2014.131
- Salvatore, C., Battista, P., & Castiglioni, I. (2015). Frontiers for the early diagnosis of AD by means of MRI brain imaging and Support Vector Machines. *Current Alzheimer Research*, 13(5), 509–533.
- Salvatore, C., Cerasa, A., Battista, P., Gilardi, M. C., Quattrone, A., & Castiglioni, I. (2015). Magnetic resonance imaging biomarkers for the early diagnosis of Alzheimer's disease: A machine learning approach. *Frontiers in Neuroscience*, 9(SEP), 307. doi:10.3389/fnins.2015.00307
- Weakley, A., Williams, J. A., Schmitter-Edgecombe, M., & Cook, D. J. (2015). Neuropsychological test selection for cognitive impairment classification: A machine learning approach. *Journal of Clinical and Experimental Neuropsychology*, 37(9), 899–916. doi:10.1080/13803395.2015.1067290
- Yang, X., Tan, M. Z., & Qiu, A. (2012). CSF and brain structural imaging markers of the Alzheimer's pathological cascade. *PLoS One*, 7(12). doi:10.1371/journal.pone.0047406

Therapeutic Effects of Repetitive Transcranial Magnetic Stimulation (rTMS) in Stroke: Moving Toward an Individualized Approach

Jason L. Neva¹, Kathryn S. Hayward^{1,2,3}, and
Lara A. Boyd^{1,4}

¹*Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada*

²*Stroke Division, Florey Institute of Neuroscience and Mental Health, University of Melbourne, Melbourne, VIC, Australia*

³*NHMRC Centre for Research Excellence in Stroke Rehabilitation and Recovery, Parkville, VIC, Australia*

⁴*Djavad Mowafaghian Centre for Brain Health, University of British Columbia, Vancouver, BC, Canada*

Overview

Transcranial magnetic stimulation (TMS) is a noninvasive technique to assess and alter excitability within the central nervous system. Through electromagnetism TMS generates activity within the cortex, which can indirectly depolarize corticospinal output neurons that ultimately activate peripheral muscle(s). This chapter focuses on the applications of TMS to index and modulate the neurophysiology of the motor system after a brain injury, specifically stroke. We will provide an overview of TMS, applications in healthy and neurologically injured populations to index cortical excitability and connectivity, along with the potential application to modulate excitability and connectivity to achieve a functional benefit (e.g., repetitive TMS [rTMS]). Throughout this chapter we will highlight limitations of a “one-size-fits-all” application of TMS to index and modulate the motor system. Together, this will support our concluding framework to develop a targeted assessment of cortical excitability and connectivity to foster application of an individualized rTMS treatment approach.

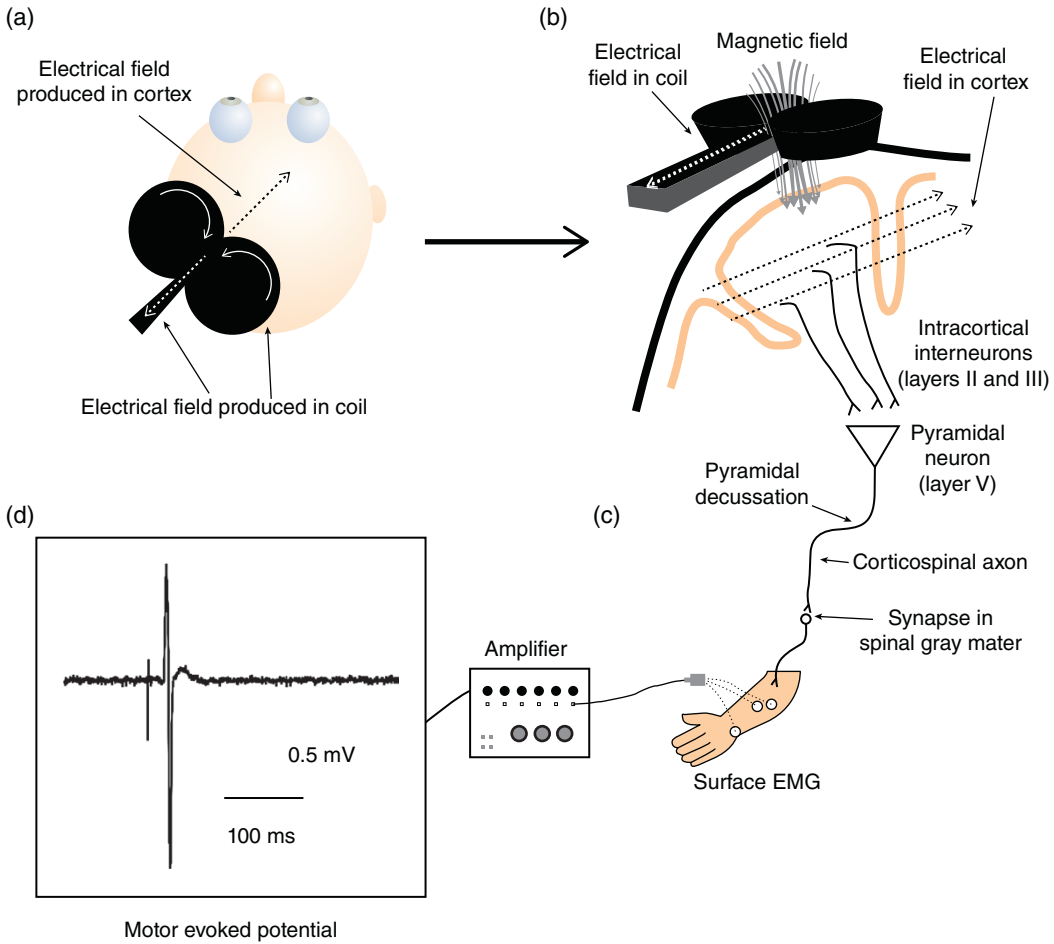


Figure 1 Schematic of transcranial magnetic stimulation (TMS). (a) A TMS coil placed over the scalp produces an electric field in the TMS coil, as well as in the opposite direction in the cortex. (b) Electric field produced by the coil induces a magnetic field traversing the skull and dura, producing electrical activity in the cortex in the opposite direction, which activates intracortical interneurons synapsing on pyramidal neurons and thus activates the corticospinal tract. The descending corticospinal tract then synapses on to a motor neuron in the spinal gray matter, which then carries the signal to the muscle. (c) The surface electromyographical (EMG) response resulting from TMS-induced activation of the corticospinal tract is captured via surface electrodes recording from the target muscle. (d) Amplified EMG is projected onto a computer screen for quantification of motor evoked potential (MEP) amplitude and latency.

Transcranial Magnetic Stimulation (TMS)

Transcranial magnetic stimulation (TMS) is a noninvasive technique to assess and alter excitability within the central nervous system (Auriat, Neva, Peters, Ferris, & Boyd, 2015; Brown, Neva, Ledwell, & Boyd, 2014; Chen et al., 2008). TMS operates under the principles of electromagnetism to activate cortical neurons. When activated electric current courses along the wires within the TMS coil, a rapidly changing transient magnetic field traverses the scalp and generates electrical activity within the cortex under the coil. If held over the primary

motor cortex (M1) and with high enough intensities, a single pulse of TMS will result in transsynaptic activation of corticospinal output neurons. The corticospinal volleys activated by TMS reach the target muscle and can be recorded by surface electromyography (EMG), which is observed as a well-defined waveform called the motor-evoked potential (MEP) (see Figure 1). The amplitude (and sometimes latency) of the MEP is the primary outcome measure of corticospinal excitability using TMS (Chen et al., 2008; Di Lazzaro & Ziemann, 2013).

TMS can be used to index and modulate cortical excitability. Methods of single and paired pulse TMS are commonly used to index corticospinal and interhemispheric excitability in healthy and neurological populations (see Table 1 and comprehensive reviews by Brown et al., 2014 and Auriat et al., 2015). In contrast, repetitive delivery of TMS pulses (rTMS) can be used to modulate cortical excitability for a period of time (from minutes to ~1 hr) (Chen et al., 1997; Huang, Edwards, Rounis, Bhatia, & Rothwell, 2005). There has been a rapid expansion in the application of rTMS as an intervention to modulate cortical excitability. This is likely underpinned by the notion that rTMS is thought to mimic the early stages of long-term potentiation (LTP) and long-term depression (LTD)-like mechanisms, which are considered to be the basic neural processes underlying experience-dependent neuroplasticity in the brain (Di Lazzaro & Ziemann, 2013; Rioult-Pedotti, Friedman, & Donoghue, 2000). There has been growing interest to use rTMS as a potential therapeutic tool for healthy and neurological populations since it has been shown to cause lasting alterations in cortical excitability and behavior beyond that of the stimulation itself (Auriat et al., 2015; Brown et al., 2014; Chen et al., 2008). Today, researchers continue to use rTMS to test whether the application of stimulation alone, or in conjunction with motor training, can enhance rehabilitation benefits in people with neurological impairment (Auriat et al., 2015; Brown et al., 2014; Hsu, Cheng, Liao, Lee, & Lin, 2012). While rTMS may be applied in many neurological impairments (Brown et al., 2014), for the purposes of this chapter, we will focus on stroke given the high prevalence of people living with the effects of stroke coupled with the large body of research that has been completed to investigate the therapeutic potential of rTMS in stroke.

TMS to Assess Cortical Excitability and Connectivity After Stroke

Studies using multiple methods of TMS demonstrate altered brain excitability and connectivity during all phases post-stroke. Firstly, there can be changes in corticospinal and intracortical excitability. After stroke the ipsilesional hemisphere typically shows less corticospinal excitability with smaller, or even absent, MEPs. In contrast, the contralesional hemisphere often demonstrates equal or abnormally increased corticospinal excitability (Liepert et al., 1998; Sawaki et al., 2008; Stinear, Barber, Petoe, Anwar, & Byblow, 2012). Importantly, individuals that demonstrate an MEP elicited from the ipsilesional hemisphere soon after stroke generally show better motor recovery (Stinear et al., 2012) compared with those who do not (Trompetto, Assini, Buccolieri, Marchese, & Abbruzzese, 2000). Similarly, intracortical excitability can be altered in the ipsilesional hemisphere but remains relatively normal in the contralesional hemisphere (Manganotti et al., 2002). Together, these alterations in corticospinal and intracortical excitability suggest an imbalance between the ipsilesional and contralesional cortices that may underpin decreased capacity for motor function of the paretic limb (Liepert et al., 1998; Sawaki et al., 2008; Stinear et al., 2012).

Secondly, there can be alterations in connectivity between the two hemispheres, often referred to as interhemispheric inhibition (IHI). After stroke, asymmetry in the level of IHI between the two hemispheres is associated with the degree of functional recovery expressed.

Table 1 Summary of single, paired pulse and repetitive transcranial magnetic stimulation (TMS) measures of cortical excitability.

Single pulse					
CNS measure	<i>MEP, A/RMT</i> Corticospinal excitability	<i>RC</i> Corticospinal excitability over a range of stimulation intensities	<i>Mapping</i> Spatial distribution and amplitudes of corticospinal excitability	<i>CSP</i> Cortical and spinal inhibitory excitability	<i>iSP</i> Transcallosal excitability
Paired pulse					
CNS measure	<i>SICI, ICF, LICI</i> Intracortical excitability	<i>SICF</i> Intracortical excitability	<i>SAI, AF, LAI</i> Sensorimotor excitability	<i>CMCT</i> Cortical output latency	<i>IHI</i> Interhemispheric excitability
Repetitive TMS (rTMS)					
CNS effect	<i><1-Hz</i> Cortical excitability decrease (~15 min)	<i>>1-Hz</i> Cortical excitability increase (~15 min)	<i>cTBS</i> Cortical excitability decrease (~60 min)	<i>iTBS</i> Cortical excitability increase (~60 min)	<i>PAS</i> PAS ₂₅ : corticospinal facilitation (~30 min) PAS ₁₀ : corticospinal suppression (~30 min)

MEP, motor evoked potential; AMT, active motor threshold; RMT, resting motor threshold; RC, recruitment curve; CSP, contralateral silent period; iSP, ipsilateral silent period; CNS, central nervous system; SICI, short-interval intracortical inhibition; ICF, intracortical facilitation; LICI, long-interval intracortical inhibition; SICF, short-interval intracortical facilitation; SAI, short afferent inhibition; AF, afferent facilitation; LAI, long afferent inhibition; CMCT, central motor conduction time; IHI, interhemispheric inhibition; cTBS, continuous theta burst stimulation; iTBS, intermittent theta burst stimulation; PAS, paired associative stimulation.

Several TMS studies show that the ipsilesional M1 produces less IHI (Boroojerdi, Diefenbach, & Ferbert, 1996; Shimizu et al., 2002) and the contralesional M1 continues to show normal, or even increased levels of IHI (Duque et al., 2005; Murase, Duque, Mazzocchio, & Cohen, 2004). The net result is increased inhibition acting on the ipsilesional hemisphere that may decrease ipsilesional M1 excitability (Liepert et al., 1998) and interfere with corticospinal function and neuroplasticity in the ipsilesional cortex (Carey, Fregni, & Pascual-Leone, 2006; Taub et al., 2006). This pattern of asymmetric IHI has been associated with poorer motor function (Duque et al., 2005; Harris-Love, Chan, Dromerick, & Cohen, 2015; Murase et al., 2004). Collectively, these findings have given rise to the idea of the interhemispheric competition model (IHCM) (Auriat et al., 2015; Di Pino et al., 2014). This model assumes that the neurologically intact brain has mutually balanced inhibition between hemispheres, but the damage due to stroke in one hemisphere disrupts this balance. The IHCM posits that poor recovery of function after stroke is hindered by decreased excitability in the ipsilesional cortex and exacerbated by excessive inhibition from the contralesional hemisphere. The idea of the IHCM has generated much discussion surrounding the notion that the contralesional hemisphere is not a “friend” to recovery after stroke but rather a “foe,” hindering its potential to drive repair and recovery after injury.

Recent research that has stratified patients by severity of motor impairment would suggest that the IHCM might be too simplistic. Work that has investigated people with chronic stroke (>6 months post injury) has found *increased* IHI from the ipsilesional to the contralesional M1 (Borich, Neva, & Boyd, 2015). This is contrary to previous findings underpinning the IHCM (Auriat et al., 2015; Di Pino et al., 2014). Further work shows that *increased* inhibition from the contralesional hemisphere is associated with *less* impairment and *better* function (Mang, Borich, Brodie, & Boyd, 2015). Moreover, those with severe upper limb impairment after stroke show *less* ipsilesional IHI associated with *greater* function (Hayward et al., 2017). Collectively, these findings have generated renewed interest to understand the neurobiological role of the contralesional hemisphere to achieve motor recovery. The contralesional hemisphere may not be the “foe” to all that the IHCM suggests, but could be a “friend,” which aids in supporting recovery of function, potentially substituting for cortical networks lost after stroke.

rTMS as a Therapeutic Approach to Improve Recovery of Function After Stroke

rTMS is a potential therapeutic approach to aid in the recovery of motor function after stroke (Corti, Patten, & Triggs, 2012). Theoretically, rTMS can be used to either *increase* ipsilesional cortical excitability by directly applying excitatory rTMS over the ipsilesional hemisphere or *decrease* contralesional cortical excitability by applying inhibitory rTMS over the contralesional hemisphere to decrease the perceived abnormally enhanced inhibitory signals directed toward the lesioned M1 and thereby increase ipsilesional excitability (Auriat et al., 2015). Although both approaches of rTMS application after stroke have shown promise in enhancing ipsilesional excitability and improving motor function, with perhaps slightly more promise from inhibitory contralesional rTMS (Hsu et al., 2012), there is accumulating evidence that the response to rTMS is inconsistent and variable (Carey et al., 2014; Héroux, Taylor, & Gandevia, 2015; Meehan, Dao, Linsdell, & Boyd, 2011). Inconsistent and variable effects are noted regardless of what form of rTMS is employed (Carey et al., 2014; Meehan et al., 2011) and irrespective of the targeted brain area or hemisphere (Carey et al., 2014; Hsu et al., 2012;

Meehan et al., 2011) (Table 1). Further, varied responses to rTMS are not explained by simple demographic factors such as age, sex, time post stroke, or stroke severity (Carey et al., 2014; Hsu et al., 2012). This supports a rationale for developing a framework to individualize the application of rTMS to achieve optimal modulation of cortical networks.

The Need for an Individualized Approach to rTMS Application After Stroke

Despite its broad use in healthy and stroke populations, a comprehensive understanding of the neurophysiological effects of rTMS on brain and behavior is lacking. Although some work has provided evidence for identifying how healthy individuals may respond to specific types of rTMS (Hamada, Murase, Hasan, Balaratnam, & Rothwell, 2013), at present there is no single measure that can accurately predict an individual's response to rTMS, particularly in neurological populations including stroke. This likely reflects the large complexity of brain connections and function and highlights the need for new approaches that attempt to categorize an individual response to rTMS interventions based on measures of their neurophysiology and anatomy. There are multiple indices of cortical excitability and connectivity via TMS (mentioned above; see Table 1) that may assist in navigating the pathway to selection and application of rTMS for an individual to respond optimally.

To date stroke recovery research has focused almost entirely on M1 in the *ipsilesional* hemisphere as the target location of cortical repair and augmentation with brain stimulation applying excitatory rTMS (Auriat et al., 2015; Plow, Cunningham, Varnerin, & Machado, 2014). The focus on M1 is likely underpinned by previous research suggesting that patients with damaged M1 generally have worse recovery and those with spared M1 have better recovery (Bhatt et al., 2007). Yet, there is growing evidence that association motor-related areas can take over the function of M1, such as the premotor cortices (Frost et al., 2003; Plow et al., 2014). These areas, anterior to M1, which include the supplementary motor area (SMA) and the dorsal (PMd) and ventral (PMv) premotor cortices, greatly influence and contribute to M1 corticospinal output. Recent work has suggested that premotor areas may be viable options for brain stimulation (e.g., rTMS) to potentiate recovery, particularly in those with more severe impairment (Plow et al., 2014). We not only agree with this hypothesis and suggestion, but we also suggest that these association motor-related areas may be tested for corticospinal excitability via single and paired pulse TMS in addition to M1 to assist in guiding the therapeutic application of rTMS. If indeed areas like PMv can compensate for the function of M1 as shown by previous work (Frost et al., 2003), then it is possible that these areas could be indexed by TMS.

Except for a very small number of studies (Brodie, Meehan, Borich, & Boyd, 2014; Meehan et al., 2011), the vast majority of research employing *contralesional* rTMS has focused solely on M1. Since there is evidence that interhemispheric connectivity is not as simple as the IHCM may suggest after stroke, this possibly suggests that other non-primary motor areas (i.e., premotor and prefrontal areas) may contribute to maintaining or compensating for altered interhemispheric connectivity following stroke. This notion is further supported by the fact that interhemispheric connectivity has been demonstrated between non-M1 areas such as primary somatosensory cortex (S1), PMd, and dorsolateral prefrontal cortex (DLPFC) to the contralateral M1 in healthy individuals (Ni et al., 2009). If this interhemispheric connectivity exists before the onset of a stroke, it is possible that some of these connections remain after

damage due to stroke, which may be useful to assess spared connectivity and be exploited for the repair and recovery of function.

Personalized Therapeutic rTMS Application After Stroke

The development of a TMS assessment protocol that indexes cortical excitability and connectivity of multiple brain regions in an attempt to build a comprehensive understanding of an individual's neurophysiology after stroke via TMS assessment is the next challenge for the field. This information would be used to inform the optimal location of stimulation, along with the type, dose, and intensity of rTMS applied to modulate cortical output. While work in this field is slowly emerging (Plow et al., 2014), we propose three steps to support the development of an individualized approach. First, there is a need to consider ipsilesional excitability of primary motor and other higher-order motor-related (e.g., PMd) areas via TMS. If ipsilesional premotor cortices are compensating for decreased M1 excitability, this may indicate that excitatory rTMS over premotor cortices may be a viable option for individuals to enhance the endogenous cortical reorganization taking place to promote better motor recovery. Second, we propose it is vital to take into account if the ICHM applies to an individual after stroke, as this would greatly impact the potential positive (or negative) response to inhibitory rTMS over the contralesional M1. If in fact the contralesional hemisphere is a "friend" after stroke, assisting in recovery of function, then suppressing contralesional M1 activity with rTMS would possibly be counterproductive to motor recovery (Bradnam, Stinear, Barber, & Byblow, 2012). Third, it is warranted to assess interhemispheric communication from non-M1 areas that may be compensating for a lack of connectivity between ipsilateral and contralateral M1, potentially due to obliteration of ipsilesional M1 in more severe stroke. Such regions include PMd, S1, and DLPFC. Since this type of IHI has been demonstrated in healthy individuals (Ni et al., 2009), and that greater inhibition from the contralesional hemisphere can be associated with greater motor function (Mang et al., 2015), it is possible that other related non-M1 areas could be modulated, and this interhemispheric connectivity may be critical to functional recovery after stroke. By determining the cortical excitability and interhemispheric connectivity profile of an individual via TMS assessment, an individualized rTMS intervention that seeks to either exploit the endogenous reorganization of the brain or assist in enhancing excitability or connectivity in the area that is needed to increase the effects of therapy in individuals with stroke can be developed.

Concluding Remarks

TMS can be used to assess and alter cortical excitability and interhemispheric connectivity in healthy and clinical populations (e.g., stroke). Increasingly, rTMS is studied for its ability to augment behavioral interventions in individuals who have had a stroke with some degree of success. Unfortunately, there is a large amount of variability in response to rTMS in individuals with stroke. We propose the use of multiple indices of cortical excitability and interhemispheric connectivity in both M1 and non-M1 areas in order to guide and inform rTMS application. This will support the formulation of an individualized approach to rTMS application that is tailored to positively modulate cortical excitability and interhemispheric connectivity to increase response to therapy and motor function after stroke.

Author Biographies



Jason L. Neva, PhD, Postdoctoral Fellow, Brain Behaviour Laboratory, Department of Physical Therapy, University of British Columbia

Jason completed his BSc and MSc in Kinesiology and Health Science at York University. He went on to complete his PhD in Kinesiology at the University of Waterloo. Building on these training foundations, Jason is currently a postdoctoral fellow in the Brain Behaviour Laboratory at the University of British Columbia where he holds fellowship grants from the Canadian Institutes of Health Research and the Michael Smith Foundation for Health Research.

His research aims are twofold: (a) to understand how experience-dependent factors such as skilled motor learning, exercise, and noninvasive brain stimulation modulate neuroplasticity in the central nervous system and (b) how this understanding of neuroplasticity can positively alter brain activity, motor performance, and motor outcomes following stroke and other neurological disorders.

Kathryn S. Hayward, PhD GCResComm BPhy(HonsI), Postdoctoral Fellow, Brain Behaviour Laboratory, Department of Physical Therapy, University of British Columbia, and Florey Institute of Neuroscience and Mental Health, University of Melbourne.

Kathryn completed her BPhy at James Cook University and her PhD at the University of Queensland, both in Australia. She is currently a National Health and Medical Research Council of Australia Neil Hamilton Fairley Early Career Research Fellow, which has positioned her between the University of British Columbia in Canada and the Florey Institute at the University of Melbourne in Australia.



Her research builds on her clinical experience as a physiotherapist and passion to enable stroke survivors to achieve real-world use of the arm post stroke. Her work seeks to understand how the brain changes in response to stroke (particularly severe stroke) to inform development of innovative training approaches that can enable stroke survivors to achieve meaningful upper limb recovery post stroke.

Dr. Lara A. Boyd, PT, PhD, is the Canada Research Chair in Neurobiology of Motor Learning, a Michael Smith Foundation for Health Career Investigator, a Peter Wall Scholar, and a professor in the Department of Physical Therapy at the University of British Columbia. She is a neuroscientist and physical therapist. Dr. Boyd directs the Brain Behaviour Lab at the University of British Columbia



and has published over 130 peer-reviewed papers. Her work is centered on answering the question of what limits, and what facilitates, neuroplasticity

References

- Auriat, A. M., Neva, J. L., Peters, S., Ferris, J. K., & Boyd, L. A. (2015). A review of transcranial magnetic stimulation and multimodal neuroimaging to characterize post-stroke neuroplasticity. *Frontiers in Neurology*, 6(October), 1–20. doi:10.3389/fneur.2015.00226
- Bhatt, E., Nagpal, A., Greer, K. H., Grunewald, T. K., Steele, J. L., Wiemiller, J. W., ... Carey, J. R. (2007). Effect of finger tracking combined with electrical stimulation on brain reorganization and hand function in subjects with stroke. *Experimental Brain Research*, 182(4), 435–447. doi:10.1007/s00221-007-1001-5
- Borich, M., Neva, J., & Boyd, L. (2015). Evaluation of differences in brain neurophysiology and morphometry associated with hand function in individuals with chronic stroke. *Restorative Neurology and Neuroscience*, 33(1), 31–42.
- Borojerdi, B., Diefenbach, K., & Ferbert, A. (1996). Transcallosal inhibition in cortical and subcortical cerebral vascular lesions. *Journal of the Neurological Sciences*, 144(1–2), 160–170.
- Bradnam, L. V., Stinear, C. M., Barber, P. A., & Byblow, W. D. (2012). Contralesional hemisphere control of the proximal paretic upper limb following stroke. *Cerebral Cortex*, 22(11), 2662–2671. doi:10.1093/cercor/bhr344
- Brodie, S. M., Meehan, S. K., Borich, M. R., & Boyd, L. A. (2014). 5 Hz repetitive transcranial magnetic stimulation over the ipsilesional sensory cortex enhances motor learning after stroke. *Frontiers in Human Neuroscience*, 8(March), 1–10. doi:10.3389/fnhum.2014.00143
- Brown, K., Neva, J., Ledwell, N., & Boyd, L. A. (2014). Use of transcranial magnetic stimulation in the treatment of selected movement disorders. *Degenerative Neurological and Neuromuscular Disease*, 4, 133–151.
- Carey, J. R., Deng, H., Gillick, B. T., Cassidy, J. M., Anderson, D. C., Zhang, L., & Thomas, W. (2014). Serial treatments of primed low-frequency rTMS in stroke: Characteristics of responders vs. nonresponders. *Restorative Neurology and Neuroscience*, 32(2), 323–335. doi:10.3233/RNN-130358
- Carey, J. R., Fregni, F., & Pascual-Leone, A. (2006). rTMS combined with motor learning training in healthy subjects. *Restorative Neurology and Neuroscience*, 24(3), 191–199.
- Chen, R., Classen, J., Gerloff, C., Celnik, P., Wassermann, E. M., Hallett, M., & Cohen, L. G. (1997). Depression of motor cortex excitability by low-frequency transcranial magnetic stimulation. *Neurology*, 48(5), 1398–1403.
- Chen, R., Cros, D., Curra, A., Di Lazzaro, V., Lefaucheur, J.-P., Magistris, M. R., ... Ziemann, U. (2008). The clinical diagnostic utility of transcranial magnetic stimulation: Report of an IFCN committee. *Clinical Neurophysiology*, 119(3), 504–532. doi:10.1016/j.clinph.2007.10.014
- Corti, M., Patten, C., & Triggs, W. (2012). Repetitive transcranial magnetic stimulation of motor cortex after stroke. *American Journal of Physical Medicine & Rehabilitation*, 91(3), 254–270. doi:10.1097/PHM.0b013e318228bf0c
- Di Lazzaro, V., & Ziemann, U. (2013). The contribution of transcranial magnetic stimulation in the functional evaluation of microcircuits in human motor cortex. *Frontiers in Neural Circuits*, 7(February), 18. doi:10.3389/fncir.2013.00018
- Di Pino, G., Pellegrino, G., Assenza, G., Capone, F., Ferreri, F., Formica, D., ... Di Lazzaro, V. (2014). Modulation of brain plasticity in stroke: A novel model for neurorehabilitation. *Nature Reviews. Neurology*, 10(10), 597–608. doi:10.1038/nrneurol.2014.162
- Duque, J., Hummel, F., Celnik, P., Murase, N., Mazzocchio, R., & Cohen, L. G. (2005). Transcallosal inhibition in chronic subcortical stroke. *NeuroImage*, 28(4), 940–946. doi:10.1016/j.neuroimage.2005.06.033

- Frost, S. B., Barbay, S., Friel, K. M., Plautz, E. J., Nudo, R. J., Frost, S. B., ... Nudo, R. J. (2003). Reorganization of remote cortical regions after ischemic brain injury: A potential substrate for stroke recovery. *Journal of Neurophysiology*, 89, 3205–3214. doi:10.1152/jn.01143.2002
- Hamada, M., Murase, N., Hasan, A., Balaratnam, M., & Rothwell, J. C. (2013). The role of interneuron networks in driving human motor cortical plasticity. *Cerebral Cortex*, 23(7), 1593–1605. doi:10.1093/cercor/bhs147
- Harris-Love, M. L., Chan, E., Dromerick, A. W., & Cohen, L. G. (2015). Neural substrates of motor recovery in severely impaired stroke patients with hand paralysis. *Neurorehabilitation and Neural Repair*. doi:10.1177/1545968315594886
- Hayward, K. S., Neva, J. L., Peters, S., Ferris, J. K., Wadden, K. P., Mang, C. S., Boyd, L. A. (2017). The contralesional hemisphere may play an important role in support of motor recovery in people with severe arm impairment after stroke. *Neural Plasticity Publication*. Philadelphia, PA, USA. doi: 10.1155/2017/4281532. PMID: 29348943
- Héroux, M. E., Taylor, J. L., & Gandevia, S. C. (2015). The use and abuse of transcranial magnetic stimulation to modulate corticospinal excitability in humans. *PLoS One*, 10(12), 1–10. doi:10.1371/journal.pone.0144151
- Hsu, W. Y., Cheng, C. H., Liao, K. K., Lee, I. H., & Lin, Y. Y. (2012). Effects of repetitive transcranial magnetic stimulation on motor functions in patients with stroke: A meta-analysis. *Stroke*, 43(7), 1849–1857. doi:10.1161/STROKEAHA.111.649756
- Huang, Y. Z., Edwards, M. J., Rounis, E., Bhatia, K. P., & Rothwell, J. C. (2005). Theta burst stimulation of the human motor cortex. *Neuron*, 45(2), 201–206. doi:10.1016/j.neuron.2004.12.033
- Liepert, J., Miltner, W. H., Bauder, H., Sommer, M., Dettmers, C., Taub, E., & Weiller, C. (1998). Motor cortex plasticity during constraint-induced movement therapy in stroke patients. *Neuroscience Letters*, 250(1), 5–8.
- Mang, C. S., Borich, M. R., Brodie, S. M., & Boyd, L. A. (2015). Diffusion imaging and transcranial magnetic stimulation assessment of transcallosal pathways in chronic stroke. *Clinical Neurophysiology*, 126(10), 1959–1971. doi:10.1016/j.clinph.2014.12.018
- Manganotti, P., Patuzzo, S., Cortese, F., Palermo, A., Smania, N., & Fiaschi, A. (2002). Motor disinhibition in affected and unaffected hemisphere in the early period of recovery after stroke. *Clinical Neurophysiology*, 113(6), 936–943. doi:10.1016/S1388-2457(02)00062-7
- Meehan, S. K., Dao, E., Lindsell, M. A., & Boyd, L. A. (2011). Continuous theta burst stimulation over the contralesional sensory and motor cortex enhances motor learning post-stroke. *Neuroscience Letters*, 500(1), 26–30. doi:10.1016/j.neulet.2011.05.237
- Murase, N., Duque, J., Mazzocchio, R., & Cohen, L. G. (2004). Influence of interhemispheric interactions on motor function in chronic stroke. *Annals of Neurology*, 55(3), 400–409. doi:10.1002/ana.10848
- Ni, Z., Gunraj, C., Nelson, A. J., Yeh, I. J., Castillo, G., Hoque, T., & Chen, R. (2009). Two phases of interhemispheric inhibition between motor related cortical areas and the primary motor cortex in human. *Cerebral Cortex*, 19(July), 1654–1665. doi:10.1093/cercor/bhn201
- Plow, E. B., Cunningham, D. A., Varnerin, N., & Machado, A. (2014). Rethinking stimulation of the brain in stroke rehabilitation: Why higher motor areas might be better alternatives for patients with greater impairments. *The Neuroscientist: A Review Journal Bringing Neurobiology, Neurology and Psychiatry*. doi:10.1177/1073858414537381
- Rioutl-Pedotti, M. S., Friedman, D., & Donoghue, J. P. (2000). Learning-induced LTP in neocortex. *Science*, 290(5491), 533–536.
- Sawaki, L., Butler, A. J., Leng, X., Wassenaar, P. A., Mohammad, Y. M., Blanton, S., ... Wittenberg, G. F. (2008). Constraint-induced movement therapy results in increased motor map area in subjects 3 to 9 months after stroke. *Neurorehabilitation and Neural Repair*, 22(5), 505–513. doi:10.1177/1545968308317531
- Shimizu, T., Hosaki, A., Hino, T., Sato, M., Komori, T., Hirai, S., & Rossini, P. M. (2002). Motor cortical disinhibition in the unaffected hemisphere after unilateral cortical stroke. *Brain: A Journal of Neurology*, 125(Pt 8), 1896–1907.

- Stinear, C. M., Barber, P. A., Petoe, M., Anwar, S., & Byblow, W. D. (2012). The PREP algorithm predicts potential for upper limb recovery after stroke. *Brain*, 135, 2527–2535. doi:10.1093/brain/aws146
- Taub, E., Uswatte, G., King, D. K., Morris, D., Crago, J. E., & Chatterjee, A. (2006). A placebo-controlled trial of constraint-induced movement therapy for upper extremity after stroke. *Stroke*, 37(4), 1045–1049. doi:10.1161/01.STR.0000206463.66461.97
- Trompetto, C., Assini, A., Buccolieri, A., Marchese, R., & Abbruzzese, G. (2000). Motor recovery following stroke: A transcranial magnetic stimulation study. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, 111(10), 1860–1867. doi:10.1016/S1388-2457(00)00419-3

Connectomics and Multimodal Imaging Techniques in Clinical Research

Olusola Ajilore¹ and Alex Leow^{1,2}

¹*Department of Psychiatry, College of Medicine, University of Illinois at Chicago
Chicago, IL, USA*

²*Department of Bioengineering, College of Medicine, University of Illinois at Chicago,
Chicago, IL, USA*

Introduction

One of the most exciting developments in clinical neuroscience is the conceptualization of the brain as an interconnected network or (mathematically speaking) a graph. This new area of study called connectomics has already yielded invaluable insights into brain function and dysfunction in the context of neuropsychiatric diseases. This chapter will introduce the reader to the topic of connectomics followed by an overview of key findings associated with a few neuropsychiatric disorders studied using this innovative technique.

What Is Connectomics?

Connectomics refers to an innovative school of methods that allow us to study system properties of the human brain by constructing comprehensive maps of brain connectivity, both structural and functional, based on noninvasive neuroimaging modalities. Mathematically, such “connectome” studies model the brain as a “graph,” composed of the nodes or “vertices” (cortical and subcortical gray matter regions) and the connections or “edges” (defined using either structural or functional information) connecting these nodes. In this context, the term connectomics refers to a collection of mathematical and computational approaches to the study of the brain, as it is represented as a network composed of the aforementioned nodes and edges. As an

example, a structural “tractography”-derived connectome has edges that are defined by white matter connectivity extracted using diffusion tensor imaging (DTI), which allows one to quantify the number of white matter “fiber” tracts between brain regions. By contrast, edges in a functional magnetic resonance imaging (fMRI)-derived connectome are typically defined based on the correlation of the blood oxygen level-dependent (BOLD) signals in fMRI between brain regions or nodes. Similarly, another form of noninvasive functional connectome can be defined using electroencephalography (EEG), where edges are defined as the correspondence (based on signal’s power spectrum, phase information, etc.) between EEG signals from electrodes. Once the brain has been modeled in this manner, various graph theory-based metrics can be calculated in order to better elucidate the functional and structural organization of the brain (Rubinov & Sporns, 2010) as a result of the interactions among all connections connecting different areas in the brain (instead restricted to a select few regions or white matter tracts as was commonly done prior to the advent of connectomics). Common metrics described in the literature include path length and global efficiency (both measures of network integration), clustering coefficient (a measure of network segregation), and centrality (a measure of nodal importance or influence). From here, the next level of graph connectome analysis can be applied in order to understand the modular or community structure of a connectome. Indeed, just as social networks can be divided into cliques that describe modes of association (family, school, etc.), a connectome can be divided into modules or communities.

To measure how efficient two nodes of a connectome communicate with each other, one computes the graph distance or shortest path length between them. Calculated by averaging the lengths of shortest paths between all pairs of nodes, the characteristic path length (CPL) (Rubinov & Sporns, 2010) is commonly used to measure overall network integration (node-level path length is defined similarly by averaging the shortest paths connecting an index node to all other nodes in the network). By contrast, the clustering coefficient is a measure of network segregation and reflects the tendency of a node’s neighbors to be connected to each other. Highly connected nodes have a high degree of centrality, that is, a large proportion of all edges pass through a given node. Sporns and colleagues have identified brain regions with typically high centrality, which they called the “rich club” (van den Heuvel & Sporns, 2011). These nodes have been shown to be particularly important when examining specific diseases from a network perspective.

Connectomics of Psychiatric Disorders

Schizophrenia

The largest body of work in the emerging field of connectomics and psychiatry comes from studies of schizophrenia. Even before the advent of connectomics, researchers have long-conceptualized schizophrenia as a connectivity disorder (Bullmore, Frangou, & Murray, 1997). In recent years, several investigators have discovered brain network abnormalities consistent with this notion of schizophrenia as a connectivity disorder. One of the first studies of connectomics and schizophrenia published by Lynall et al. revealed that participants with schizophrenia had reduced network strength and increased variance of functional connectivity strengths, among other network alterations (Lynall et al., 2010). The authors concluded that in schizophrenia, brain networks were less integrated with decreased influence of “hub” nodes. Altered hub nodes were also noted in a study that examined rich club organization in schizophrenia; here the authors found a reduced density of highly connected rich club nodes that linked frontal, parietal, and insular regions and thus resulted in reduced global communication capacity (van den Heuvel et al., 2013).

How might some of these network alterations be related to one of the cardinal symptoms of schizophrenia, hallucinations? In a study of patients with the 22q11.2 deletion syndrome, hallucination symptom severity was significantly associated with local efficiency in speech centers like Broca's area and Wernicke's area (Ottet et al., 2013). Further, two recent studies have identified that abnormal connectome properties in the default mode network are associated with hallucinations. Chang and colleagues showed that auditory-visual hallucinations were especially associated with abnormal bilateral connectivity of the default mode network and inferior frontal gyrus (Chang et al., 2015). A similar pattern was identified by Wang et al. showing increased connectivity between the default mode network and heteromodal association areas (Wang, Zeng, et al., 2015). While these studies demonstrate connectome alterations associated with specific symptoms and the syndrome of schizophrenia in general, one area that requires further study is the impact of antipsychotic treatments on connectome properties and whether there are connectome-based predictors of treatment response.

Mood Disorders

Connectomics has been extensively studied in the area of mood disorders. Studies of structural connectomes in late life depression have revealed impaired network efficiency, both in the active and remitted state of the disease (Ajilore et al., 2014; Bai et al., 2012). Furthermore, it has been shown that late life depression is associated with impaired network resiliency that is partially mediated by white matter hyperintensity burden (a common radiological finding in LLD) (Ajilore et al., 2014; Mak, Colloby, Thomas, & O'Brien, 2016). In examining alterations in hub regions in both late life depression and bipolar depression, there is evidence of altered network influence and network organization implicating key regions in the default mode network (Ajilore et al., 2014; Charlton et al., 2015; Gadelkarim et al., 2014). Examining functional connectomes with fMRI, late life depression is associated with reduced edge strength in circuits involved in emotion regulation and reward processing (Tadayonnejad, Yang, Kumar, & Ajilore, 2014).

Connectomic studies in bipolar disorder have revealed impaired white matter integrity in the corpus callosum that is related to impaired global network segregation and integration. Furthermore, like unipolar depression, patients with bipolar disorder exhibited decreased global network efficiency and interhemispheric efficiency (Leow et al., 2013). Demonstrating how these structural alterations relate to function, it has been shown that these structural connectome properties mediate abnormalities in fMRI activation and cognitive performance in euthymic bipolar I patients (Ajilore et al., 2015).

The impact of treatment on connectome alterations in mood disorders has been demonstrated in several studies across different therapeutic modalities. For example, betweenness centrality (a measure of "hubness") of the subgenual cingulate distinguished transcranial magnetic stimulation (rTMS) responders from nonresponders (Downar et al., 2014). Antidepressant treatment has been shown to reduce functional connectivity strength in the bilateral dorsomedial prefrontal cortex and increase hippocampal functional connectivity strength (Wang, Xia, et al., 2015). Additionally, electroconvulsive therapy (ECT) treatment response is associated with structural connectome changes in limbic regions such as the amygdala and parahippocampus and with nodal strength increases in paralimbic regions (Zeng et al., 2015).

Anxiety Disorders

Compared with schizophrenia and mood disorders, there have been fewer studies in anxiety disorders. A recent study by Servaas et al. showed that people with high levels of neuroticism had more randomly organized brain networks compared with those with low levels of neuroticism

(Servaas et al., 2015). Furthermore, these participants had relatively higher efficiency in an affective subnetwork. In a connectome-wide analysis study by Satterthwaite and colleagues, anxiety was associated increased connectivity between ventromedial prefrontal cortex and the amygdala (Satterthwaite et al., 2016).

In obsessive-compulsive disorder (OCD), there have been several interesting studies examining the impact of treatment on connectomics measures. For example, it has been shown that cognitive behavioral therapy (CBT) can enhance functional network efficiency in OCD (Feusner et al., 2015). Additionally, pharmacological treatment with selective serotonin reuptake inhibitors (SSRI) has been shown to impact brain network characteristics in OCD. Shin and colleagues found that participants with OCD had reduced functional network efficiency that increased with SSRI treatment. Furthermore, they found that treatment response correlated with changes in network connectivity of the right ventral frontal cortex (Shin et al., 2014).

Future Directions

The majority of studies described above have primarily used a single modality such as fMRI or DTI to generate connectomes. However, combining multimodal neuroimaging techniques allows for more complete models of brain function and dysfunction. In a study using a multimodal fusion technique called functional-by-structural hierarchical mapping, it was shown that combining two modalities enhanced the power to detect significant differences compared with either modality alone (Ajilore et al., 2013).

In addition to incorporating multiple modalities, studies are currently underway to improve how we characterize functional connections in brain networks. Current fMRI methods typically use Pearson correlations as a measure of connectivity. However, this method may overestimate the number of “true” functional connections. Using partial correlations and other information theory-based methods may lead to more accurate functional connectomes (Cassidy, Rae, & Solo, 2015).

Another advance in the characterization of functional connectomes derives from the recognition that resting-state fMRI does not truly reflect the brain at rest but instead, brain activity is very dynamic at all times. Novel connectomics methods, such as time-resolved functional connectivity and sliding-window techniques, are being developed to capture these dynamics. Along this line, work by Bassett and colleagues has shown how network dynamics relate to cognitive function and adaptive flexibility (Bassett et al., 2011; Braun et al., 2015).

Conclusion

Understanding the brain as a network has tremendous implications for how we characterize neuropsychiatric disorders. Rather than conceptualize diseases as arising from dysfunction in a few select brain regions, we understand that these complicated disorders often arise from disruptions in connectivity between regions. By thinking of the brain as a complex network of nodes and connections, novel treatments can be designed to target key nodes in the network defined by connectivity patterns. As described above, innovative neuroimaging and integrative techniques employing multimodal synthesis of imaging data and graph theory-based network analysis help to characterize the impact of disrupted tracts, networks, and whole-brain connectivity in neuropsychiatric diseases. Identifying abnormal circuits in this population and variances at the individual level could be used to develop patient-specific targets for future

interventions, such as brain stimulation techniques or psychotherapies designed to engage, augment, or bypass these circuits.

Author Biographies

Dr. Olusola Ajilore is an associate professor in the Department of Psychiatry at the University of Illinois–Chicago. He graduated magna cum laude from Harvard University with a degree in biology. Dr. Ajilore went on to do his MD/PhD degree at Stanford University where he studied the deleterious effects of stress hormones on the brain. He joined the research track residency at UCLA where he transitioned into neuroimaging in major depression. He is currently using novel neuroimaging techniques to better understand the pathophysiology of mood disorders.

Dr. Alex Leow is an associate professor in the Departments of Psychiatry, Bioengineering, and Computer Science at the University of Illinois–Chicago. With both a doctoral degree in applied mathematics and a medical board certification in adult psychiatry, Dr. Leow's interdisciplinary research efforts have included the development and application of state-of-the-art computational neuroimaging methods, across multiple MR imaging modalities (structural, diffusion weighted, functional, etc.) and more recently EEG. In addition, clinically Dr. Leow has evaluated, diagnosed, treated, and followed thousands of individuals with complex neuropsychiatric conditions.

References

- Ajilore, O., Lamar, M., Leow, A., Zhang, A., Yang, S., & Kumar, A. (2014). Graph theory analysis of cortical-subcortical networks in late-life depression. *The American Journal of Geriatric Psychiatry*, 22(2), 195–206.
- Ajilore, O., Vizuetta, N., Walshaw, P., Zhan, L., Leow, A., & Altshuler, L. L. (2015). Connectome signatures of neurocognitive abnormalities in euthymic bipolar I disorder. *Journal of Psychiatric Research*, 68(0), 37–44.
- Ajilore, O., Zhan, L., Gadelkarim, J., Zhang, A., Feusner, J. D., Yang, S., et al. (2013). Constructing the resting state structural connectome. *Frontiers in Neuroinformatics*, 7, 30.
- Bai, F., Shu, N., Yuan, Y., Shi, Y., Yu, H., Wu, D., et al. (2012). Topologically convergent and divergent structural connectivity patterns between patients with remitted geriatric depression and amnesic mild cognitive impairment. *The Journal of Neuroscience*, 32(12), 4307–4318.
- Bassett, D. S., Wymbs, N. F., Porter, M. A., Mucha, P. J., Carlson, J. M., & Grafton, S. T. (2011). Dynamic reconfiguration of human brain networks during learning. *Proceedings of the National Academy of Sciences of the United States of America*, 108(18), 7641–7646.
- Braun, U., Schafer, A., Walter, H., Erk, S., Romanczuk-Seiferth, N., Haddad, L., et al. (2015). Dynamic reconfiguration of frontal brain networks during executive cognition in humans. *Proceedings of the National Academy of Sciences of the United States of America*, 112(37), 11678–11683.
- Bullmore, E. T., Frangou, S., & Murray, R. M. (1997). The dysplastic net hypothesis: An integration of developmental and dysconnectivity theories of schizophrenia. *Schizophrenia Research*, 28(2-3), 143–156.
- Cassidy, B., Rae, C., & Solo, V. (2015). Brain activity: Connectivity, sparsity, and mutual information. *IEEE Transactions on Medical Imaging*, 34(4), 846–860.
- Chang, X., Xi, Y. B., Cui, L. B., Wang, H. N., Sun, J. B., Zhu, Y. Q., et al. (2015). Distinct inter-hemispheric dysconnectivity in schizophrenia patients with and without auditory verbal hallucinations. *Scientific Reports*, 5, 11218.

- Charlton, R. A., Leow, A., GadElkarim, J., Zhang, A., Ajilore, O., Yang, S., et al. (2015). Brain connectivity in late-life depression and aging revealed by network analysis. *American Journal of Geriatric Psychiatry*, 23(6), 642–650.
- Downar, J., Geraci, J., Salomons, T. V., Dunlop, K., Wheeler, S., McAndrews, M. P., et al. (2014). Anhedonia and reward-circuit connectivity distinguish nonresponders from responders to dorsomedial prefrontal repetitive transcranial magnetic stimulation in major depression. *Biological Psychiatry*, 76(3), 176–185.
- Feusner, J. D., Moody, T., Lai, T. M., Sheen, C., Khalsa, S., Brown, J., et al. (2015). Brain connectivity and prediction of relapse after cognitive-behavioral therapy in obsessive-compulsive disorder. *Frontiers in Psychiatry*, 6, 74.
- Gadelkarim, J. J., Ajilore, O., Schonfeld, D., Zhan, L., Thompson, P. M., Feusner, J. D., et al. (2014). Investigating brain community structure abnormalities in bipolar disorder using path length associated community estimation. *Human Brain Mapping*, 35(5), 2253–2264.
- Leow, A., Ajilore, O., Zhan, L., Arienzo, D., Gadelkarim, J., Zhang, A., et al. (2013). Impaired inter-hemispheric integration in bipolar disorder revealed with brain network analyses. *Biological Psychiatry*, 73(2), 183–193.
- Lynall, M. E., Bassett, D. S., Kerwin, R., McKenna, P. J., Kitzbichler, M., Muller, U., et al. (2010). Functional connectivity and brain networks in schizophrenia. *The Journal of Neuroscience*, 30(28), 9477–9487.
- Mak, E., Colloby, S. J., Thomas, A., & O'Brien, J. T. (2016). The segregated connectome of late-life depression: A combined cortical thickness and structural covariance analysis. *Neurobiology of Aging*, 48, 212–221.
- Ottet, M. C., Schaer, M., Debbane, M., Cammoun, L., Thiran, J. P., & Eliez, S. (2013). Graph theory reveals dysconnected hubs in 22q11DS and altered nodal efficiency in patients with hallucinations. *Frontiers in Human Neuroscience*, 7, 402.
- Rubinov, M., & Sporns, O. (2010). Complex network measures of brain connectivity: Uses and interpretations. *NeuroImage*, 52(3), 1059–1069.
- Satterthwaite, T. D., Cook, P. A., Bruce, S. E., Conway, C., Mikkelsen, E., Satchell, E., et al. (2016). Dimensional depression severity in women with major depression and post-traumatic stress disorder correlates with fronto-amygdalar hypoconnectivity. *Molecular Psychiatry*, 21(7), 894–902.
- Servaes, M. N., Geerligs, L., Renken, R. J., Marsman, J. B., Ormel, J., Riese, H., et al. (2015). Connectomics and neuroticism: An altered functional network organization. *Neuropsychopharmacology*, 40(2), 296–304.
- Shin, D. J., Jung, W. H., He, Y., Wang, J., Shim, G., Byun, M. S., et al. (2014). The effects of pharmacological treatment on functional brain connectome in obsessive-compulsive disorder. *Biological Psychiatry*, 75(8), 606–614.
- Tadayonnejad, R., Yang, S., Kumar, A., & Ajilore, O. (2014). Multimodal brain connectivity analysis in unmedicated late-life depression. *PLoS One*, 9(4), e96033.
- van den Heuvel, M. P., & Sporns, O. (2011). Rich-club organization of the human connectome. *The Journal of Neuroscience*, 31(44), 15775–15786.
- van den Heuvel, M. P., Sporns, O., Collin, G., Scheewe, T., Mandl, R. C., Cahn, W., et al. (2013). Abnormal rich club organization and functional brain dynamics in schizophrenia. *JAMA Psychiatry*, 70(8), 783–792.
- Wang, H., Zeng, L. L., Chen, Y., Yin, H., Tan, Q., & Hu, D. (2015). Evidence of a dissociation pattern in default mode subnetwork functional connectivity in schizophrenia. *Scientific Reports*, 5, 14655.
- Wang, L., Xia, M., Li, K., Zeng, Y., Su, Y., Dai, W., et al. (2015). The effects of antidepressant treatment on resting-state functional brain networks in patients with major depressive disorder. *Human Brain Mapping*, 36(2), 768–778.
- Zeng, J., Luo, Q., Du, L., Liao, W., Li, Y., Liu, H., et al. (2015). Reorganization of anatomical connectome following electroconvulsive therapy in major depressive disorder. *Neural Plasticity*, 2015, 271674.

Asperger Syndrome

Laurent Mottron

*Department of Psychiatry, Faculty of Medicine, Universite de Montreal,
Montreal, QC, Canada*

Introduction

Asperger syndrome (AS) has two independent lives. One is based on popular psychology, pervading TV series and crime literature presenting socially awkward, highly intelligent characters with ritual habits and acute deductive skills. The other is an ephemeral, almost clandestine one based on science, concerning a neurodevelopmental condition included in the autism spectrum. Within current scientific understanding, AS still has an ambiguous existence, based on the decisions of its inventors, re-inventors, or committees' manufacturing consensus, without acknowledging that some people profoundly recognize themselves as having or *being* it. This unstable existence has resulted in an unavoidable circularity between its changing definitions and the reported findings of its characteristics. Here, we present the history of AS, its delineation within and outside of the autism spectrum, its neurobiological and psychiatric counterparts, and AS during childhood development and adulthood and conclude with the reciprocal relations that AS people may engage in with non-autistic people in the modern world.

History: The Four Ages of Asperger Syndrome

Discovery

In 1944, Hans Asperger, a Wiener pediatrician whose career partially overlapped with the years of the Nazi regime, published a series of four case studies of what he called “autistic psychopathy.” Asperger’s detailed description of their atypical social interactions included an abnormal gaze and voice, reduced facial expressions, atypical language and conversation, and self-absorption. These manifestations were associated with several challenges (attention and learning difficulties, stereotypic behaviors, clumsiness, reduced autonomy, and, in some cases, acts of delinquency), as well as advantages, such as creativity, outstanding mastery of speech, logical

thinking, and an at least normal level of general intelligence. Circumscribed interests, a hallmark of the condition, were considered to be useful and beneficial to the person. Up to one-fourth of AS individuals have various specific, above average abilities. Autistic psychopathy was considered to be a lifelong condition, genetically determined, with an onset before the age of 2 years, and was predominately found in males.

Rediscovery

Most important features of autistic psychopathy were identical to Kanner's description of autism 1 year earlier, except for the absence of "autistic language" features, the addition of attention-deficit clumsiness, and the genetic origin (in this latter aspect, Asperger was in advance of Kanner). In the quarter century following Asperger's description, there was an unequal competition of quotations between him and Kanner. Asperger quoted Kanner several times, whereas Kanner ignored him. Asperger's writings were virtually unquoted in non-German literature until 1981, despite the striking similarity between his autistic psychopathy and Kanner's autism, until L. Wing, an English psychiatrist, described 34 individuals that she personally diagnosed, roughly corresponding to Asperger's initial description. She also coined the term Asperger syndrome (Wing, 1981). Wing admittedly modified many important AS features and priorities. In particular, she attenuated the differences between Asperger's and Kanner's description and introduced her own beliefs. She particularly emphasized the sex ratio, clumsiness, and atypical social reciprocity, along with learning difficulties. She assumed a misunderstanding of other's nonverbal expressions, "lack of ability to understand and use the rules governing social behavior," and a lack of empathy. Most of the people that Wing described in her paper would have been classified as autistic based on further DSM-IV criteria, due to the presence of signs now included in the definition of verbal autism. These included early signs, such as overt, reduced interest and pleasure in human company, and the absence of pretend play during the first year, and later signs, such as pronoun reversal, stereotypical language, delayed echolalia, and a perceptual fascination with spinning objects. She minimized the role of genetics, in favor of acquired neurological conditions.

Among the changes introduced to Asperger's definition in that of Wing were an emphasis on the negative aspects of the syndrome and a generally more severe and pessimistic view of AS people. Although 76% of the individuals in her group had specific interests, their recognized skills were limited to rote memory, chess play, and music, despite that some may had the ability to follow careers in their domain of interest. She minimized the specific relation that AS people have with language and negatively described their language abilities. She was also highly critical of their originality and creativity in her description, as well as the notion of their high intelligence. These potentially positive assessments were replaced by an overall "lack of common sense." The study emphasized psychiatric co-occurring conditions, especially depression. Finally, she presented the reaction of others to AS people as varying "between compassion and exasperation."

Wing's paper generated great interest in Western countries and inaugurated a self-organized nosographic period where numerous important figures in autism research, liberated by Wing's first attempt to describe the condition and by the absence of a biological or symptomatic gold standard, produced their own sets of criteria (Gillberg & Gillberg, 1989; Szatmari, 1992; Szatmari, Bartolucci, & Bremner, 1989). The operationalization of some of these criteria in short and nonspecific questionnaires contributed to the popularity of AS but undermined the specificity of its initial description—foreshadowing the future loss of the information signal detected by its inventor.

Transient Life as an Official Entity (1994–2013)

In a concerted but awkward attempt to restore some order to the AS field, the DSM-IV committee tried to establish consensual criteria to capture the specificity between Asperger's and Kanner's initial descriptions. They re-separated the criteria merged by L. Wing into two distinct entities. The DSM-IV committee defined autism by three groups of symptoms (social, communication, and restricted interests and repetitive behaviors [RIRB]) and AS by only two (social and RIRB), which were identical to those of autism. They also proposed a priority rule: anybody satisfying criteria for autism could not be considered to have AS. The diagnosis of AS could be used only in the absence of a delay in the onset of speech, cognition, or autonomy.

Many were dissatisfied with the DSM-IV criteria during its era of application. The exclusion of language signs from the definition of AS combined with a priority rule was unfortunate. Language use is clearly atypical, even in prototypical AS. Prosody, quantity and rhythm, the amount of information provided, formalism, and topic adjustment are altered in AS, but the structural aspects of language use may be of very high quality. As a consequence, all clinically defined AS satisfied the criteria for DSM-IV-defined autism and disappeared. Moreover, the use of identical RIRB signs to diagnose AS and autism did not take into account that RIRB may take a different form, depending on whether or not the onset of speech is delayed (e.g., perceptual fixations in autism, thematic interests in AS). Another aspect that highlighted the unreliability of the autism vs. AS distinction was the reported absence of distinctive cognitive profiles. Its demonstration would have required a clear behavioral distinction between the two subgroups, which was not possible using the DSM-IV criteria.

Official Disappearance and Resistance (2013 to the Present)

The DSM-5 committee (American Psychiatric Association, 2013) took a very different view and merged the precedent subcategories of pervasive developmental disorders (autism, Asperger, and pervasive developmental disorders not otherwise specified) into an autism spectrum, possibly more specific than that of the DSM-IV. The DSM-5 recommendations introduce speech and intelligence levels among the clinical specifiers, merging the pervasive developmental disorder subgroups of the DSM-IV into an infinite number of possible clinical pictures within the autistic spectrum. It follows a "build your own autistic" strategy to cope with the presumed heterogeneity of the spectrum. However, the DSM-5 suggests keeping the old diagnoses and not recoding them according to the new criteria. The ICD-10 maintains the category but blurs it, disregards it, and poorly influences research decisions: "A disorder of uncertain nosological validity, characterized by the same type of qualitative atypicalities of reciprocal social interaction that typify autism, together with a restricted, stereotyped, repetitive repertoire of interests and activities. It differs from autism primarily in the fact that there is no general delay or retardation in language or in cognitive development. This disorder is often associated with marked clumsiness. There is a strong tendency for the abnormalities to persist into adolescence and adult life. Psychotic episodes occasionally occur in early adult life."

The disappearance of AS had been in preparation since the late 1980s by the diagnostic instruments (ADI and ADOS) that operationalize the DSM diagnostic criteria. Accordingly, there was no accepted standardized instrument to diagnose it, consistent with the doubt of the independent existence of the AS category. ADI and ADOS either merged AS people into a unique autism category by giving them an autism diagnosis or failed to detect it. The ADI

questionnaire and the ADOS observation scale are indeed not sufficiently sensitive concerning communication to detect AS people, who speak fluently and do not present *major* communication issues. Either the person has sufficient communication issues to be considered autistic, or, more frequently, this is not the case, and the person is considered to be subthreshold. In both cases, AS disappears as an independent entity.

A strong scientific (Lai, Lombardo, Chakrabarti, & Baron-Cohen, 2013) and journalistic reaction between the pros and cons of the DSM-5 decision followed and is still active, sometimes fueled by information on Asperger's role during World War II (Czech, 2018). However, Asperger's inexcusable attitude toward the Nazi occupants is irrelevant to the scientific validity of his description of the condition, although it may play a role in the decision to attach his name to it. There remains an as yet unresolved contradiction between the weak scientific consensus still plaguing AS and the strong self-recognition of it by numerous individuals. As a result, research focused on AS has diminished considerably. Another detrimental consequence of including AS within autism is that it has allowed the inclusion of a more heterogeneous population in research studies than during the DSM-IV era, with the obvious result: the marked absence of major discoveries in prototypical autism, AS, or even autism spectrum disorders in brain imaging studies and genetics during the last 10 years.

Our Position

Our position in 2016 is that a sufficiently large number of individuals present prototypical AS of a sufficiently similar form to justify its autonomous designation. Despite its fuzzy definition, the condition still exists. Within the autistic spectrum, the co-segregation of the absence of a speech onset delay (SOD) with strong, verbally defined interests and a unique profile of cognitive strengths justifies its recognition. The spectrum of RIRB of AS people is also different from those with autism: when people diagnosed with autism at school age, who have access to oral speech, are stratified according to whether or not they had an SOD (or at least, clearly defined echolalia and stereotyped language), self-injury, preoccupation with parts of objects, or manual stereotypies are found only in those with an SOD. In contrast, AS people present stronger and different motor atypicalities than autistic individuals.

Despite controversies concerning its validity and diagnostic borders, AS exists in a prototypical form and is well characterized by the DSM-IV criteria, granted one withdraws the rule giving priority to autism, adds the possibility of minor communication atypicalities, and gives greater weight to the cognitive profile. A conservative view of the verbal autism vs. AS distinction is that when faced with an autistic clinical presentation as an adult, different predictions and clinical symptoms can be expected depending on whether the patient had an SOD or not. A prototypical presentation of AS involves a past history of reduced sharing of interests (theirs or his) with peers, self-consciousness of being different beginning at school age, and strong intellectual and emotional investment in thematic domains developing in coherent networks, together with normal or higher syntactic and vocabulary abilities and the absence of an SOD.

We have been able to arrive to this conclusion, despite the notorious difficulty in finding criteria that distinguish the two subgroups, ultimately resulting in the DSM-5 decision. It is possible to unravel the roots of the neurocognitive differences between *clinically defined* autistic and AS individual by comparing strictly defined individuals who are equivalent in their magnitude of social atypicalities and differ only by the presence or absence of an SOD (SOD vs. no SOD). Typical versus delayed speech acquisition milestones predict better developmental (Mayo, Chlebowski, Fein, & Eigsti, 2013) and language (Kenworthy et al., 2012) outcome

in the autism spectrum. Concerning cognition, individuals with an SOD exhibit faster visual processing (Barbeau, Soulières, Dawson, Zeffiro, & Mottron, 2013), better dexterity and bimanual coordination (Barbeau, Meilleur, Zeffiro, & Mottron, 2015), superior auditory discrimination but impaired motion discrimination, and increased visual pursuit latency (Takarae, Luna, Minshew, & Sweeney, 2008) than individuals without an SOD. One fMRI study reported greater activity in the auditory-processing region for individuals with an SOD vs. greater activity in the language-processing region in those without an SOD when listening to nonspeech sounds. The AS cognitive profile also presents a higher VIQ than PIQ. DSM-5 autistics with or without SOD display distinct cognitive profiles by standard Wechsler intelligence profiling, with SOD individuals presenting superior visual and visuospatial performance relative to baseline, whereas individuals without SOD display distinct abilities in similitude and vocabulary (Sahyoun, Soulieres, Belliveau, Mottron, & Mody, 2009). The loss of this information, which was subsequently never replicated, results from the clinical definition of the participants, mixing verbal autistic adults with and without a history of SOD. Distinguishing individuals with or without SOD on the basis of their cognitive strengths and superior cortical reallocation suggests that autism is a condition that exhibits enhanced cortical plasticity, for which the targets, perception or language, differ according to subgroups, and suggests that AS individuals are speech experts (Samson, Zeffiro, Doyon, Benali, & Mottron, 2015).

Epidemiology

We are certain that prototypical AS individuals exist and are similar to each other, but the epidemiology of AS is still hazardous because of its fuzzy definition and ill-defined boundaries. If we limit AS to autistic individuals without SOD, its prevalence is still dependent on the level of atypicality required for the diagnosis of AD itself. For example, do we include the Japanese hikikomoris (close to the socio-communicative aspects of AS) or otakus (close to the RIRB aspects of AS) within AS? The difference between narrow and wide case ascertainment substantially affects the resulting prevalence. The occurrence of autistic people without SOD is, by definition, less frequent than the autism spectrum in its entirety, but also probably less frequent than autistic people with SOD. The recent emergence of a possible difference of AS according to gender, and the unavoidable circularity between case ascertainment based on gender and gender-specific criteria, prevents establishing a final sex ratio. Asperger's files following the war that could be studied (1950–1986) show the sex ratio for autistic psychopathy to be as high as 24/1. Another issue that prevents epidemiology from providing reliable numbers is the lack of a consensus regarding whether or not to exclude alternative diagnoses, the over-inclusivity of diagnostic instruments, and their lack of specificity. Many autism experts only know autism and fail to apply an “economy principle” when faced with ambiguous diagnoses. The prevalence of AS that they diagnose is therefore inversely correlated to their level of ignorance of other conditions.

Genetic Issues

An association between AS and autism was suspected before DSM-5. The clinical similarity between the broader autistic phenotype and the sociocognitive atypicalities evident in the first-degree relative of autistic and AS individuals contributed to the unification of the various forms within the autism spectrum. Twenty-five years ago, the common opinion was that

familial genetic mechanisms were even more important in the AS than autism subtype. Asperger was convinced of this due to the apparent male transmission and high sex ratio. The broadening of the criteria and the return of syndromic autism (were *de novo* mutations are predominant) as a heuristic way to investigate neurobiological aspects of autism made this notion disappear. We predict that it will come back, in the same way that macrocephaly, evident for Kanner, was rediscovered 50 years later. Despite strong claims to the contrary, the role of environmental factors (beyond the intrinsic relation between gene expression and environment) has not been empirically demonstrated and has only been demonstrated for a few limited syndromic, para-autistic clinical presentations. Similarly, *de novo* mutations produce mostly intellectual disability syndromes for which the link with the presentation of prototypical AS is, at least, a matter of debate.

Differential Diagnoses

The absence of gold standards for determining whether patients fall into the autism spectrum or not leads to unavoidable disagreement on the actual diagnoses of people at the margin of prototypical descriptions. Confusing AS with “mild” autism within the autistic spectrum puts the patient at risk of missing that intelligence and better speech abilities may increase the risk for depressive thoughts. AS is not mild when things go wrong, and severity (in terms of well-being) and marginality (distance from the prototypical non-autistic phenotype) should be clearly distinguished.

The most frequently evoked diagnoses outside of the autism spectrum are schizoid personality, obsessive-compulsive personality, and social anxiety. There is disagreement concerning the distinction between the schizophrenic galaxy and AS. Wing recognized the possibility of hallucination in AS but carefully drew a distinction with schizophrenia, based, in particular, on the contrast between “local” AS thought and the “delusional and vague woolliness” of schizophrenic thought. However, she incorporated Wolf’s schizoid personality with AS, although, in our opinion, there are crucial differences. These differences are well captured by the DSM-IV description, distinguishing between AS people, whose positive emotions are specifically triggered by a narrow range of interests, and people with schizoid personality who display a reduced range of positive and negative emotions. It can be difficult to distinguish between the negative symptoms of schizophrenia and the reduction of social interest in AS in the absence of knowledge of the individual’s developmental history. Indeed, AS patients do not present progressive lessening of their cognitive ability, which is also the hallmark of simple-type schizophrenia. Other confusion is possible between AS and various personality disorders, such as avoidant and obsessive-compulsive personality. There is even more confusion in diagnosing girls due to our ignorance of a possibly distinct feminine AS phenotype. TDAH, anxiety, and depression are considered to be frequent comorbidities rather than differentially diagnosed, which they can still be.

The Basis of Social “Impairment” in AS

Wing’s contribution led to the scientific and public recognition of AS in the non-German world, but at the cost of losing some of the signal detected by its discoverer, and increased pathologization. Wing’s ideas had a considerable influence on further cognitive neuroscience for better and for worse. In the 1990s, during the golden age of cognitive models, the

supposedly broken module was the theory of mind and the understanding of implicit rules of social behavior. This was premature, especially in light of numerous studies demonstrating intact subcomponents of social function in the autism spectrum. AS people do not follow social rules and a typical style of conduct, but it does not necessarily mean that they do not *process* them. Their introspection and ability to describe their own psychosocial functioning is usually excellent and not related with their capacity to modify it according to society's demands. This suggests that the social issues of AS people are the product of reactions to their differences by those around them as well as the idiosyncratic ways that thought and action function in a person with AS. It may not result from a *misunderstanding* of social rules, but of atypicalities in relative priorities, as well as in language–action and emotion–perception relationships (for an alternative view see Klin, 2000). Cognitive scientists are now more prudent and do not directly conclude a defect of function based on the reduction or atypicality of a behavior. They also criticize compensation models where the overdevelopment of one function is believed to compensate for an impaired function. An alternative understanding of AS people could focus on their strengths, for example, language expertise, rather than on modeling their negative aspects (Samson et al., 2015).

Developmental Course and Educational Issues

A diagnosis of AS is generally possible only after 5 years of age, when limited to early talkers and clearly distinguishable from that of autistics who become verbal as adults. A frequent error is to mistake children with AS as gifted non-autistic children due to their early speech and grammatically complex sentences, together with sophisticated scientific or historical interests. AS people have a normal distribution on the intelligence curve, but a distinct profile. Even if they often appear to be geniuses, they may or may not be.

The social problems of AS people depend on the nature and intensity of their social demands. Bullying and conflict at school are frequent, with the results ranging from depression to hetero-aggressivity, as in neurotypical kids. School performance is also extremely variable, from being excellent when the methods and domain suit one particular schoolboy to school refusal at the opposite extreme. The level of autonomy given to the child is crucial, as well as the availability of a rational explanation of what school is useful for. Individual variations in temper/personality are more important than a diagnosis of AS, with some children being passive and easy learners, whereas others are strong opponents to irrelevant material. Over-logical thought may enter in conflict with illogical presentation of the content to be learned. Temporary home education may be the necessary solution in some cases.

The effect of AS people on non-autistics, such as irritation due to excess demands or dependence, must be taken into account, but should not justify segregation. Segregation can be prevented by adapting how pedagogical content is transmitted and how the child's performance is assessed. Individualized learning, tolerance for atypical learning strategies, teaching methods to match the person's intelligence, the surveillance of schoolmates, the possibility to withdraw during unstructured situations, and quick referral to a qualified problem solver are required. Adaptation of the school curriculum and the selection of academic domains according to the person's expertise and specialization are not allowed in most countries, but should be applied to the same extent as they are for other learning or motor disabilities (e.g., dyslexia, sensory impairments). School success and the obtention of a diploma are essential as many AS people will pursue university training and need these years to learn how to cope with the non-autistic world.

Employment and Adult Outcome

Despite the triumphalism of press claims, the employment of people diagnosed with AS is still rare (with a good outcome for only 1/4, Howlin, 2003). Unemployment is far more frequent together with a poor family life. Women with AS are less negatively affected than men with AS. This pessimistic figure is influenced by the “hidden cohort” issue. It is possible that more AS people are employed but are undetected because of the large number of individuals predicted by prevalence studies, but not currently identified. Quality-of-life studies as adults, as well as studies on its predictors during childhood, are also lacking. There are, however, indications that self-recognition as a person with AS and their association with other AS people or verbal autism, through self-organized web groups, improves quality of life. Most knowledge on the adult life of AS people should, however, be considered to be provisional at best and is based on poor quality information. Consequently, as for school integration, we have very little knowledge of the social and employment outcome of AS people in a lifelong context, as well as their support and tolerance by others.

Treatment and Support Issues

The concept of treatment is not straightforward when applied to a condition that overlaps with the personality of the individual themselves. It assumes that the AS phenotype should be normalized to satisfy social constraints and improve quality of life. This has been gradually recognized over the last 40 years, to the point that the dominant belief is that AS should not and cannot be cured, but that its comorbidity should. Even this cannot be taken for granted, as a large part of the so-called comorbidity of AS originates from reactions to external constraints. The belief by the scientific community that anxiety and depression are comorbidities of AS overlooks the specific relation that AS people have with conflicts. Modifying reality (which would, for a psychotic person, be criticized as sharing delusions with the patient) is an option in family, school, or employment crises involving a person with AS, when this reality is unbearable. For example, “depression” related to bullying should be primarily treated by policing the bully’s conduct and/or by temporarily withdrawing the bullied child from school, unlike the treatment of endogenous depression, which may, of course, nevertheless occur.

It was acknowledged that behavioral modification “involved heroic effort and (...) limited results” (Frith, 1991, p. 16). Nonetheless, the conviction dominating the 1980s was that AS people do not understand other people’s intentions and emotions and should learn them, leading to the development of social skills training groups. Their purpose was somewhere between training a supposedly missing function and providing help for crisis situations. The results were disappointing due to disinterest or overapplication of the learned rules. Moreover, social skills training is centered on the demands of people other than the patient himself, diminishing their interest for the person with AS. Focusing instead on differences between them and the world around them, and how to pursue their own interests, while avoiding conflict with the outside world, may restore self-esteem, while maintaining motivation for support.

Individual psychological support is recommended, but its purpose is yet to be determined. The necessity to abandon the psychodynamic framework threw psychologists in ready-made cognitive therapy, which AS people may find naïve and trivial. We suggest that “skills” learning should be replaced by a mechanistic learning of how the social world works. Two messages have to be transmitted: other people do not function like they do, and non-autistic people are

stronger and more numerous. AS people may benefit from establishing compromises between their own needs and modes of functioning and those of others, as well as from opportunistic problem solving strategies (half psychological support, half intervention *in situ*). It is beneficial for them to have a resource person, in whom they have gained confidence, to solve administrative, financial, or employment imbroglios in which they are trapped. Choosing the right treatment targets is essential, as for autism, and the basic model of global assessment, detecting deficits, and providing training is not valid. For example, motor clumsiness is not a suitable target of intervention when it does not interfere with the person's interests.

Conclusion

AS history reflects an interaction between scientific, historical, and personal issues. It spans from its discovery on a clinical basis, its merging with autism, and its transient status under an ill-conceived American and psychiatric centered definition to its official disappearance under questionable scientific principles and has led to its current semi-clandestine existence. Confronting the shifting foundations of modern psychiatry, it exemplifies how uncertain, arbitrary, and unstable the delineation of a psychiatric category is when it does not coincide with the discovery of its neurobiological mechanisms. Aside from its historical journey, AS also confronts important aspects of human neuroscience and psychology, the ongoing evolution of human rights, and the biological and ethical nature of human society. The questions raised by a diagnosis of AS, as well as those by AS people themselves, have participated in the progress of all of these domains. While it is justified to question the nature of its boundaries, the diagnosis of AS remains essential for the virtual community of people who recognize themselves to be in it. Their confidence in having a place among human society is anchored in the name other humans give to them.

Author Biography

Laurent Mottron is a psychiatrist and PhD, researcher, and professor in the Department of Psychiatry at the Université de Montréal. He holds the Marcelle and Rolande Gosselin research chair on cognitive neuroscience in autism and has signed some hundred scientific articles about visual and auditory perception in savant and non-savant autism, investigated by brain imaging and cognitive tasks. Montréal group's Enhanced Perceptual Functioning model is one of the leading theories for interpreting cognitive and fMRI data in autism.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Barbeau, E. B., Meilleur, A. A., Zeffiro, T. A., & Mottron, L. (2015). Comparing motor skills in autism spectrum individuals with and without speech delay. *Autism Research*, 8(6), 682–693. doi:10.1002/aur.1483
- Barbeau, E. B., Soulières, I., Dawson, M., Zeffiro, T. A., & Mottron, L. (2013). The level and nature of autistic intelligence III: Inspection time. *Journal of Abnormal Psychology*, 122(1), 295–301. doi:10.1037/a0029984
- Czech, H. (2018). Hans Asperger, national socialism, and “race hygiene” in Nazi-era Vienna. *Molecular Autism*. doi:10.1186/s13229-018-0208-6

- Frith, U. (Ed.) (1991). *Autism and Asperger syndrome*. Cambridge, UK; New York, NY: Cambridge University Press.
- Gillberg, I. C., & Gillberg, C. (1989). Asperger syndrome—Some epidemiological considerations: A research note. *Journal of Child Psychology and Psychiatry*, 30(4), 631–638. doi:0.1111/j.1469-7610.1989.tb00275.x
- Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: Implications for the differentiation between autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 33(1), 3–13. doi:10.1023/A:1022270118899
- Kenworthy, L., Wallace, G. L., Powell, K., Anselmo, C., Martin, A., & Black, D. O. (2012). Early language milestones predict later language, but not autism symptoms in higher functioning children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6(3), 1194–1202. doi:10.1016/j.rasd.2012.03.009
- Klin, A. (2000). Attributing social meaning to ambiguous visual stimuli in higher-functioning autism and Asperger syndrome: The social attribution task. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, 41(7), 831–846.
- Lai, M. C., Lombardo, M. V., Chakrabarti, B., & Baron-Cohen, S. (2013). Subgrouping the autism “spectrum”: Reflections on DSM-5. *PLoS Biology*, 11(4), e1001544. doi:10.1371/journal.pbio.1001544
- Mayo, J., Chlebowski, C., Fein, D. A., & Eigsti, I. M. (2013). Age of first words predicts cognitive ability and adaptive skills in children with ASD. *Journal of Autism and Developmental Disorders*, 43(2), 253–264. doi:10.1007/s10803-012-1558-0
- Sahyoun, C. P., Soulieres, I., Belliveau, J. W., Mottron, L., & Mody, M. (2009). Cognitive differences in pictorial reasoning between high-functioning autism and Asperger’s syndrome. *Journal of Autism and Developmental Disorders*, 39(7), 1014–1023. doi:10.1007/s10803-009-0712-9
- Samson, F., Zeffiro, T. A., Doyon, J., Benali, H., & Mottron, L. (2015). Speech acquisition predicts regions of enhanced cortical response to auditory stimulation in autism spectrum individuals. *Journal of Psychiatric Research*, 68, 285–292. doi:10.1016/j.jpsychires.2015.05.011
- Szatmari, P. (1992). The validity of autistic spectrum disorders: A literature review. *Journal of Autism and Developmental Disorders*, 22(4), 583–600. doi:10.1007/BF01046329
- Szatmari, P., Bartolucci, G., & Bremner, R. (1989). Asperger’s syndrome and autism: Comparison of early history and outcome. *Developmental Medicine and Child Neurology*, 31(6), 709–720. doi:10.1111/j.1469-8749.1989.tb04066.x
- Takarae, Y., Luna, B., Minshew, N. J., & Sweeney, J. A. (2008). Patterns of visual sensory and sensorimotor abnormalities in autism vary in relation to history of early language delay. *Journal of the International Neuropsychological Society*, 14(6), 980–989. doi:10.1017/S1355617708081277
- Wing, L. (1981). Asperger’s syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–129. doi:10.1017/S0033291700053332

Neurobiology of Down Syndrome

Stella Sakhon and Jamie Edgin

Department of Psychology, The University of Arizona, Tucson, AZ, USA

Down syndrome (DS/trisomy 21) is the most common genetic cause of intellectual disability, occurring in 1/700 live births (Parker et al., 2010). The syndrome involves the triplication of over 300 genes and is associated with characteristic facial features, delays in physical growth, and impairments in intellectual and adaptive function (Epstein et al., 1991). The cognitive and behavioral phenotype is well characterized (Dierssen, 2012; Edgin, 2013; Fernandez & Reeves, 2015; Grieco, Pulsifer, Seligsohn, Skotko, & Schwartz, 2015), but more information is required about the trajectories of cognitive and neural development in the syndrome and the bases for individual differences, which are extensive (Karmiloff-Smith et al., 2016).

The study of DS is associated with some notable “firsts” in biological science. In 1959, it was the first chromosomal abnormality to be discovered (Lejeune, Gautier, & Turpin, 1959), just 4 years after Tjio discovered the total number of human chromosomes (Tjio & Levan, 1956). This discovery paved the way for other notable accomplishments in medical genetics (Patterson & Costa, 2005). DS is also among the first intellectual disability syndromes to have representative animal models (the Ts65Dn mouse model, Davisson et al., 1992), allowing for substantial advances in the understanding of the neurobiology of the condition as well as the development of therapeutic treatments (Bartesaghi et al., 2015; Fernandez et al., 2007). Further, neural and cognitive development across the lifespan in DS may be of great interest to the broader scientific community. People with DS advance through the developmental period while bearing significant β -amyloid burden linked to the triplication of the amyloid precursor protein (APP) gene and harboring certain medical comorbidities (i.e., including sleep disturbance). In fact, DS stands as the condition with the greatest risk for the early onset of Alzheimer’s disease (AD), demonstrating risk of the disease decades in advance of the general population (Wiseman et al., 2015). DS is currently regarded as a population with significant potential for successful pharmacological and behavioral treatments, both in the infant period and in aging adults who are susceptible to AD-related decline (Bartesaghi et al., 2015; Rafii et al., 2015). Findings from studies examining sleep disorders in this group suggest additional avenues of treatment, based on remediation of sleep deficits (Breslin et al., 2014;

Edgin et al., 2015). We will now overview the wealth of findings on the neurobiology of DS to date, including our current understanding of brain development and aging in humans and animal models of the condition.

Brain Structure Across Development in Down Syndrome

There are rich sets of structural brain data from individuals with DS, primarily collected postpartum. Less *in vivo* neuroimaging data exist with DS, and most have focused on the adult period in reference to AD cognitive-related decline. It is known that brain alterations start during the fetal period (by the second trimester) and progress through adulthood (Ábrahám et al., 2012; Guidi et al., 2008; Wisniewski, 1990). During prenatal development (17–21 weeks of gestation), individuals with DS show altered neurogenesis and reduced cell numbers in the hippocampus and parahippocampal cortex, including the presence of apoptotic cell death (Guidi et al., 2008).

Differences in the DS brain become more pronounced across the first year of life (Ábrahám et al., 2012). One study examining brain development in 101 individuals with DS from birth to age 5 years found delays in myelination and proposed that delays could alter the function of the hippocampal circuitry. Although at birth, the brain shape in infants with DS did not differ from non-DS infants, around 3–5 months of age, the anterior–posterior diameter was shorter, the frontal lobes were reduced, the occipital poles were flatter, and the brain stem and cerebellum were smaller in DS infants compared with non-DS infants (Wisniewski, 1990). Wisniewski (1990) also reported decreases in neurons of layers II and IV and myelination delay in 22.5% DS and only 6.8% non-DS children.

Structural neuroimaging studies have also confirmed structural brain abnormalities in DS, including reduced global brain volume (Lee et al., 2015; Pinter, Eliez, Schmitt, Capone, & Reiss, 2001; White, Alkire, & Haier, 2003). A voxel-based morphometric study found reductions in gray matter (GM) volume, with significant decreases in the CA2/CA3 region of the left hippocampus (White et al., 2003). A structural neuroimaging study by Lee et al. (2015) found a 7% reduction, specifically, in cortical GM volume (frontal, temporal, and occipital) in individuals with DS. They found a 12% reduction of cortical surface area (SA) but a 4% reduction of cortical thickness (CT) in DS, suggesting that reduced cortical volume may be driven by cortical SA rather than CT. Another study using automatic measurements of CT from structural MRI found a negative correlation between CT and age in the bilateral frontal, temporal, and parietal lobes and cingulate gyrus in nondemented individuals with DS (Romano et al., 2016), suggesting brain regions associated with age-related decline are impacted before the onset of any symptoms of dementia.

Several studies have also suggested major alterations in white matter (WM) development. Using diffusion tensor imaging (DTI), researchers found adults with DS had significantly reduced WM pathways in the frontal lobes compared with controls (Powell et al., 2014). One study examining the differences in myelination in the hippocampus, in fetuses and patients with DS from midgestation to adulthood, showed that those with DS differed in the number and density of myelinated fibers compared with age-matched controls. The difference in fiber density was subtle in the first few months after birth but became more evident across age (Ábrahám et al., 2012). Recent work has shown that genes regulating oligodendrocyte differentiation and myelination display altered expression in DS, suggesting that poor myelination may contribute to slow neural transmission and perhaps even underdevelopment of some later-developing brain structures (Edgin, 2013; Olmos-Serrano, Kang, et al., 2016).

In addition to structural brain abnormalities, individuals with DS show aberrations in functional connectivity. A study testing resting-state functional connectivity showed significantly greater between-network connectivity in individuals with DS as compared with a typically developing (TD) group (Vega, Hohman, Pryweller, Dykens, & Thornton-Wells, 2015). Another study examined neural activation between individuals with DS and a TD group when listening to a short story. Significantly greater activation in midline regions of the frontal lobe and cingulate cortex in DS suggested altered functional neural organization during language processing compared with TD individuals (Jacola et al., 2014).

In comparison to the general population, individuals with DS have a higher risk of developing AD and present AD neuropathology at much younger ages (Malamud, 1972). Almost all individuals with DS over 35–40 years of age develop β -amyloid ($A\beta$) plaques and neurofibrillary tangles (characteristic of AD neuropathology), evidenced by autopsied patient data and PET imaging with β -amyloid markers (Zigman & Lott, 2007). Recent work seeks to address whether the presence of amyloid deposition or associated biomarkers could predict AD neuropathology before the development of dementia (Handen et al., 2012; Head et al., 2011; Rafii et al., 2015; Teipel & Hampel, 2006). Head et al. (2011) suggested that levels of plasma $A\beta$ could help predict variation in cognitive function in DS with or without dementia; however, group differences in plasma $A\beta$ did not predict early signs of AD neuropathology in DS. A recent study by Rafii et al. (2015) sought to identify AD biomarkers. They successfully collected volumetric MRI, amyloid positron emission tomography (PET), fluorodeoxyglucose (FDG) PET, and plasma $A\beta$ data that are consistent with preclinical AD in DS. While evidence is accumulating to suggest links between sleep disturbance and the development of AD in the general population, there is little extant work relating these risk factors in DS, and some have hypothesized poor sleep to be an important predictor of this group's progression to dementia (Fernandez & Edgin, 2013).

Mouse models of DS (e.g., Ts65Dn and Tcl) have also provided insight to the neurobiology of DS and numerous potential pharmacological therapies (Bartesaghi et al., 2015). Recent work has further refined the profile of hippocampal dysfunction in these models, a profile consistently demonstrated from decades of work (Reeves et al., 1995). Specifically, the Tcl mouse model displays reduced entorhinal cortex input to the dentate gyrus (DG) and abnormal DG–CA3 connectivity, coupled with impaired information processing in CA3 and CA1 networks (Witton et al., 2015). A recent longitudinal behavioral study from birth to adulthood in the Ts65Dn mouse model found reversal learning impairments across the lifespan and evidence for an age-dependent decline, findings suggesting that early behavioral markers may be predictive of later cognitive status (Olmos-Serrano, Tyler, Cabral, & Haydar, 2016). More information is needed regarding the longitudinal profiles of specific cognitive and neural deficits from animal models, such as the data gathered from the elegant lifespan approach employed by Haydar and colleagues. These data would help to guide early interventions for improving cognitive function in humans with DS.

Also important to understanding the neurobiology of DS are approaches that incorporate the influence of health and environment on these brain and cognitive phenotypes. DS is not a homogeneous condition, but is characterized by extensive variation in developmental trajectories and cognitive end points that may reflect individual differences in health, environmental resources, or background genetics. For instance, individual differences in sleep among people with DS, including diagnosed obstructive sleep apnea (present in approximately 50%), significantly correlate with variation in verbal IQ and executive function across a wide range of ages (Breslin et al., 2014). Other studies have suggested that the behavioral profile of DS may also relate to background genetics in addition to chromosome 21 variation, including risk alleles

for attention deficit hyperactivity disorder (ADHD; Mason, Spano, & Edgin, 2015). Further, individuals with DS are embedded within a social environment, and some groups have shown a better response to intervention in the context of environmental enrichment (Catuara-Solarz et al., 2015). We believe that the study of DS offers a wealth of information to inform how sleep may impact learning (Edgin et al., 2015) and clues to the development of AD. However, a careful examination of these links requires the field to fully recognize the multisystem complexity and profiles of individual differences associated with trisomy 21.

Future Directions and Conclusion

Work already conducted in humans and animal models of DS provides us with a wealth of information regarding brain development in a population at great risk to develop both AD and sleep disturbance. Open questions include when may individuals with DS suffer from the impact of AD progression and what factors, including variability in health, genetics, and environment, may predict those with different developmental trajectories. Future studies that provide more information about the developmental trajectories in DS may influence pharmacological and behavioral treatments.

Author Biographies

Stella Sakhon is a graduate student in Cognition and Neural Systems at the University of Arizona. Stella is investigating alternative language learning mechanisms in individuals with Down syndrome. The results of her work could have applications for designing intervention programs for language learning deficits in Down syndrome and will provide additional information about the influence of attention and sleep on word learning. Ms. Sakhon is a 2016 Galileo scholar in the University of Arizona, College of Science.

Jamie Edgin, PhD, is an assistant professor of Cognition and Neural Systems program at the University of Arizona. He is an assistant professor and the director of the Memory Development and Disorders Laboratory in the Department of Psychology at the University of Arizona. Her research centers on typical and atypical memory development and sleep. Currently she is creating a new computerized battery for the assessment of memory in preschoolers and special populations. She has received awards for her work on Down syndrome, including the Inaugural David Cox Rising Star Award for Down Syndrome Research from the LuMind Research Down Syndrome Foundation and the Charles Epstein Award. She is a fellow of the Psychonomics Society and holds grants from the LuMind Research Down Syndrome Foundation, the NIH, and the Bill and Melinda Gates Foundation.

References

- Ábrahám, H., Vincze, A., Veszprémi, B., Kravják, A., Gömöri, É., Kovács, G. G., & Seress, L. (2012). Impaired myelination of the human hippocampal formation in Down syndrome. *International Journal of Developmental Neuroscience*, 30(2), 147–158. doi:10.1016/j.ijdevneu.2011.11.005
- Bartasaghi, R., Haydar, T. F., Delabar, J. M., Dierssen, M., Martínez-Cué, C., & Bianchi, D. W. (2015). New perspectives for the rescue of cognitive disability in Down syndrome. *The Journal of Neuroscience*, 35(41), 13843–13852. doi:10.1523/JNEUROSCI.2775-15.2015

- Breslin, J., Spanò, G., Bootzin, R., Anand, P., Nadel, L., & Edgin, J. (2014). Obstructive sleep apnea syndrome and cognition in Down syndrome. *Developmental Medicine and Child Neurology*, 56(7), 657–664. doi:10.1111/dmcn.12376
- Catuara-Solarz, S., Espinosa-Carrasco, J., Erb, I., Langohr, K., Notredame, C., Gonzalez, J. R., & Dierssen, M. (2015). Principal component analysis of the effects of environmental enrichment and (–)-epigallocatechin-3-gallate on age-associated learning deficits in a mouse model of Down syndrome. *Frontiers in Behavioral Neuroscience*, 9, 330. doi:10.3389/fnbeh.2015.00330
- Davisson, M. T., Schmidt, C., Reeves, R. H., Irving, N. G., Akeson, E. C., Harris, B. S., & Bronson, R. T. (1992). Segmental trisomy as a mouse model for Down syndrome. *Progress in Clinical and Biological Research*, 384, 117–133.
- Dierssen, M. (2012). Down syndrome: The brain in trisomic mode. *Nature Reviews Neuroscience*, 13(12), 844–858. doi:10.1038/nrn3314
- Edgin, J. O. (2013). Cognition in Down syndrome: A developmental cognitive neuroscience perspective. *WIREs Cognitive Science*, 4, 307–317. doi:10.1002/wcs.1221
- Edgin, J. O., Tooley, U., Demara, B., Nyhuis, C., Anand, P., & Spanò, G. (2015). Sleep disturbance and expressive language development in preschool-age children with Down syndrome. *Child Development*, 86(6), 1984–1998. doi:10.1111/cdev.12443
- Epstein, C. J., Korenberg, J. R., Annerén, G., Antonarakis, S. E., Aymé, S., Courchesne, E., ... Huret, J. L. (1991). Protocols to establish genotype-phenotype correlations in Down syndrome. *American Journal of Human Genetics*, 49(1), 207.
- Fernandez, F., & Edgin, J. O. (2013). Poor sleep as a precursor to cognitive decline in Down syndrome: A hypothesis. *Journal of Alzheimer's Disease & Parkinsonism*, 3(2), 124.
- Fernandez, F., Morishita, W., Zuniga, E., Nguyen, J., Blank, M., Malenka, R. C., & Garner, C. C. (2007). Pharmacotherapy for cognitive impairment in a mouse model of Down syndrome. *Nature Neuroscience*, 10(4), 411–413. doi:10.1038/nn1860
- Fernandez, F., & Reeves, R. H. (2015). Assessing cognitive improvement in people with Down syndrome: Important considerations for drug-efficacy trials. *Handbook of Experimental Pharmacology*, 228, 335–380. doi:10.1007/978-3-319-16522-6
- Grieco, J., Pulsifer, M., Seligsohn, K., Skotko, B., & Schwartz, A. (2015). Down syndrome: Cognitive and behavioral functioning across the lifespan. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, 169(2), 135–149. doi:10.1002/ajmg.c.31439
- Guidi, S., Bonasoni, P., Ceccarelli, C., Santini, D., Gualtieri, F., Ciani, E., & Bartesaghi, R. (2008). Neurogenesis impairment and increased cell death reduce total neuron number in the hippocampal region of fetuses with Down syndrome. *Brain Pathology*, 18(2), 180–197. doi:10.1111/j.1750-3639.2007.00113.x
- Handen, B. L., Cohen, A. D., Channamalappa, U., Bulova, P., Cannon, S. A., Cohen, W. I., ... Klunk, W. E. (2012). Imaging brain amyloid in nondemented young adults with Down syndrome using Pittsburgh compound B. *Alzheimer's & Dementia*, 8(6), 496–501. doi:10.1016/j.jalz.2011.09.229
- Head, E., Doran, E., Nistor, M., Hill, M., Schmitt, F. A., Haier, R. J., & Lott, I. T. (2011). Plasma amyloid- β as a function of age, level of intellectual disability, and presence of dementia in Down syndrome. *Journal of Alzheimer's Disease*, 23(3), 399–409. doi:10.3233/JAD-2010-101335
- Jacola, L. M., Byars, A. W., Hickey, F., Vannest, J., Holland, S. K., & Schapiro, M. B. (2014). Functional magnetic resonance imaging of story listening in adolescents and young adults with Down syndrome: Evidence for atypical neurodevelopment. *Journal of Intellectual Disability Research*, 58(10), 892–902. doi:10.1111/jir.12089
- Karmiloff-Smith, A., Al-Janabi, T., D'Souza, H., Groet, J., Massand, E., Mok, K., ... Strydom, A. (2016). The importance of understanding individual differences in Down syndrome. *F1000Research*, 5. doi:10.12688/f1000research.7506.1
- Lee, N. R., Adeyemi, E. I., Lin, A., Clasen, L. S., Lalonde, F. M., Condon, E., ... Giedd, J. N. (2015). Dissociations in cortical morphometry in youth with Down syndrome: Evidence for reduced surface area but increased thickness. *Cerebral Cortex*, bhv107. doi:10.1093/cercor/bhv107

- Lejeune, J., Gautier, M., & Turpin, R. (1959). Study of somatic chromosomes from 9 mongoloid children. *Comptes Rendus Hebdomadaires des Séances de l'Académie des Sciences*, 248(11), 1721.
- Malamud, N. (1972). Neuropathology of organic brain syndromes associated with aging. In C. M. Gaitz (Ed.), *Aging and the brain. Advances in behavioral biology* (Vol. 3, pp. 63–87). Boston, MA: Springer. doi:10.1007/978-1-4684-8503-5_6
- Mason, G. M., Spanó, G., & Edgin, J. (2015). Symptoms of attention-deficit/hyperactivity disorder in Down syndrome: Effects of the dopamine receptor D4 gene. *American Journal on Intellectual Developmental Disabilities*, 120(1), 58–71. doi:10.1352/1944-7558-120.1.58
- Olmos-Serrano, J. L., Kang, H. J., Tyler, W. A., Silbereis, J. C., Cheng, F., Zhu, Y., ... Goodliffe, J. (2016). Down syndrome developmental brain transcriptome reveals defective oligodendrocyte differentiation and myelination. *Neuron*. doi:10.1016/j.neuron.2016.01.042
- Olmos-Serrano, J. L., Tyler, W. A., Cabral, H. J., & Haydar, T. F. (2016). Longitudinal measures of cognition in the Ts65Dn mouse: Refining windows and defining modalities for therapeutic intervention in Down syndrome. *Experimental Neurology*, 279, 40–56. doi:10.1016/j.expneurol.2016.02.005
- Parker, S. E., Mai, C. T., Canfield, M. A., Rickard, R., Wang, Y., Meyer, R. E., ... Correa, A. (2010). Updated national birth prevalence estimates for selected birth defects in the United States, 2004–2006. *Birth Defects Research Part A: Clinical and Molecular Teratology*, 88(12), 1008–1016. doi:10.1002/bdra.20735
- Patterson, D., & Costa, A. C. (2005). Down syndrome and genetics—A case of linked histories. *Nature Reviews Genetics*, 6(2), 137–147. doi:10.1038/nrg1525
- Pinter, J. D., Eliez, S., Schmitt, J. E., Capone, G. T., & Reiss, A. L. (2001). Neuroanatomy of Down's syndrome: A high-resolution MRI study. *American Journal of Psychiatry*, 158(10), 1659–1665. doi:10.1176/appi.ajp.158.10.1659
- Powell, D., Caban-Holt, A., Jicha, G., Robertson, W., Davis, R., Gold, B. T., ... Head, E. (2014). Frontal white matter integrity in adults with Down syndrome with and without dementia. *Neurobiology of Aging*, 35(7), 1562–1569. doi:10.1016/j.neurobiolaging.2014.01.137
- Rafii, M. S., Wishnek, H., Brewer, J. B., Donohue, M. C., Ness, S., Mobley, W. C., ... Rissman, R. A. (2015). The Down syndrome biomarker initiative (DSBI) pilot: Proof of concept for deep phenotyping of Alzheimer's disease biomarkers in Down syndrome. *Frontiers in Behavioral Neuroscience*, 9. doi:10.3389/fnbeh.2015.00239
- Reeves, R. H., Irving, N. G., Moran, T. H., Wohn, A., Kitt, C., Sisodia, S. S., ... Davisson, M. T. (1995). A mouse model for Down syndrome exhibits learning and behaviour deficits. *Nature Genetics*, 11, 177–184.
- Romano, A., Cornia, R., Moraschi, M., Bozzao, A., Chiacchiararelli, L., Coppola, V., ... Pierallini, A. (2016). Age-related cortical thickness reduction in non-demented Down's syndrome subjects. *Journal of Neuroimaging*, 26(1), 95–102. doi:10.1111/N/jon.12259
- Teipel, S. J., & Hampel, H. (2006). Neuroanatomy of Down syndrome in vivo: A model of preclinical Alzheimer's disease. *Behavior Genetics*, 36(3), 405–415. doi:10.1007/s10519-006-9047-x
- Tjio, J. H., & Levan, A. (1956). The chromosome number of man. *Hereditas*, 42(1–2), 1–6. doi:10.1111/j.1601-5223.1956.tb03010.x
- Vega, J. N., Hohman, T. J., Pryweller, J. R., Dykens, E. M., & Thornton-Wells, T. A. (2015). Resting-state functional connectivity in individuals with Down syndrome and Williams syndrome compared with typically developing controls. *Brain Connectivity*, 5(8), 461–475. doi:10.1089/brain.2014.0266
- White, N. S., Alkire, M. T., & Haier, R. J. (2003). A voxel-based morphometric study of nondemented adults with Down syndrome. *NeuroImage*, 20(1), 393–403. doi:10.1016/S1053-8119(03)00273-8
- Wiseman, F. K., Al-Janabi, T., Hardy, J., Karmiloff-Smith, A., Nizetic, D., Tybulewicz, V. L., ... Strydom, A. (2015). A genetic cause of Alzheimer disease: Mechanistic insights from Down syndrome. *Nature Reviews. Neuroscience*. doi:10.1038/nrn3983

- Wisniewski, K. E. (1990). Down syndrome children often have brain with maturation delay, retardation of growth, and cortical dysgenesis. *American Journal of Medical Genetics*, 37(S7), 274–281. doi:10.1002/ajmg.1320370755
- Witton, J., Padmashri, R., Zinyuk, L. E., Popov, V. I., Kraev, I., Line, S. J., ... Fisher, E. M. (2015). Hippocampal circuit dysfunction in the Tcl mouse model of Down syndrome. *Nature Neuroscience*, 18(9), 1291–1298. doi:10.1038/nn.4072
- Zigman, W. B., & Lott, I. T. (2007). Alzheimer's disease in Down syndrome: Neurobiology and risk. *Mental Retardation and Developmental Disabilities Research Reviews*, 13(3), 237–246. doi:10.1002/mrdd.20163

Vascular Dementia

Paola García-Egan¹, Jenna Haddock¹, and
Robert H. Paul^{1,2}

¹*Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA*

²*Missouri Institute of Mental Health, St. Louis, MO, USA*

Vascular dementia (VaD) defines impaired cognitive function due to cerebrovascular disease. VaD is most common in older individuals (Middleton, Grinberg, Miller, Kawas, & Yaffe, 2011) and accounts for 15–20% of all forms of dementia (Gorelick et al., 2011). Alzheimer's disease (AD) is the most prevalent etiology of age-related dementia; however a combination of VaD and AD (i.e., mixed dementia) is the dominant form of neuropathology in postmortem studies of aged individuals with antemortem diagnoses of dementia (Lee, 2011). The differences in prevalence observed in clinic versus autopsy have led some groups to suggest that cerebrovascular disease is a primary driver of AD-related pathology, though the position is not universally endorsed.

In addition to advanced age, VaD risk factors include low educational attainment, family history of dementia, diabetes, cardiovascular disease, and lipid abnormalities (Gorelick et al., 2011). The impact of gender as a risk factor remains unclear since the historic literature prioritized outcomes in males (Desmond et al., 2000; Korczyn, Vakhapova, & Grinberg, 2012; Lee, 2011). By contrast, racial/ethnic differences are more well defined. Compared with Caucasians, African Americans and Asians have higher risks of VaD, with a 50% increase in risk reported in some studies (Fitzpatrick et al., 2004).

Subtypes of Vascular Dementia

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is the dominant genetic subtype. CADASIL is related to a mutation on the Notch-3 region of chromosome 19 (Desmond, 2004). First symptoms appear between the ages of 20 and 40 and typically include cognitive impairment, migraine with aura, and/or mood disorders. While the young age of onset can lead to misdiagnosis, CADASIL is believed

to account for a minority of VaD cases (Chabriat, Joutel, Dichgans, Tournier-Lasserre, & Bousser, 2009).

Large vessel cerebral infarcts account for approximately 15% of VaD cases and typically reflect an embolic etiology (Korczyn et al., 2012). One-third of older individuals who experience a large cortical stroke meet diagnostic criteria for VaD within 3 months of the infarct (Desmond et al., 2000), particularly when the language network is damaged (Jellinger, 2008). Ischemia can also cause cortical injury when insufficient blood exchange occurs in the “watershed” or border zone regions along the dorsal surface of the cortical mantle (Korczyn et al., 2012).

Small vessel ischemic disease results from the occlusion of penetrating arteries that provide blood to the deep structures and white matter pathways (Desmond, 2004), which are evidenced on magnetic resonance imaging as white matter hyperintensities (WMH). These vascular injuries are common in individuals over the age of 65 and correlate with the degree of cognitive impairment. Increased burden of WMH correlates with cardiovascular disease (Korczyn et al., 2012) and is believed to involve microangiopathy, venous collagenosis, and Wallerian degeneration (Korczyn et al., 2012). Significant ischemic injury in subcortical brain regions perfused by the lenticulostriate arteries is sufficient to result in dementia. Unfortunately, many individuals with subcortical ischemia are prone to additional cortical and subcortical infarcts due to the underlying cardiovascular disease (Jellinger, 2013; Korczyn et al., 2012; Lee, 2011).

Symptoms of Vascular Dementia

Symptoms may appear gradually over time, or suddenly after a first cortical or strategically located subcortical stroke. The typical VaD cognitive phenotype includes prominent executive dysfunction characterized by abnormalities in divided attention, planning, multitasking, sequencing, etc. (Sachdev et al., 2014). However, other symptoms may be present depending on the location and amount of brain tissue involved, including classic stroke syndromes (e.g., neglect, prosopagnosia, aphasia, etc.). Physical symptoms include motor abnormalities and gait disturbance that mimic other etiologies (e.g., Parkinson’s disease, normal pressure hydrocephalus) that complicate the differential diagnosis.

Treatment of Vascular Dementia

Clinical intervention for VaD includes two parallel paths. The first is to treat the underlying mechanisms, which typically includes a focus on the cardiovascular system. Aspirin, medications (e.g., antihypertensives), exercise, smoking cessation, and weight management can reduce the risk of additional cerebrovascular events. The second line of treatment targets brain mechanisms using medications originally developed for AD. Cholinesterase inhibitors provide modest therapeutic benefit in terms of cognitive symptoms and apathy (Erkinjuntti et al., 2002; Wilkinson et al., 2010). By contrast, glutamate receptor antagonism is not effective (McShane, Areosa Sastre, & Minakaran, 2006).

Future Directions

For the majority of cases caused by ischemic injury, VaD can be difficult to differentiate from other etiologies with high base rates in the elderly population (Gorelick et al., 2011; Jellinger, 2013). This is particularly true when symptom onset is slow and insidious as in the majority of

subcortical ischemic disease (Lee, 2011). Further, the near ubiquitous nature of WMH on neuroimaging among older individuals requires a determination if the vascular burden exceeds expectations for age. Refining the diagnostic process represents a critical future direction. Novel neuroimaging methods using diffusion-based MR sequences and multimodal approaches offer enhanced opportunities to improve diagnostic accuracy (Attems & Jellinger, 2014). Further, non-pharmaceutical interventions, such as direct current magnetic stimulation, are needed to improve cognitive and psychiatric symptoms associated with vascular injury. Direct current stimulation is an exciting therapeutic modality to support compensatory recruitment of healthy (non-infarcted) brain tissue.

Author Biographies

Paola García-Egan is a PhD candidate at the University of Missouri–St. Louis. She has published articles on standardization of neuropsychological tests for Latin American population and parental ethnotheories and book chapters on vascular dementia and myasthenia gravis. Her current research includes early life stress and aging.

Jenna Haddock is a research assistant in the Neuropsychology and Applied Neuroimaging Laboratory (NAN), directed by Dr. Robert Paul.

Dr. Robert H. Paul is director of the Missouri Institute of Mental Health and professor of psychological sciences, University of Missouri–St. Louis. His research program is focused on delineating the underlying mechanisms of cognitive phenotypes in adult and pediatric neurologic disease.

References

- Attems, J., & Jellinger, K. A. (2014). The overlap between vascular disease and Alzheimer's disease—Lessons from pathology. *BMC Medicine*, 12, 206. doi:10.1186/s12916-014-0206-2
- Chabriat, H., Joutel, A., Dichgans, M., Tournier-Lasserre, E., & Boussier, M. G. (2009). Cadasil. *The Lancet Neurology*, 8(7), 643–653. doi:10.1016/S1474-4422(09)70127-9
- Desmond, D. W. (2004). Vascular dementia. *Clinical Neuroscience Research*, 3(6), 437–448.
- Desmond, D. W., Moroney, J. T., Paik, M. C., Sano, M., Mohr, J. P., Aboumatar, S., ... Hauser, W. A. (2000). Frequency and clinical determinants of dementia after ischemic stroke. *Neurology*, 54(5), 1124–1131.
- Erkinjuntti, T., Kurz, A., Gauthier, S., Bullock, R., Lillienfeld, S., & Damaraju, C. V. (2002). Efficacy of galantamine in probable vascular dementia and Alzheimer's disease combined with cerebrovascular disease: A randomized clinical trial. *Lancet*, 359, 1283–1290.
- Fitzpatrick, A. L., Kuller, L. H., Ives, D. G., Lopez, O. L., Jagust, W., Breitner, J., ... Dulberg, C. (2004). Incidence and prevalence of dementia in the Cardiovascular Health Study. *Journal of the American Geriatrics Society*, 52(2), 195–204.
- Gorelick, P. B., Scuteri, A., Black, S. E., DeCarli, C., Greenberg, S. M., Iadecola, C., ... Petersen, R. C. (2011). Vascular contributions to cognitive impairment and dementia: A statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*, 42(9), 2672–2713. doi:10.1161/STR.0b013e3182299496
- Jellinger, K. A. (2008). Morphologic diagnosis of “vascular dementia”—A critical update. *Journal of the Neurological Sciences*, 270(1), 1–12. doi:10.1016/j.jns.2008.03.006
- Jellinger, K. A. (2013). Pathology and pathogenesis of vascular cognitive impairment—A critical update. *Frontiers in Aging Neuroscience*, 5, 17. doi:10.3389/fnagi.2013.00017
- Korczyn, A. D., Vakhapova, V., & Grinberg, L. T. (2012). Vascular dementia. *Journal of the Neurological Sciences*, 322(1), 2–10. doi:10.1016/j.jns.2012.03.027

- Lee, A. Y. (2011). Vascular dementia. *Chonnam Medical Journal*, 47(2), 66–71. doi:10.4068/cmj.2011.47.2.66
- McShane, R., Areosa Sastre, A., & Minakaran, N. (2006). Memantine for dementia. *The Cochrane Database of Systematic Reviews*, 2, CD003154.
- Middleton, L. E., Grinberg, L. T., Miller, B., Kawas, C., & Yaffe, K. (2011). Neuropathologic features associated with Alzheimer disease diagnosis age matters. *Neurology*, 77(19), 1737–1744. doi:10.1212/WNL.0b013e318236f0cf
- Sachdev, P., Kalaria, R., O'Brien, J., Skoog, I., Alladi, S., Black, S. E., ... Ganguli, M. (2014). Diagnostic criteria for vascular cognitive disorders: A VASCOG statement. *Alzheimer Disease and Associated Disorders*, 28(3), 206. doi:10.1097/WAD.0000000000000034
- Wilkinson, D., Róman, G., Salloway, S., Hecker, J., Boundy, K., Kumar, D., ... Schindler, R. (2010). The long-term efficacy and tolerability of donepezil in patients with vascular dementia. *International Journal of Geriatric Psychiatry*, 25(3), 305–313.

Neurodegenerative Conditions: FTD

Abigail O. Kramer^{1,2}, Andrea G. Alioto^{1,3}, and
Joel H. Kramer²

¹*Department of Psychology, Palo Alto University, Palo Alto, CA, USA*

²*Weill Institute for Neurosciences, University of California, San Francisco Memory and
Aging Center, San Francisco, CA, USA*

³*Department of Neurology, University of California, Davis, CA, USA*

Frontotemporal dementia (FTD) is the collective term for the various clinical presentations resulting from frontotemporal lobar degeneration (FTLD). This pathological process involves progressive neuronal loss primarily in the frontal and temporal lobes. Unlike Alzheimer's disease, FTD is particularly prevalent in individuals aged 65 or younger. The current diagnostic framework identifies three core syndromic diagnoses: one that presents with prominent behavioral changes (behavioral variant) and two that initially present as changes to the understanding and production of language (primary progressive aphasia [PPA]; semantic variant and nonfluent variant). FTLD can also present as predominantly motor syndromes including corticobasal syndrome (CBS), progressive supranuclear palsy (PSP-S), and motor neuron disease (MND). Although individuals tend to meet criteria for one syndrome in the early stages, as the neurodegeneration inevitably spreads, signs and symptoms of two or more syndromes can emerge (Boxer et al., 2012). In addition to the existence of multiple FTD clinical syndromes, the underlying molecular pathology (e.g., different strains of tau or TDP-43 aggregates) is heterogeneous.

Clinical Presentations

Behavioral variant FTD (bvFTD) is the most common clinical FTD syndrome and is marked by severe behavioral and personality changes often in the context of relatively preserved global cognitive functioning (Lanata & Miller, 2016). A diagnosis of possible bvFTD requires the emergence of three out of the following six behavioral/cognitive changes as the first symptom: behavioral disinhibition, apathy or inertia, loss of sympathy or empathy, perseveration or

stereotyped/compulsive/ritualistic behavior, hyperorality, and deficits in executive tasks with relative sparing in episodic memory and visuospatial skills (Rascovsky et al., 2011). Behavioral disinhibition may be described as inappropriate and impulsive reactions manifesting as disregard for social conventions that are out of character for the individual. Examples may include cursing, telling offensive jokes, new onset of substance abuse, urinating in public, or committing criminal behavior. Apathy or inertia is a lack of interest or engagement in activities, exhibiting reduced motivation or concern for what was once important. This may include poor hygiene or disregard for social responsibilities (Lanata & Miller, 2016). Loss of sympathy or empathy is typically detected when the patient demonstrates indifference to the suffering of a loved one or an inability to interpret or process others' emotional experience. Examples of perseveration or stereotyped behavior may include simple, motoric movements such as tapping, humming, or rubbing. Compulsive behaviors may also be more complex such as detailed routines, counting, or verbalizing repetitive phrases. Hyperorality is commonly associated with an increased affinity toward sweet foods to the extent of weight gain.

While the behavioral changes described above are most prominent in bvFTD, cognitive changes can also emerge, especially later in the disease course. These changes include difficulty with multitasking, poor working memory, and mental rigidity (Rascovsky et al., 2011). Since changes in behavior, emotional expression, and personality typically present before the onset of measurable cognitive impairment in middle-aged individuals, bvFTD is often misdiagnosed as a psychiatric disorder rather than a neurodegenerative disease (Lanata & Miller, 2016). This can lead to increased distress due to inappropriate treatment and a delayed proper diagnosis (Woolley, Khan, Murthy, Miller, & Rankin, 2011). Therefore, continued education and awareness of this syndrome across mental health and medical settings is critical to accurately and swiftly diagnose this disorder earlier in the disease process. In fact, it is recommended that middle-to-late life patients with new onset of neuropsychiatric symptoms be considered for a neurodegenerative evaluation (Woolley et al., 2011).

PPA is the other major subset of FTD, which is defined as the insidious onset of communication difficulties as the predominant source of disability in the early stages of the disease process. At the early stages of PPA, communication deficits are isolated, and focal atrophy can be observed on brain imaging. However, as the disease progresses, other cognitive, behavioral, and motor problems can emerge (Gorno-Tempini et al., 2006). PPA is broken down into three subtypes: nonfluent variant (nfvPPA), semantic variant (svPPA), and logopenic variant (lvPPA). The underlying neuropathology of nfvPPA and svPPA subtypes is FTLT, whereas Alzheimer's pathology is most commonly associated with the logopenic subtype. Therefore, the current entry will focus on the first two subtypes.

The nonfluent variant of PPA is characterized by effortful, halting speech with inconsistent speech sound errors and distortions (apraxia of speech) and agrammatism in language production. Brain imaging typically shows atrophy localized to the frontoinsula region at the early stages of the disease (Gorno-Tempini et al., 2011). These impairments are typically in the context of spared single-word comprehension and spared object knowledge (naming and semantics). Apraxia of speech, meaning deficits in planning the articulation of words or inconsistent speech sound errors with distortions, is often the first symptom of this syndrome (Caso et al., 2013). Later in the disease process, more agrammatism emerges. Agrammatism in the production of speech may involve impairments in the accuracy of tense use (i.e., "bought" instead of "bought") or the progressive reduction in use of prepositions, resulting in speech that primarily consists of nouns (Thompson & Mack, 2014). Inability to comprehend grammar may also be present, such as the diminishing ability to understand syntactically complex sentences (i.e., "went to the store after eating lunch"; Charles et al., 2014). These speech

impairments increase throughout the disease process until the affected individual becomes mute (Gorno-Tempini et al., 2006).

Semantic variant (svPPA) is characterized by the loss of semantic knowledge for low-frequency nouns, inability to name objects, and impaired single-word comprehension (Gorno-Tempini et al., 2011). This variant is highly localized to the anterior temporal lobes (ATL), with atrophy typically more severe on the left side in comparison with the right (Mesulam et al., 2014; Wilson et al., 2010). However, individuals that do have pronounced atrophy in the right ATL also commonly struggle with identifying well-known faces and knowing information associated with those people (Kamminga et al., 2015). Patients with svPPA may present with overly general and empty speech and the use of nonspecific words when referring to objects (i.e., “apple” when referring to any fruit). Due to semantic knowledge loss, individuals may also have surface dyslexia, meaning that they lose the ability to read irregular words (e.g., knight, island) due to the loss of meaning associated with the word. Loss of semantic knowledge commonly occurs within the context of preserved repetition and speech production (Gorno-Tempini et al., 2011).

Related Frontotemporal Dementia Syndromes

Other clinical syndromes associated with FTLTD can present with primary changes in motor control and sensorimotor integration, though this presentation is less common among the FTLTD syndromes (Lanata & Miller, 2016). These symptoms can emerge early in the disease course or can accompany one of the behavioral or language syndromes. In fact, approximately 18% of bvFTD cases show movement changes, while these findings are rarely seen in svPPA (Park & Chung, 2013). When motor changes present as the first clinical symptom, PSP-S, CBS, or MND should be considered.

Progressive supranuclear palsy (PSP) is an atypical parkinsonian disorder characterized by abnormal vertical saccades (supranuclear gaze palsy) and postural instability with frequent falls (Litvan et al., 1996). The reason for falls may be multifactorial, including postural instability, impaired eye movements, and impulsivity. Since slowed movement and impaired postural reflexes are also typically present among individuals with PSP, falls can be particularly dangerous since these individuals have difficulty catching or protecting themselves. Cognitive symptoms can also be present with predominant executive dysfunction along with reduced mental speed, attention deficits, and possible personality changes (Donker Kaat et al., 2007).

CBS is also classified as an atypical parkinsonian syndrome. To meet criteria for CBS, individuals must present with an insidious onset and gradual progression of at least two motor symptoms within two separate clusters. The first cluster includes limb rigidity or loss of power for voluntary movement, abnormal muscle tone, and jerky muscle contractions. The second cluster includes inability to perform planned movement, sensory loss, and alien limb syndrome (condition in which limbs move without conscious control; Armstrong et al., 2013). Some CBS patients might also show signs of executive dysfunction, behavioral or personality changes, visuospatial deficits, or nonfluent language changes such as effortful, agrammatic speech.

Several overlapping symptoms within PSP and CBS complicate the diagnostic process. More than 50% of autopsy-confirmed cases of PSP are misdiagnosed as CBS prior to death (Wadia & Lang, 2007). Overlapping presentation includes rapid disease progression, parkinsonism that responds poorly to levodopa treatment, ocular abnormalities, and cognitive symptoms (Litvan et al., 1999). Furthermore, although asymmetry is classically associated with CBS,

asymmetric motor symptoms can occur in both PSP and CBS and are not necessarily more common in CBS (Armstrong et al., 2013).

MND is typically associated with amyotrophic lateral sclerosis (ALS) (Lou Gehrig's disease). To meet criteria for MND, individuals must show signs of neuronal abnormalities in both the upper and lower extremities on physical examination or electromyogram (EMG) (Geevasinga et al., 2016). Typical presentation involves fasciculations, muscle weakness, and difficulty with swallowing (Olney, Spina, & Miller, 2017). While MND can present with any of the core clinical FTD variants, it is most common in individuals with bvFTD. However, the survival rate among those with both MND and bvFTD is lower since individuals with bvFTD are less likely to be compliant with treatment recommendations (Olney et al., 2005).

Epidemiology of FTD

FTD is one of the most common causes of dementia with onset prior to age 65 and might be even more prevalent than AD in those under age 60 (Knopman, Petersen, Edland, Cha, & Rocca 2004). The average age onset of FTD is 56 years old and typically emerges between the ages of 45 and 64. However, cases of younger onset have been cited (Mercy, Hodges, Dawson, Barker, & Brayne, 2008). FTD can occur as early as within the second decade of life with 13% of cases occurring before the age of 50 (Onyike & Diehl-Schmid, 2013). Earlier onset cases more commonly present with svPPA or bvFTD, and later onset typically presents with the nvPPA syndrome (Flanagan et al., 2015). In general, FTD progresses faster than Alzheimer's disease, but the rate of decline in FTD varies based on the syndrome. The svPPA subtype has the longest course, with estimates of approximately 12 years of life on average after diagnosis. The survival rate of nvPPA and bvFTD are similar, ranging from 8 to 9 years on average (Kansal et al., 2016). Nevertheless, a small subset of individuals progress very slowly and may live up to 20 years after diagnosis (Suhonen et al., 2015).

The true prevalence of FTD is difficult to estimate within the general population given that it is easily confused with or confounded by psychiatric disorders and evolving diagnostic criteria. According to the most recent estimates, between 10 and 46 incidences per 100,000 individuals aged 45–64 are currently diagnosed with FTD. This translates to an estimated point prevalence of about 20,000–30,000 individuals in the United States (Knopman & Roberts, 2011). Of those with FTLT, approximately 50–60% present with a bvFTD syndrome, and up to 40% present with a PPA disorder (Johnson et al., 2005; Rohrer et al., 2011). Less than 5% of cases with FTLT manifest as a motor syndrome (i.e., CBS, PSP-S, MND), making these presentations rare. Although few studies report a higher prevalence in males (Johnson et al., 2005), most studies consider FTLT to be equally prevalent among males and females (Hogan et al., 2016; Onyike & Diehl-Schmid, 2013).

Neuropsychological Assessment

Neuropsychological assessment can be a helpful tool to distinguish clinical presentations of FTD. In the early stages, some bvFTD patients show executive functioning deficits, while others may have relatively intact cognition. As the disease progresses, executive dysfunction becomes more evident, while episodic memory, language, and visuospatial functioning remain more intact (Piguet & Hodges, 2013; Rascovsky et al., 2011; Seeley et al., 2008). For instance, they may perform poorly on tasks of working memory, phonemic verbal fluency, set shifting,

and inhibition (Bott, Radke, Stephens, & Kramer, 2014). Inability to monitor errors on testing has also been associated with bvFTD. For instance, bvFTD patients are more likely to make a higher number of repetition and rule violation errors on testing than patients with other neurodegenerative diseases (Carey et al., 2008; Possin et al., 2012). They may also have deficits in emotion recognition (Kumfor & Piguet, 2012) and theory of mind (Gregory et al., 2002).

Since the hallmark feature of svPPA is loss of object knowledge, these patients typically have profound impairment on tasks of confrontation naming without benefit from phonemic or semantic cueing (Gorno-Tempini et al., 2011). They also show impairment in semantic verbal fluency, in which they are asked to generate as many words related to a specific category (i.e., animals, boys' names) as they can within 1 min. This becomes a challenging task for individuals with svPPA due to the loss of conceptual meaning of the category presented. Performance on tasks related to single-word comprehension and reading irregular words is also commonly impaired (Hodges & Patterson, 2007; Mesulam et al., 2009). Episodic memory for nonverbal information (Irish & Piolino, 2016), executive functioning (Marra et al., 2007), and visuospatial abilities (Seelaar, Rohrer, Fox, van Swieten, & Pijnenburg, 2011) are relatively spared.

The hallmark features of agrammatism and effortful, halting speech in nvfPPA are accompanied by neuropsychological deficits primarily related to language and executive functions. These individuals may show slowed word retrieval, commit errors in relation to word retrieval, exhibit an inability to understand complex syntax, and have difficulty repeating words (Leyton et al., 2014; Rohrer et al., 2010). Mild to moderate executive dysfunction may also be present, such as impairment on tasks of phonemic fluency, working memory, set shifting, and inhibition (Gorno-Tempini et al., 2004; Libon et al., 2007). In comparison with svPPA, nvfPPA individuals have spared single-word comprehension and spared object knowledge and therefore are expected to benefit from cueing on confrontational naming tasks, for example (Kertesz, Blair, McMonagle, & Munoz, 2007). These individuals also have relatively spared episodic memory and visuospatial processing early in the disease (Gorno-Tempini et al., 2004; Murray et al., 2007).

Treatment Planning and Safety Considerations

The cognitive, behavioral, and movement changes place individuals with FTD at increased risk for injury and functional decline. Therefore, careful planning and consideration for safety is crucial as the disease progresses. Caregivers of those with bvFTD should be aware of the unique challenges associated with this disease process that differ from Alzheimer's disease and other variants of FTD. For instance, poor judgment, impulsivity, and disinhibition may lead to reckless conduct including overspending or inappropriate social behavior. Environmental controls are needed, including limited access to bank accounts, keys to vehicles, and dangerous medications. Additionally, individuals with FTD benefit from routines and familiar settings (e.g., restaurants where the individual is already known; Manoochehri & Huey, 2012). Hyperorality can be managed through increased supervision during meals to avoid excessive weight gain or dangerous placement of objects in the mouth (Manoochehri & Huey, 2012). Furthermore, removal of dangerous items from the home such as firearms and weapons is important (Merrilees, Klapper, Murphy, Lomen-Hoerth, & Miller, 2010). Clinicians are encouraged to use high-frequency words to enhance comprehension and improve communication (Gorno-Tempini et al., 2011). Additionally, due to agrammatism in speech comprehension in patients with nvfFTD, it is recommended that clinicians use simple grammar, ideally with one verb per sentence, in order to optimize communication. Engaging speech-language

pathologists into the clinical care plan helps to tailor communication styles to accommodate for the patient's deficits. Additionally, family members of individuals diagnosed with FTD benefit from strategies to reduce defensiveness and emotional turmoil related to socially and personally distressing symptoms (Kortte, Stevenson, Hosey, Castillo, & Wegener, 2012).

For individuals experiencing weakness and any changes in movement, clutter on the floor, rugs, furniture, and unnecessary height discrepancies on the ground should be minimized as much as possible to protect against injury (Merrilees et al., 2010; Talerico & Evans, 2001). Lack of insight into the presence and/or severity of cognitive/motor symptoms and the poor impulse control increase fall risks. In severe cases, seatbelts on wheelchairs are needed to limit unsupervised ambulation.

Addressing caregiver burnout is especially important for those caring for a loved one with FTD. Several studies have shown that the stress associated with caring for an individual diagnosed with FTD is greater than those caring for a loved one with Alzheimer's disease (Boutoleau-Bretonnière, Vercelletto, Volteau, Renou, & Lamy, 2008; Mioshi, Bristow, Cook, & Hodges, 2009; Rascovsky et al., 2011). This stress is related to physical demand, disease progression, and depression (Mioshi et al., 2009). Caring for a loved one with bvFTD is particularly challenging due the emotional and personality changes that disrupt the bond and empathy within the relationship. Consistent with this point, empirical studies demonstrate that caring for an individual with bvFTD induces greater distress burden than caring for a loved one with svPPA or nfvPPA (Mioshi et al., 2009, 2013). These caregivers typically experience poorer sleep quality, apathy, and reduced daytime activity (Merrilees et al., 2013). Recommendations to address caregiver burden and improve quality of life include engaging the whole family system in care to relieve one family member of sole responsibility, identifying rewarding aspects of caregiving, reminiscing about positive memories, and seeking outside assistance for skills-based care if the financial means are available (Caceres et al., 2016). Referrals to support groups or individual therapy may also promote healthy coping for caregivers (Mioshi et al., 2013), as well as assist family members to develop realistic expectations of their loved one's cognitive, behavioral, and physical limitations (Caceres et al., 2016).

Author Biographies

Abigail O. Kramer, MS is a psychology doctoral student at Palo Alto University with an emphasis in clinical neuropsychology and geropsychology. She is completing her advanced practicum training at the University of California, San Francisco, Memory and Aging Center, where she evaluates patients with neurodegenerative diseases and conducts research on healthy aging. She is also involved with research in cognition, aging, and mental illness within the Sierra Pacific Mental Illness Research Education and Clinical Center (MIRECC) at the VA Palo Alto Health Care System.

Dr. Andrea G. Alioto, PhD is a postdoctoral fellow in clinical neuropsychology at the University of California, Davis Alzheimer's Disease Center where she provides neuropsychological evaluations for patients with neurodegenerative diseases. She earned her doctorate in clinical psychology at Palo Alto University with an emphasis in neuropsychology and completed her predoctoral internship in the neuropsychology track at the Minneapolis VA Health Care System. In addition to clinical work, Dr. Alioto conducts research examining how lifestyle behaviors are related to cognitive aging and how neuropsychological assessments can improve early detection of dementia syndromes.

Dr. Joel H. Kramer, PsyD is a professor of neuropsychology in neurology and the director of the Memory and Aging Center Neuropsychology program. He earned his doctorate in psychology at Baylor University and completed a postdoctoral fellowship in neuropsychology at the Martinez VA hospital. He is board certified in clinical neuropsychology. Dr. Kramer has been extensively involved in studying the cognitive changes associated with brain disorders for the past three decades. He has co-authored widely used neuropsychological measures of memory and executive functioning. Much of his work has been devoted to identifying the different ways in which aging and neurodegenerative diseases affect memory and other abilities and in utilizing these differences to improve differential diagnosis in clinic.

References

- Armstrong, M. J., Litvan, I., Lang, A. E., Bak, T. H., Bhatia, K. P., Borroni, B., ... Weiner, W. J. (2013). Criteria for the diagnosis of corticobasal degeneration. *Neurology*, 80(5), 496–503. doi:10.1212/WNL.0b013e31827f0fd1
- Bott, N. T., Radke, A., Stephens, M. L., & Kramer, J. H. (2014). Frontotemporal dementia: Diagnosis, deficits and management. *Future Medicine*, 4, 439–454. doi:10.2217/nmt.14.34
- Boutoleau-Brettonnière, C., Vercelletto, M., Volteau, C., Renou, P., & Lamy, E. (2008). Zarit burden inventory and activities of daily living in the behavioral variant of frontotemporal dementia. *Dementia and Geriatric Cognitive Disorders*, 25(3), 272–277. doi:10.1159/000117394
- Boxer, A. L., Barbutt, S., Seeley, W. W., Jafari, A., Heuer, H. W., Mirsky, J., ... Miller, B. L. (2012). Saccade abnormalities in autopsy-confirmed frontotemporal lobar degeneration and Alzheimer's disease. *JAMA Neurology*, 69(4), 509–514. doi:10.1001/archneurol.2011.1021
- Caceres, B. A., Frank, M. O., Jun, J., Martelly, M. T., Sadarangani, T., & de Sales, P. C. (2016). Family caregivers of patients with frontotemporal dementia: An integrative review. *International Journal of Nursing Studies*, 55, 71–84. doi:10.1016/j.ijnurstu.2015.10.016
- Carey, C. L., Woods, S. P., Damon, J., Halabi, C., Dean, D., Delis, D. C., ... Kramer, J. H. (2008). Discriminant validity and neuroanatomical correlates of rule monitoring in frontotemporal dementia and Alzheimer's disease. *Neuropsychologia*, 46, 1081–1087. doi:10.1016/j.neuropsychologia.2007.11.001
- Caso, F., Gesierich, B., Henry, M., Sidhu, M., LaMarre, A., Babiak, M., ... Gorno-Tempini, M. L. Nonfluent/agrammatic PPA with in-vivo cortical amyloidosis and Pick's disease pathology.(2013). *Behavioural Neurology*, 26(1-2), 95–106.
- Charles, D., Olm, C., Powers, J., Ash, S., Irwin, D. J., McMillan, C. T., ... Grossman, M. (2014). Grammatical comprehension deficits in non-fluent/agrammatic primary progressive aphasia. *Journal of Neurology, Neurosurgery and Psychiatry*, 85(3), 249–256.
- Donker Kaat, L., Boon, A. W., Kamphorst, W., Ravid, R., Duivenvoorden, H. J., & van Swieten, J. C. (2007). Frontal presentation in progressive supranuclear palsy. *Neurology*, 69(8), 723–729.
- Flanagan, E. P., Baker, M. C., Perkerson, R. B., Duffy, J. R., Strand, E. A., Whitwell, J. L., & Josephs, K. A. (2015). Dominant frontotemporal dementia mutations in 140 cases of primary progressive aphasia and speech apraxia. *Dementia and Geriatric Cognitive Disorders*, 39(5–6), 281–286. doi:10.1159/000375299
- Geevasinga, N., Loy, C. T., Menon, P., de Carvalho, M., Swash, M., Schrooten, M., ... Vucic, S. (2016). Awaji criteria improves the diagnostic sensitivity in amyotrophic lateral sclerosis: A systematic review using individual patient data. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, 127(7), 2684–2691. doi:10.1016/j.clinph.2016.04.005
- Gorno-Tempini, M. L., Dronkers, N. F., Rankin, K. P., Ogar, J. M., Phengrasamy, L., Rosen, H. J., & Miller, B. L. (2004). Cognition and anatomy in three variants of primary progressive aphasia. *Annals of Neurology*, 55(3), 335–346. doi:10.1002/ana.10825

- Gorno-Tempini, M. L., Hillis, A. E., Weintraub, S., Kertesz, A., Mendez, M., Cappa, S. F., & Grossman, M. (2011). Classification of primary progressive aphasia and its variants. *Neurology*, 76(11), 1006–1014. doi:10.1212/WNL.0b013e31821103e6
- Gorno-Tempini, M. L., Ogar, J. M., Brambati, S. M., Wang, P., Jeong, J. H., Rankin, K. P., & Miller, B. L. (2006). Anatomical correlates of early mutism in progressive nonfluent aphasia. *Neurology*, 67(10), 1849–1851. doi:10.1212/01.wnl.0000237038.55627.5b
- Gregory, C., Lough, S., Stone, V., Erzincinoglu, S., Martin, L., Baron-Cohen, S., & Hodges, J. R. (2002). Theory of mind in patients with frontal variant frontotemporal dementia and Alzheimer's disease: Theoretical and practical implications. *Brain*, 125(4), 752–764. doi:10.1093/brain/awf079
- Hodges, J. R., & Patterson, K. (2007). Semantic dementia: A unique clinicopathological syndrome. *The Lancet Neurology*, 6(11), 1004–1014. doi:10.1016/S1474-4422(07)70266-1
- Hogan, D. B., Jetté, N., Fiest, K. M., Roberts, J. I., Pearson, D., Smith, E. E., & Maxwell, C. J. (2016). The prevalence and incidence of frontotemporal dementia: A systematic review. *Canadian Journal of Neurological Sciences/Journal Canadien des Sciences Neurologiques*, 43(S1), S96–S109. doi:10.1017/cjn.2016.25
- Irish, M., & Piolino, P. (2016). Impaired capacity for prospection in the dementias – Theoretical and clinical implications. *British Journal of Clinical Psychology*, 55(1), 49–68.
- Johnson, J. K., Diehl, J., Mendez, M. F., Neuhaus, J., Shapira, J. S., Forman, M., ... Miller, B. L. (2005). Frontotemporal lobar degeneration: Demographic characteristics of 353 patients. *Archives of Neurology*, 62(6), 925–930. doi:10.1001/archneur.62.6.925
- Kamminga, J., Kumfor, F., Burrell, J. R., Piguet, O., Hodges, J. R., & Irish, M. (2015). Differentiating between right-lateralized semantic dementia and behavioral-variant frontotemporal dementia: An examination of clinical characteristics and emotion processing. *Journal of Neurology, Neurosurgery & Psychiatry*, 88(10), 1082–1088. doi:10.1136/jnnp-2014-309120
- Kansal, K., Mareddy, M., Sloane, K. L., Minc, A. A., Rabins, P. V., McGready, J. B., & Onyike, C. U. (2016). Survival in frontotemporal dementia phenotypes: A meta-analysis. *Dementia and Geriatric Cognitive Disorders*, 41(1-2), 109–122. doi:10.1159/000443205
- Kertesz, A., Blair, M., McMonagle, P., & Munoz, D. G. (2007). The diagnosis and course of frontotemporal dementia. *Alzheimer Disease and Associated Disorders*, 21(2), 155–163. doi:10.1097/WAD.0b013e31806547eb
- Knopman, D. S., Petersen, R. C., Edland, S. D., Cha, R. H., & Rocca, W. A. (2004). The incidence of frontotemporal lobar degeneration in Rochester, Minnesota, 1990 through 1994. *Neurology*, 62(3), 506–508.
- Knopman, D. S., & Roberts, R. O. (2011). Estimating the number of persons with frontotemporal lobar degeneration in the US population. *Journal of Molecular Neuroscience*, 45(3), 330–335. doi:10.1007/s12031-011-9538-y
- Kortte, K. B., Stevenson, J. E., Hosey, M. M., Castillo, R., & Wegener, S. T. (2012). Hope predicts positive functional role outcomes in acute rehabilitation populations. *Rehabilitation Psychology*, 57(3), 248–255.
- Kumfor, F., & Piguet, O. (2012). Disturbance of emotion processing in frontotemporal dementia: A synthesis of cognitive and neuroimaging findings. *Neuropsychology Review*, 22(3), 280–297. doi:10.1007/s11065-012-9201-6
- Lanata, S. C., & Miller, B. L. (2016). The behavioural variant frontotemporal dementia (bvFTD) syndrome in psychiatry. *Journal of Neurology, Neurosurgery & Psychiatry*, 87(5), 501–511. doi:10.1136/jnnp-2015-310697
- Leyton, C. E., Savage, S., Irish, M., Schubert, S., Piguet, O., Ballard, K. J., & Hodges, J. R. (2014). Verbal repetition in primary progressive aphasia and Alzheimer's disease. *Journal of Alzheimer's Disease: JAD*, 41(2), 575–585. doi:10.3233/JAD-132468
- Libon, D. J., Xie, S. X., Moore, P., Farmer, J., Antani, S., McCawley, G., ... Grossman, M. (2007). Patterns of neuropsychological impairment in frontotemporal dementia. *Neurology*, 68(5), 369–375.

- Litvan, I., Agid, Y., Calne, D., Campbell, G., Dubois, B., Duvoisin, R. C., ... Zee, D. S. (1996). Clinical research criteria for the diagnosis of progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome): Report of the NINDS-SPSP international workshop. *Neurology*, 47(1), 1–9. doi:10.1212/WNL.47.1.1
- Litvan, I., Grimes, D. A., Lang, A. E., Jankovic, J., McKee, A., Verny, M., ... Pearce, R. K. B. (1999). Clinical features differentiating patients with postmortem confirmed progressive supranuclear palsy and corticobasal degeneration. *Journal of Neurology*, 246, 1–15.
- Manoochchri, M., & Huey, E. D. (2012). Diagnosis and management of behavioral issues in frontotemporal dementia. *Current Neurology and Neuroscience Reports*, 12(5), 528–536. doi:10.1007/s11910-012-0302-7
- Marra, C., Quaranta, D., Zinno, M., Misciagna, S., Bizzarro, A., Masullo, C., ... Gainotti, G. (2007). Clusters of cognitive and behavioral disorders clearly distinguish primary progressive aphasia from frontal lobe dementia, and Alzheimer's disease. *Dementia & Geriatric Cognitive Disorders*, 24(5), 317–326. doi:10.1159/000108115
- Mercy, L., Hodges, J. R., Dawson, K., Barker, R. A., & Brayne, C. (2008). Incidence of early-onset dementias in Cambridgeshire, United Kingdom. *Neurology*, 71(19), 1496–1499. doi:10.1212/01.wnl.0000334277.16896.fa
- Merrilees, J., Dowling, G. A., Hubbard, E., Mastick, J., Ketelle, R., & Miller, B. L. (2013). Characterization of apathy in persons with frontotemporal dementia and the impact on family caregivers. *Alzheimer Disease and Associated Disorders*, 27(1), 62. doi:10.1097/W AD.0b013e3182471c54
- Merrilees, J., Klapper, J., Murphy, J., Lomen-Hoerth, C., & Miller, B. L. (2010). Cognitive and behavioral challenges in caring for patients with frontotemporal dementia and amyotrophic lateral sclerosis. *Amyotrophic Lateral Sclerosis*, 11(3), 298–302. doi:10.3109/17482961003605788
- Mesulam, M., Rogalski, E., Wieneke, C., Cobia, D., Rademaker, A., Thompson, C., & Weintraub, S. (2009). Neurology of anomia in the semantic variant of primary progressive aphasia. *Brain*, 132(Pt 9), 2553–2565. doi:10.1093/brain/awp138
- Mesulam, M. M., Weintraub, S., Rogalski, E. J., Wieneke, C., Geula, C., & Bigio, E. H. (2014). Asymmetry and heterogeneity of Alzheimer's and frontotemporal pathology in primary progressive aphasia. *Brain*, 137(4), 1176–1192. doi:10.1093/brain/awu024
- Mioshi, E., Bristow, M., Cook, R., & Hodges, J. R. (2009). Factors underlying caregiver stress in frontotemporal dementia and Alzheimer's disease. *Dementia and Geriatric Cognitive Disorders*, 27(1), 76–81. doi:10.1159/000193626
- Mioshi, E., Foxe, D., Leslie, F., Savage, S., Hsieh, S., Miller, L., & Piguet, O. (2013). The impact of dementia severity on caregiver burden in frontotemporal dementia and Alzheimer disease. *Alzheimer Disease & Associated Disorders*, 27(1), 68–73. doi:10.1097/W AD.0b013e318247a0bc
- Murray, R., Neumann, M., Forman, M. S., Farmer, J., Massimo, L., Rice, A., & Gorno-Tempini, M. L. (2007). Cognitive and motor assessment in autopsy-proven corticobasal degeneration. *Neurology*, 68(16), 1274–1283. doi:10.1212/01.wnl.0000259519.78480.c3
- Olney, N. T., Spina, S., & Miller, B. L. (2017). Frontotemporal dementia. *Neurologic Clinics*, 35(2), 339–374. doi:10.1016/j.ncl.2017.01.008
- Olney, R. K., Murphy, J., Forshaw, D., Garwood, E., Miller, B. L., Langmore, S., ... Lomen-Hoerth, C. (2005). The effects of executive and behavioral dysfunction on the course of ALS. *Neurology*, 65(11), 1774–1777.
- Onyike, C. U., & Diehl-Schmid, J. (2013). The epidemiology of frontotemporal dementia. *International Review of Psychiatry*, 25(2), 130–137. doi:10.3109/09540261.2013.776523
- Park, H. K., & Chung, S. J. (2013). New perspective on parkinsonism in frontotemporal lobar degeneration. *Journal of Movement Disorders*, 6(1), 1. doi:10.14802/jmd.13001
- Piguet, O., & Hodges, J. R. (2013). Behavioural-variant frontotemporal dementia: An update. *Dementia & Neuropsychologia*, 7(1), 10–18. doi:10.1590/S1980- 57642013DN70100003
- Possin, K. L., Chester, S. K., Laluz, V., Bostrom, A., Rosen, H. J., Miller, B. L., & Kramer, J. H. (2012). The frontal-anatomic specificity of design fluency repetitions and their diagnostic relevance for

- behavioral variant frontotemporal dementia. *Journal of the International Neuropsychological Society*, 18(05), 834–844. doi:10.1017/S1355617712000604
- Rascovsky, K., Hodges, J. R., Knopman, D., Mendez, M. F., Kramer, J. H., Neuhaus, J., ... Miller, B. L. (2011). Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. *Brain: A Journal of Neurology*, 134(9), 2456–2477. doi:10.1093/brain/awr179
- Rohrer, J. D., Lashley, T., Schott, J. M., Warren, J. E., Mead, S., Isaacs, A. M., ... Warren, J. D. (2011). Clinical and neuroanatomical signatures of tissue pathology in frontotemporal lobar degeneration. *Brain: A Journal of Neurology*, 134(9), 2565–2581. doi:10.1093/brain/awr198
- Rohrer, J. D., Rossor, M. N., & Warren, J. D. (2010). Syndromes of nonfluent primary progressive aphasia: A clinical and neurolinguistic analysis. *Neurology*, 75(7), 603–610. doi:10.1212/WNL.0b013e3181ed9c6b
- Seelaar, H., Rohrer, J. D., Fox, N. C., van Swieten, J. C., & Pijnenburg, Y. A. L. (2011). Clinical, genetic and pathological heterogeneity of frontotemporal dementia: A review. *Journal of Neurology, Neurosurgery, and Psychiatry*, 82, 476–486.
- Seeley, W. W., Crawford, R., Rascovsky, K., Kramer, J. H., Weiner, M., Miller, B. L., & Gorno-Tempini, M. L. (2008). Frontal paralimbic network atrophy in very mild behavioral variant frontotemporal dementia. *Archives of Neurology*, 65(2), 249–255. doi:10.1001/archneurol.2007.38
- Suhonen, N., Kaivorinne, A., Moilanen, V., Bode, M., Takalo, R., Hänninen, T., & Remes, A. M. (2015). Slowly progressive frontotemporal lobar degeneration caused by the C9ORF72 repeat expansion: A 20-year follow-up study. *Neurocase*, 21(1), 85–89. doi:10.1080/13554794.2013.873057
- Talerico, K. A., & Evans, L. K. (2001). Responding to safety issues in frontotemporal dementias. *Neurology*, 56(suppl 4), S52–S55. doi:10.1212/WNL.56.suppl_4.S52
- Thompson, C. K., & Mack, J. E. (2014). Grammatical Impairments in PPA. *Aphasiology*, 28(8–9), 1018–1037.
- Wadia, P. M., & Lang, A. E. (2007). The many faces of corticobasal degeneration. *Parkinsonism & Related Disorders*, 13, 336–340. doi:10.1016/S1353-8020(08)70027-0
- Wilson, S. M., Henry, M. L., Besbris, M., Ogar, J. M., Dronkers, N. F., Jarrold, W., & Gorno-Tempini, M. L. (2010). Connected speech production in three variants of primary progressive aphasia. *Brain*, 133(7), 2069–2088. doi:10.1093/brain/awq129
- Woolley, J. D., Khan, B. K., Murthy, N. K., Miller, B. L., & Rankin, K. P. (2011). The diagnostic challenge of psychiatric symptoms in neurodegenerative disease: Rates of and risk factors for prior psychiatric diagnosis in patients with early neurodegenerative disease. *Journal of Clinical Psychiatry*, 72(2), 126–133. doi:10.4088/JCP.10m06382oli

Suggested Reading

- Bott, N. T., Radke, A., Stephens, M. L., & Kramer, J. H. (2014). Frontotemporal dementia: Diagnosis, deficits and management. *Future Medicine*, 4, 439–454.
- Finger, E. C. (2016). Frontotemporal dementias. *Continuum (Minneapolis, Minn.)*, 22(2 Dementia), 464–489.
- Perry, D. C., & Miller, B. L. (2018). Clinical aspects of frontotemporal dementia. In B. L. Miller & J. L. Cummings (Eds.), *The human frontal lobes: Functions and disorders* (3rd ed., pp. 239–252). New York, NY: Guilford Press.
- Perry, D. C., & Rosen, H. J. (2016). Frontotemporal dementia. In M. D. Geschwind & C. R. Belkoura (Eds.), *Non-Alzheimer's and atypical dementia* (pp. 49–63). West Sussex, UK: Wiley.

Traumatic Brain Injury (TBI)

Matthew R. Powell¹ and Michael McCrea²

¹*Division of Neurocognitive Disorders, Department of Psychiatry & Psychology,
Mayo Clinic Minnesota, Rochester, MN, USA*

²*Brain Injury Research Program, Department of Neurosurgery, Medical College of Wisconsin,
Milwaukee, WI, USA*

Introduction

Traumatic brain injury (TBI) is a significant public health problem facing all industrialized countries. Estimates of TBI in the United States suggest that as many as 4 million brain injuries occur per year (Coronado, Johnson, Faul, & Kegler, 2006; Silver, McAllister, & Yudofsky, 2005), resulting in over 1 million hospital emergency department visits, 300,000 hospitalizations, and 50,000 deaths result from TBI annually (Rutland-Brown, Langlois, Thomas, & Xi, 2006).

This chapter will provide an overview of TBI across the spectrum of severity. Particular focus will be placed on evaluation and management of mild traumatic brain injury (mTBI), given (a) its high incidence relative to moderate and severe TBI and (b) the importance of understanding the complex array of neurobiological, psychosocial, and environmental factors known to influence recovery and outcome from mTBI. The behavioral approaches to managing persisting symptoms following mTBI should be of interest to neuropsychologists or other mental health providers involved in care delivery for mTBI patients in a variety of settings.

Definition/Classification

TBI is commonly defined as *an alteration in brain function, or other evidence of brain pathology, caused by an external force, and characterized by the following: 1) any period of loss or decreased level of consciousness (LOC), 2) any loss of memory for events immediately before (retrograde) or after (posttraumatic) the injury, 3) any neurological deficits, and/or 4) any alteration in mental state at the time of injury* (National Institute for Neurologic Disorders and Stroke [NINDS])

Common Data Elements [CDE] for TBI; <https://www.commondataelements.ninds.nih.gov/Traumatic%20Brain%20Injury>).

Evidence of TBI can include visual, neuroradiologic, or laboratory confirmation of damage to the brain, but TBI is more often diagnosed on the basis of acute clinical criteria. Modern structural (e.g., diffusion tensor MRI) and functional imaging (resting-state functional MRI) techniques show promise as emerging tools to evaluate TBI, and it is possible that other sensitive biomarkers may be developed in the future.

Common mechanisms of TBI include the head being struck by an object, the head striking an object, the brain undergoing an acceleration/deceleration movement, a foreign body penetrating the brain, or forces generated from events such as a blast or explosion. Motor vehicle crashes have consistently been cited as the most common cause of TBI, accounting for 40–60% of all TBIs annually (Kraus & Chu, 2005). All forms of transportation, however, are common causes of TBI, including motorcycle crashes, bicycle accidents, and pedestrian injuries. Certainly, there has been an increased focus on the high frequency of concussion or mTBI encountered by athletes participating in contact and collision sports at all competitive levels.

Numerous systems have been developed over the years to define and classify TBI severity along a continuum from mild to moderate to severe. The Glasgow Coma Scale (GCS) (Jennett & Teasdale, 1981) is the most recognized and widely used method for grading TBI severity and can often be retrieved from hospital records. The GCS provides an indicator of gross neurologic status by assessing motor function, verbal responding, and the patient’s ability to open his or her eyes voluntarily or in response to external commands and stimuli. Other classification systems that use multiple severity indicators improve the sensitivity in the detection of mTBI, while also taking into consideration traditional acute injury characteristics that have been presumed to predict outcome following mild and moderate brain injury (see Table 1 for example).

Functional Neuroanatomy of TBI

Focal Brain Injury

In more severe forms of TBI, structural brain damage after trauma can be categorized based on clinical and neuroradiologic findings. Common forms of focal brain injury include skull fracture, contusions and lacerations, intracranial hematoma, epidural hematoma (EDH), subdural hematoma (SDH), and brain damage due to increased intracranial pressure. Contusions are the most common brain damage due to TBI and have a characteristic distribution preferentially affecting the pole of the frontal lobe, the inferior aspect of the frontal lobes, the temporal poles, the lateral and inferior aspects of the temporal lobe, and the cortex above and

Table 1 Multiple severity indicators of traumatic brain injury.

<i>Measure</i>	<i>Severity classification</i>		
	<i>Mild</i>	<i>Moderate</i>	<i>Severe</i>
Glasgow coma scale	13–15	9–12	3–8
Loss of consciousness	<30 min	30 min to 24 hr	>24 hr
Posttraumatic amnesia	<24 hr	1–7 days	>7 days

below the operculum of the Sylvian fissures. Intracranial hematoma is the most common cause of clinical deterioration and death in patients who were initially lucid after TBI. SDH is a common occurrence in TBIs of any severity, especially among older adults, which is often associated with swelling of the ipsilateral cerebral hemisphere and can cause secondary complications from mass effects.

Diffuse Brain Injury

Acceleration/deceleration and rotational forces often produce effects that are microscopic and distributed widely in the brain. The most common of these, and essentially the signature injury in mTBI, is traumatic axonal injury (TAI). TAI, which historically has been referred to as diffuse axonal injury (DAI), occurs when the brain is deformed from traumatic forces causing axons to be stretched sufficiently to produce at least a transient disruption in neural functioning.

Secondary Brain Injury

Primary traumatic damage to the brain may be further complicated by secondary insults that occurred acutely or subacutely. In patients with severe brain injury, hypoxia is considered the most common secondary insult. Other forms of secondary injury may include diffuse (multifocal) vascular injury, focal or widespread cerebral swelling, or disruption of normal brain electrical activity that may cause posttraumatic seizures.

Pathophysiology of mTBI or Concussion

Collectively, the underlying pathophysiological processes of concussion have been characterized as a “neurometabolic cascade,” which has been eloquently characterized by Hovda and colleagues (Giza & Hovda, 2001). The clinical manifestation of concussion signs and symptoms results from sequential neuronal dysfunction due to ionic shifts, altered metabolism, impaired connectivity, or changes in neurotransmission. A concussion or mTBI does not typically result in identifiable structural injury to neurons and axons or any measureable cell death. The time course of this physiological disruption is considered to generally parallel that of clinical symptoms and recovery time.

Traumatic Brain Injury and Depression

Depression is frequently diagnosed after brain injury, with symptoms of dysphoria, anhedonia, and fatigue. Additionally, patients with TBI typically report lower life satisfaction (Jacobsson, Westerberg, & Lexell, 2010), and individuals with TBI frequently have more difficulty reintegrating back into social roles. Although TBI patients appear at risk of developing depression following their injury, the relationship between depression and injury severity is not well characterized. For example, while there is data to suggest that TBI severity groups do not differ on emotional domains like depression, anxiety, or irritability, even when cognitive differences are accounted for (Malec, Testa, Rush, Brown, & Moessner, 2007; Rapoport, McCauley, Levin, Song, & Feinstein, 2002), it would not be disputed that the psychology of neurobehavioral change following mild versus severe brain injury differs. Regardless, it is well recognized that depression following all-severity TBI has some neurobiological

underpinnings. Nevertheless, mood and adjustment following injury is strongly related to patient perception about success resuming important psychosocial roles (Ownsworth et al., 2011). The nuances associated with emotional functioning across the spectrum of brain injury that are moderated by patient variables and other psychological and social factors need further investigation.

Treatment of Mild Traumatic Brain Injury

It is estimated that 70–90% of all treated traumatic brain injuries are mild in severity based on acute injury characteristics (Cassidy et al., 2004). For the majority of individuals with mTBI, the symptoms of PCS gradually resolve within a few weeks of injury. For a subset of individuals with mTBI, however, symptoms can persist well beyond expectations (i.e., months). The term post-concussive disorder (PCD) has been proposed for diagnostic use when symptoms following mTBI such as neurologic, cognitive, behavioral, or somatic complaints persist beyond the acute and subacute periods and become chronic (Iverson, Zasler, & Lange, 2007), often operationalized as persisting beyond three months. In most studies, brain injury severity characteristics are not strongly associated with risk of PCS. Rather, factors such as pre-injury adverse life events or stress or post-injury psychological factors (e.g., anxiety, attributions) are often more associated with persistent symptoms.

McCrea (2008) and Ruff and colleagues (Ruff, Camenzuli, & Mueller, 1996; Ruff & Richardson, 1999) highlight the complexity of the issues associated with PCD by stressing that PCD is not a unidimensional brain-based condition but rather an outcome influenced by cognitive, emotional, medical, psychosocial, and motivational factors, now referred to as the neurobiopsychosocial model of PCD (Iverson et al., 2007). Because of this complexity, treatments targeting PCD-related symptoms need to be equally diverse (e.g., they should follow an integrated model of care). Additionally, patients are frequently referred to behavioral health providers such as neuropsychologists, rehabilitation psychologists, health psychologists, and/or psychiatrists for a variety of reasons, but particularly when they are experiencing cognitive, emotional, or behavioral changes that accompany PCD.

Behavioral health intervention for PCD is not a singular treatment approach but should be conceptualized in three distinct ways: (a) symptom management (e.g., symptom reduction), (b) cognitive restructuring, or (c) preventative treatment. Treatment for PCD, like many treatments in medicine, is often geared toward *symptom management*. Behavioral health specialists help patients develop a behavioral program to facilitate symptom management (e.g., reduction of pain, improving sleep hygiene, using moderation during daily activities) of persistent symptoms. For example, patients with chronic daily headache or neck pain may benefit from learning progressive muscle relaxation or biofeedback procedures from a qualified health psychologist. Patients with mood disorders (e.g., depression), anxiety disorders (e.g., posttraumatic stress disorder), or adjustment reactions may benefit from psychiatric consultation or time-limited cognitive behavioral therapy (CBT) to increase patients' awareness of factors contributing to psychiatric difficulties, reduce their symptoms, or help them develop appropriate short- and long-term goals post-injury.

Because the symptoms of PCD are nonspecific to PCD and because they are so prevalent in normal, healthy non-brain-injured persons, some patients with PCD may be misattributing symptoms to their injury that are better explained by another source (i.e., highlighting importance of *cognitive restructuring* treatment). For example, attention and memory concerns post-injury may be related to chronic pain, psychoactive medications, or a posttraumatic stress reaction, rather than his or her mTBI in isolation. Iverson et al. (2007) reviews

cognitive errors or biases exhibited by patients with PCD that can complicate recovery and promote disability.

While symptom management and cognitive restructuring approaches are very important for patient health and quality of life, treatment approaches focusing on *prevention* of PCD are ideal. Early preventative interventions for PCD essentially inoculate patients against a purely neurobiological perspective of concussion, which helps them take personal control over aspects of their recovery that are modifiable and view their injury, recovery, and symptoms as objectively as possible (Ferguson & Mittenberg, 1996; Miller & Mittenberg, 1998; Mittenberg, Canyock, Condit, & Patton, 2001; Mittenberg & Fischera, 1993; Mittenberg, Tremont, Zielinski, Fichera, & Rayls, 1996). Because psychological and social factors of disease (and PCD) are clearly modifiable variables (e.g., the patient, the provider and the patient's environment can influence outcome), it should not be surprising that mTBI patients who receive brief behavioral health treatments very early post-injury (hours or days) report fewer and less severe PCD-related symptoms months later relative to control patients with mTBI who do not receive these interventions (Borg et al., 2004; Comper, Bisschop, Carnide, & Tricco, 2005; Mittenberg et al., 1996; Ponsford et al., 2002).

Conclusion

TBI is a major public health problem worldwide that is associated with significant morbidity and mortality. Moderate and severe TBI present the highest risk for serious and permanent disabilities. The overwhelming majority of TBIs are classified as mild in severity, and major scientific advances have emerged to inform the underlying pathophysiology, assessment techniques, and therapeutic interventions to facilitate recovery, improve outcome, and reduce disability for those who have persisting symptoms following mTBI. TBI of any severity can be associated with neuropsychiatric sequelae (e.g., depressive disorders), but further research is needed to better understand the nuance of how injury severity is associated with risk of psychological health problems. Additional research is also needed to improve outcomes for those with the most intractable symptoms, as etiology may be complex and multifactorial. Including neuropsychologists and behavioral health professionals in the treatment of mTBI and PCD is empirically supported by research exploring the efficacy of psychoeducational and cognitive behavioral treatment paradigms for preventing or reducing PCD-related symptoms.

Author Biographies

Dr. Michael McCrea is a board-certified clinical neuropsychologist. He is a professor of neurosurgery and neurology and director of *Brain Injury Research* at the Medical College of Wisconsin in Milwaukee, Wisconsin. Dr. McCrea is a past president of the American Academy of Clinical Neuropsychology (AACN). Dr. McCrea has been an active researcher in the neurosciences, with numerous scientific publications, book chapters, and national and international lectures on the topic of traumatic brain injury. He is a member of the United States Department of Defense Health Board External Advisory Committee on Traumatic Brain Injury advising the Office of the Secretary of Defense on management and research of military-related traumatic brain injury.

Dr. Matthew Powell is a senior associate consultant and assistant professor of psychiatry and psychology at Mayo Clinic. His research interests have involved clinical recovery and management of sport concussion and mild traumatic brain injury.

References

- Borg, J., Holm, L., Peloso, P. M., Cassidy, J. D., Carroll, L. J., von Holst, H., ... Yates, D. (2004). Non-surgical intervention and cost for mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, 36, 76–83.
- Cassidy, J. D., Carroll, L. J., Peloso, P. M., Borg, J., von Holst, H., Holm, L., ... Coronado, V. G. (2004). Incidence, risk factors and prevention of mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, 36, 28–60.
- Comper, P., Bisschop, S. M., Carnide, N., & Tricco, A. (2005). A systematic review of treatments for mild traumatic brain injury. *Brain Injury*, 19(11), 863–880.
- Coronado, V. G., Johnson, R. L., Faul, M., & Kegler, S. R. (2006). Incidence rates of hospitalization related to traumatic brain injury—12 states, 2002. *MMWR. Morbidity and Mortality Weekly Report*, 55(8), 201–204.
- Ferguson, R. J., & Mittenberg, W. (1996). Cognitive-behavioral treatment of postconcussion syndrome. In V. B. Van Hasselt & M. Hersen (Eds.), *Sourcebook of psychological treatment manuals for adult disorders* (pp. 615–655). New York, NY: Plenum Press.
- Giza, C. C., & Hovda, D. A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training*, 36(3), 228–235.
- Iverson, G. L., Zasler, N. D., & Lange, R. T. (2007). Post-concussive disorder. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 373–405). New York, NY: Demos Medical Publishing.
- Jacobsson, L. J., Westerberg, M., & Lexell, J. (2010). Health-related quality-of-life and life satisfaction 6–15 years after traumatic brain injuries in northern Sweden. *Brain Injury*, 24(9), 1075–1086. doi:10.3109/02699052.2010.494590
- Jennett, B., & Teasdale, G. (1981). *Management of head injuries*. Philadelphia, PA: FA Davis.
- Kraus, J. F., & Chu, L. D. (2005). Epidemiology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 3–26). Washington, DC; London, England: American Psychiatric Publishing.
- Malec, J. F., Testa, J. A., Rush, B. K., Brown, A. W., & Moessner, A. M. (2007). Self-assessment of impairment, impaired self-awareness, and depression after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 22(3), 156–166. doi:10.1097/01.HTR.0000271116.12028.af
- McCrea, M. (2008). *Mild traumatic brain injury and post-concussion syndrome: The new evidence base for diagnosis and treatment*. New York, NY: Oxford Press.
- Miller, L. J., & Mittenberg, W. (1998). Brief cognitive behavioral interventions in mild traumatic brain injury. *Applied Neuropsychology*, 5(4), 172–183.
- Mittenberg, W., Canyock, E. M., Condit, D., & Patton, C. (2001). Treatment of post-concussion syndrome following mild head injury. *Journal of Clinical and Experimental Neuropsychology*, 23(6), 829–836.
- Mittenberg, W., & Fischera, S. (1993). Recovery from mild head injury: A treatment manual for patients. *Psychotherapy in Private Practice*, 12, 37–52.
- Mittenberg, W., Tremont, G., Zielinski, R. E., Fichera, S., & Rayls, K. R. (1996). Cognitive-behavioral prevention of postconcussion syndrome. *Archives of Clinical Neuropsychology*, 11(2), 139–145.
- Owensworth, T., Fleming, J., Haines, T., Cornwell, P., Kendall, M., Nalder, E., & Gordon, C. (2011). Development of depressive symptoms during early community reintegration after traumatic brain injury. *Journal of the International Neuropsychological Society*, 17(1), 112–119. doi:10.1017/S1355617710001311
- Ponsford, J., Willmott, C., Rothwell, A., Cameron, P., Kelly, A. M., Nelms, R., & Curran, C. (2002). Impact of early intervention on outcome following mild head injury in adults. *Journal of Neurology, Neurosurgery, and Psychiatry*, 73(3), 330–332.
- Rapoport, M., McCauley, S., Levin, H., Song, J., & Feinstein, A. (2002). The role of injury severity in neurobehavioral outcome 3 months after traumatic brain injury. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 15(2), 123–132.

- Ruff, R. M., Camenzuli, L., & Mueller, J. (1996). Miserable minority: Emotional risk factors that influence the outcome of a mild traumatic brain injury. *Brain Injury*, 10(8), 551–565.
- Ruff, R. M., & Richardson, A. M. (1999). Mild traumatic brain injury. In J. J. Sweet (Ed.), *Forensic neuropsychology: Fundamentals and practice. Studies on neuropsychology, development and cognition* (pp. 315–338). Bristol, UK: Swets & Seitinger.
- Rutland-Brown, W., Langlois, J. A., Thomas, K. E., & Xi, Y. L. (2006). Incidence of traumatic brain injury in the United States, 2003. *The Journal of Head Trauma Rehabilitation*, 21(6), 544–548.
- Silver, J. M., McAllister, T. W., & Yudofsky, S. C. (Eds.) (2005). *Textbook of traumatic brain injury* (1st ed.). Washington, DC: American Psychiatric Publishing, Inc.

Chronic Traumatic Encephalopathy: A Long-Term Consequence of Repetitive Head Impacts

Michael L. Alosco¹, Alyssa Phelps¹, and
Robert A. Stern^{1,2}

¹*Boston University Alzheimer's Disease Center and Boston University CTE Center,
Department of Neurology, Boston University School of Medicine, Boston, MA, USA*

²*Department of Neurosurgery and Anatomy & Neurobiology, Boston
University School of Medicine, Boston, MA, USA*

This chapter is intended to serve as a quick reference guide on chronic traumatic encephalopathy (CTE). CTE is a progressive neurodegenerative disease that results in a decline of clinical function (McKee et al., 2013; Mez et al., 2017; Stern et al., 2013) and is believed to be the consequence of exposure to repetitive head impacts (RHI), both concussive and subconcussive injuries (Montenigro et al., 2016). CTE was first recognized in 1928 by Harrison Martland who published a paper coining the phrase “punch drunk” to describe a series of behavioral and cognitive disturbances in Olympic prize fighters (Martland, 1928). Clinical dysfunction (e.g., behavior, mood, cognitive, and motor disturbances) (Millspaugh, 1937), as well as neuropathological evidence of neurodegeneration (e.g., hyperphosphorylated tau [p-tau]) (Corsellis, Bruton, & Freeman-Browne, 1973), was evident in boxers throughout the twentieth century. CTE, however, did not emerge as a public health concern and topic of lay conversation until 2005, following the publication of a case study of a former professional American football player who had progressive cognitive and behavioral/mood problems during life, as well as evidence of CTE neuropathology at postmortem (Omalu et al., 2005). Since 2005, CTE has been discovered in a variety of contact sports athletes (e.g., American football players, boxers, rugby players), as well as military veterans, victims of domestic violence, and other individuals with a history of RHI exposure (McKee et al., 2013, 2016; Mez et al., 2017). Over the past decade immense progress has been made in research and

awareness of CTE, and CTE may represent a major public health concern. This chapter will provide an overview of the neuropathological and clinical presentation of CTE and discuss targets of ongoing and future research.

Neuropathology of CTE

McKee et al. at the Boston University (BU) CTE Center and the Veterans Administration-BU-Concussion Legacy Foundation (VA-BU-CLF) brain bank have refined and validated the neuropathology of CTE. In 2013, as part of a National Institute of Neurological Disorders and Stroke (NINDS)-funded study known as “Understanding Neurologic Injury in Traumatic Encephalopathy (UNITE)” (PI: Ann McKee) (Mez et al., 2015), McKee et al. proposed neuropathological diagnostic criteria for CTE (McKee et al., 2013). A NINDS and National Institute of Biomedical Imaging and Bioengineering (NIBIB)-sponsored conference of 7 independent neuropathologists with expertise in neurodegenerative disease evaluated McKee and colleague’s proposed criteria using 25 selected cases of various tauopathies. There was strong agreement among the panel for the neuropathological diagnosis of CTE ($\kappa = 0.78$), with the pathognomonic lesion defined as an irregular perivascular deposition of p-tau at the depths of the cortical sulci. The neuropathology of CTE was distinct from any other neurodegenerative disease, including Alzheimer’s disease (AD).

The neuropathology of CTE involves a diverse range of progressive macro- and microscopic alterations (McKee et al., 2013; Mez et al., 2017). Although gross pathology is generally unremarkable in the early stages of CTE, widespread cortical and subcortical neural degeneration is evident with the progression of the disease, in addition to dilation of the lateral and third ventricles, septal abnormalities (e.g., cavum septum), and pallor of the locus coeruleus and substantia nigra. Microscopically, perivascular p-tau neurofibrillary and astrocytic tangles are initially localized to the sulcal depths of the superior, dorsolateral, and inferior frontal cortices. As the disease advances, the p-tau becomes widespread throughout the cortex and subcortex (including the medial temporal lobe [MTL]) and extends into the diencephalon, brain stem, and spinal cord. Axonal degeneration, fiber loss, white matter disease, neuroinflammation, and extensive deposition of the TAR DNA-binding protein 43 (TDP-43) can also be found in the majority of CTE cases. Mez et al. (2017) found beta-amyloid protein deposits in a subset of participants at all stages of CTE pathology, and in stage IV CTE, deposition occurred in 91% of cases. However, beta-amyloid protein deposits are often diffuse, and not neuritic, plaques and associated with age and the APOE $\epsilon 4$ allele (Mez et al., 2017; Stein, Montenegro, et al., 2015). CTE is frequently comorbid with other neurodegenerative diseases, including AD, Lewy body disease (LBD), motor neuron disease (MND), and FTLN (Ling et al., 2017; McKee et al., 2013; Mez et al., 2017). Recent research proposes argyrophilic grain disease and Parkinson’s disease as potential comorbidities of CTE as well (Armstrong, McKee, Stein, Alvarez, & Cairns, 2016).

RHI Exposure and CTE

RHI seems to be a necessary risk factor for CTE. However, it is not sufficient as not all individuals exposed to RHI develop CTE. There are a number of other risk factors thought to interact with RHI that may contribute to the development of CTE and clinical decline. A few potential factors include cumulative exposure to RHI (Montenegro et al., 2016), early age at

first exposure (Alosco, Kasimis, et al., 2017; Schultz et al., 2017; Stamm, Bourlas, et al., 2015; Stamm, Koerte, et al., 2015), presence of the *APOE* genotype (Stern et al., 2013), older age (McKee et al., 2013, Mez et al., 2017), cognitive reserve (Alosco et al., 2016), and cerebrovascular health (Alosco, Koerte, et al., 2017). Despite common belief, recurrent subconcussive head trauma, not concussion, seems to play a prominent role in the development of CTE (McKee et al., 2013; Mez et al., 2017; Stein, Alvarez, & McKee, 2015). In a Mez et al. (2017) case series of 202 deceased former American football players, 3 of 14 high school (21%), 48 of 53 college (91%), 9 of 14 semiprofessional (64%), 7 of 8 Canadian Football League (88%), and 110 of 111 National Football League (NFL) (99%) players all showed evidence of CTE neuropathology. All three former high school players had mild pathology (stages I and II), and the majority of higher level players had severe pathology (stages III and IV) (Mez et al., 2017). Additionally, a recent study by Ling et al. (2017) hypothesized that CTE in retired soccer players may be related to their prolonged exposure to repetitive subconcussive head impacts from heading and head-to-player collisions. The concussion rate was limited in 6 of the 14 cases to one event during their careers; however, all cases developed a progressive dementing illness in later life. All six of the histological examinations revealed some features supportive of CTE, including characteristic tau pathologies, dilation of the third ventricle, and septal abnormalities (Ling et al., 2017). Research from the Mayo Clinic brain bank supports the relationship between RHI exposure and CTE (Bieniek et al., 2015). The contact sports history of 1,700 brain donation cases was ascertained from the Mayo Clinic brain bank and was correlated with their neuropathology. The brains of sixty-six males who participated in contact sports at the amateur level (34 of which played American football) were neuropathologically examined, revealing that 21 of the 66 donation cases had the diagnostic lesion of CTE (Bieniek et al., 2015). CTE neuropathology was *not* present in 198 age- and disease-matched men and women who did not have a history of contact sports participation. The findings presented by the Mayo Clinic are remarkable in that they are not limited by sample ascertainment bias due to recruitment of subjects with a history of RHI exposure and suspected neuropathology or CTE.

Clinical Presentation of CTE

Stern et al. (2013) performed retrospective family telephone interviews and medical record reviews for 36 contact sports male athletes (29 American football players, 3 professional hockey players, 1 professional wrestler, and 3 former boxers) who were neuropathologically diagnosed with *only* CTE in order to describe the clinical presentation of the disease. Of the 36 subjects, a subgroup of 22 exhibited initial declines in behavior and mood at a mean age of 35, primarily characterized by explosivity, impulsivity, and physical and verbal violence, in addition to depression and related symptoms (e.g., apathy, suicidality). This group ultimately developed cognitive symptoms; however, there was a second subgroup of 11 subjects who had cognitive difficulties as the initial symptom at a mean age of 60, especially in episodic memory and executive function. This subgroup tended to develop dementia and had more advanced CTE neuropathology. Three subjects were asymptomatic, potentially because one was only 17 years old and had early-stage CTE neuropathology (stage I), and the other two had very high cognitive reserve (based on their advanced graduate degrees and professional success). Despite previous research on boxers describing motor disturbances (e.g., parkinsonism) as a prominent feature of CTE, Stern et al. found little evidence of this feature in a sample of mostly former American football players. This is consistent with previous research indicating that boxers tended to have more pathology in brain regions (e.g., cerebellum) that cause

parkinsonism (McKee et al., 2013). This may be due to differences in type and angle of force related to the biomechanics of head trauma, in which boxers receive focal stress to the brain stem and cerebellar regions.

A recent study by Mez et al. (2017) further describes the clinical presentation of CTE in 111 cases of former American football players. Both mild and severe cases of CTE pathology reported a progressive clinical course, and there were no asymptomatic cases. Impulsivity, depression, apathy, and anxiety, among other behavioral or mood symptoms, were frequently reported in 26 (96%) mild cases and 75 (89%) severe cases. Cognitive symptoms such as memory, executive function, and attention difficulties were also frequently reported in 23 (85%) mild cases and 80 (95%) severe cases. Both motor symptoms and dementia were common only in severe cases. Among the 111 CTE cases with standardized informant reports on clinical symptoms, 47 (42.3%) initially presented with cognitive symptoms, 48 (43.2%) initially presented with behavior or mood symptoms, and 16 (14.4%) initially presented with both. These clinical observations confirm and expand on previous reports of clinical presentations of CTE (Stern et al., 2013).

Clinical Research Diagnostic Criteria

CTE cannot currently be detected during life. A number of different research groups (Jordan, 2013; Montenegro et al., 2014; Victoroff, 2013) have proposed provisional research diagnostic criteria for the clinical syndrome associated with CTE and/or exposure to RHI, most recently being traumatic encephalopathy syndrome (TES) (Montenegro et al., 2014). The purpose of the TES criteria, and other proposed criteria, is to facilitate clinical research investigations on CTE and is not intended for clinical diagnostic purposes. TES is based on the clinical features of neuropathologically confirmed cases of CTE. A TES diagnosis requires a history of multiple impacts to the head (e.g., from contact sports participation, military service, domestic violence), including concussions, subconcussive injuries, and/or moderate to severe traumatic brain injury (TBI). Based on signs and symptoms found in a majority of neuropathologically confirmed CTE cases (Stern et al., 2013), at least one of three core clinical features must be present: (a) behavioral disturbances (e.g., aggression, impulsivity), (b) mood dysfunction (e.g., depression), and (c) cognitive decline, particularly in executive function and episodic memory, that is corroborated by standardized testing. Two supportive features (impulsivity, anxiety, apathy, paranoia, suicidality, headache, motor signs, documented decline, delayed onset) are also required for a TES diagnosis, as well as a symptom duration of at least 12 months. No other neurological disorder can fully account for the clinical features. These core features form four distinct TES diagnostic variants: behavioral/mood, cognitive, mixed, and dementia. For all diagnostic variants, motor disturbances and the clinical course (i.e., “stable,” “progressive,” or “unknown/inconsistent”) must be indicated. TES dementia requires a progressive clinical course and functional impairment.

TES is used to describe the clinical syndrome associated with exposure to RHI and is not intended to indicate underlying neuropathology. TES can be a manifestation of other neurological conditions and/or neurodegenerative diseases associated with head trauma. If an individual meets TES diagnostic criteria, the next step is to indicate the likelihood of CTE being the etiology through the designations of “probable CTE,” “possible CTE,” or “unlikely CTE.” A “probable CTE” diagnosis requires the presence of an *in vivo* biomarker that is suggestive of underlying CTE neuropathology. Without confirmation of a biomarker, only a diagnosis of “possible CTE” can be made.

Proposed *In Vivo* Biomarkers of CTE

Although validated *in vivo* biomarkers of CTE are not yet available, several have been proposed based on the neuropathology of CTE and preliminary biomarker studies in subjects at high risk for CTE, that is, former professional American football players. Various magnetic resonance imaging (MRI) technologies/approaches (i.e., high-resolution T1 anatomical MRI, diffusion tensor imaging [DTI], functional MRI [fMRI], arterial spin labeling [ASL], single-photon emission computed tomography [SPECT], MR spectroscopy [MRS]) have been examined in former professional American football players and provide support for the following as potential “proxy” markers of CTE neuropathological changes, but not of p-tau deposition: *cavum septum pellucidum* (Gardner et al., 2016; Koerte et al., 2016), *cortical atrophy and thinning* (Adler et al., 2016; Coughlin et al., 2015; Strain et al., 2015), *white matter alterations* (Alosco, Koerte, et al., 2017; Goswami et al., 2016; Strain et al., 2013), *cerebral hypoperfusion and functional hypoactivity* (Ford, Giovanello, & Guskiewicz, 2013; Hampshire, MacDonald, & Owen, 2013; Hart et al., 2013; Terry, Adams, Ferrara, & Miller, 2015), and *neurochemical alterations* (Lin et al., 2015).

Currently, the most promising method for the *in vivo* detection of underlying CTE neuropathology (i.e., p-tau) is positron emission tomography (PET) amyloid and tau imaging. In the case of AD, the Food and Drug Administration (FDA) has approved PET ligands that bind to amyloid (e.g., [(18)F]-florbetapir) and are used for the detection of amyloid uptake. There is preliminary support for the use of p-tau PET ligands in CTE, such as FDDNP (Barrio et al., 2015; Small et al., 2013) and [(18)F]-flortaucipir (also known as AV-1451 and T807) (Mitsis et al., 2014). Of particular interest is flortaucipir because of its improved specificity to p-tau, in general, and to paired helical filament (PHF) tau isoforms seen in CTE (e.g., 3R/4R). Not only will the combination of amyloid and tau PET imaging play a critical role in the detection of CTE during life, but it will also help in differentiating from similar neurodegenerative diseases (e.g., AD).

Fluid biomarkers, such as blood, saliva, and cerebrospinal fluid (CSF), are practical alternatives to PET imaging that can detect underlying AD neuropathology (Olsson et al., 2016) and may have similar applications in CTE. Recent research in symptomatic former NFL players and controls provides preliminary evidence for plasma exosomal tau (Stern et al., 2016) and plasma total tau (t-tau) (Alosco, Tripodis, et al., 2017) as candidate biomarkers for CTE. Alosco, Tripodis, et al. (2017) examined plasma t-tau in the blood of symptomatic former NFL players and revealed that a higher exposure to RHI predicted greater plasma t-tau later in life. However, plasma t-tau did not predict clinical function. CSF measures of t-tau, p-tau, and beta-amyloid are also of particular interest as potential biomarkers of CTE. Analysis of CSF concentrations in deceased professional American football players with neuropathologically confirmed CTE found a trend for increased CCL11, a protein previously associated with age-associated cognitive decline (Cherry et al., 2017). There was also an association between CSF CCL11 levels, and the number of years exposed to football was observed independent of age (Cherry et al., 2017). These biomarkers require more research but may ultimately become a simple diagnostic measure of CTE.

Conclusions and Future Directions

CTE is a distinct, debilitating, and progressive neurodegenerative disease associated with exposure to RHI. Given millions of contact sports athletes, military personnel, and domestic

violence victims exposed to RHI each year, CTE may become a major public health concern. Research on the disease is still in its early phases. Provisional clinical research diagnostic criteria have been proposed, that is, TES (Montenigro et al., 2014), but require refinement and validation until it can be used for clinical diagnostic purposes. It is currently not possible to detect CTE during life, partially because validated *in vivo* biomarkers do not yet exist, and the clinical presentation of CTE remains ill defined (Asken et al., 2017; Iverson et al., 2017). More research needs to be done in order to assess the risk factors and epidemiology of CTE, as well as testing of proposed pathophysiological mechanisms that underpin the association between RHI and exposure and CTE (Cherry et al., 2016). A NINDS-funded, 7-year multicenter study, referred to as “Diagnostics, Imaging, and Genetics Network for the Objective Study and Evaluation of Chronic Traumatic Encephalopathy (DIAGNOSE CTE) Research Project” (PIs: Robert A. Stern [Contact PI], Jeffrey Cummings, Eric Reiman, Martha Shenton) is aiming to fulfill some of these needs. DIAGNOSE CTE is a longitudinal examination (baseline and 3-year follow-up) of symptomatic and asymptomatic former NFL players, symptomatic and asymptomatic former college football players, and an asymptomatic control group without a history of brain trauma or contact sports participation. At each time point, subjects complete clinical exams (neurological, motor, neuropsychological, neuropsychiatric, and daily functioning), neuroimaging (PET tau and amyloid, MRI, fMRI, DTI, MRS), lumbar puncture, and blood draw for DNA extraction and blood protein analysis. The ultimate endpoint for DIAGNOSE CTE is to develop and refine methods and criteria to diagnose CTE during life in order to initiate clinical trials and to facilitate epidemiological studies on CTE.

Author Biographies

Dr. Michael L. Alosco, PhD is an assistant professor of neurology at the Boston University School of Medicine (BUSM). He completed his undergraduate studies at Providence College, and he earned his doctoral degree in clinical psychology, with a focus in neuropsychology, in 2015 from Kent State University. He completed his clinical internship in neuropsychology at the VA Boston Healthcare System. Dr. Alosco then completed a 3-year postdoctoral fellowship at the BU Alzheimer’s Disease (AD) Center and CTE Center, initially through the NIA-funded T32 Alzheimer’s Disease Translational Research Training Program, followed by a National Institute of Neurological Disorders and Stroke-funded Ruth L. Kirschstein F32 National Research Service Award (NRSA). Dr. Alosco’s research focuses on risk factors and biomarkers of neurodegenerative conditions, with a focus on AD and CTE. Dr. Alosco is particularly interested in the contribution of cerebrovascular disease to the clinical and neuropathological presentation of AD and CTE. He is additionally involved in research examining the relationship between exposure to repetitive head impacts and long-term neurological consequences. Dr. Alosco plays a critical role in several large-scale *in vivo* and *ex vivo* studies on AD and CTE at the BU AD and CTE Center. Dr. Alosco has accumulated >100 peer-reviewed publications, has written numerous book chapters, serves as an ad hoc reviewer for several journals, and has received several honors and awards.

Ms. Alyssa Phelps, BA completed her undergraduate studies at Cornell University where she graduated with honors in psychology. Her undergraduate research focused on integrative neuroethology and cognitive neuroscience. Since graduating, Alyssa transitioned to the Boston University (BU) Alzheimer’s Disease (AD) and CTE Center where she is an intern working on the Longitudinal Examination to Gather Evidence of Neurodegenerative Disease (LEGEND) Study. In the future, Alyssa hopes to pursue a doctoral degree in clinical psychology.

Dr. Robert L. Stern, PhD received his PhD in clinical psychology from the University of Rhode Island. He completed his predoctoral internship training under Dr. Edith Kaplan at the Boston VA Medical Center and his postdoctoral fellowship at the University of North Carolina School of Medicine. He is currently professor of Neurology, Neurosurgery, and Anatomy and Neurobiology at Boston University (BU) School of Medicine, where he is also director of the Clinical Core of the NIH-funded BU Alzheimer's Disease Center and director of Clinical Research for the BU Chronic Traumatic Encephalopathy (CTE) Center. A major focus of his research involves the long-term effects of repetitive head impacts in athletes, including the neurodegenerative disease, CTE. He is the lead co-principal investigator of a \$16 million NIH grant for a multicenter longitudinal study to develop methods of diagnosing CTE during life as well as examining potential risk factors of the disease. Dr. Stern's other current major area of funded research involves the diagnosis and treatment of Alzheimer's disease. He has published on various aspects of cognitive assessment and is the senior author of the *Neuropsychological Assessment Battery* (NAB), as well as the *Boston Qualitative Scoring System for the Rey-Osterrieth Complex Figure*. He has received numerous NIH and other national grants, and he is a Fellow of both the National Academy of Neuropsychology and the American Neuropsychiatric Association. Dr. Stern has over 175 peer-reviewed publications, is on several journal editorial boards, and is the co-editor of two upcoming books: *Sports Neurology* (part of the Handbook in Clinical Neurology series published by Elsevier) and *The Oxford Handbook of Adult Cognitive Disorders*. He is a member of the medical advisory boards of several biotech/pharma companies as well as the Mackey-White Health and Safety Committee of the NFL Players Association and the Medical Scientific Committee for the NCAA Student-Athlete Concussion Injury Litigation.

References

- Adler, C. M., DelBello, M. P., Weber, W., Williams, M., Duran, L. R. P., Fleck, D., ... Divine, J. (2016). MRI evidence of neuropathic changes in former college football players. *Clinical Journal of Sport Medicine*. doi:10.1097/JSM.0000000000000391
- Alosco, M. L., Kasimis, A. B., Stamm, J. M., Chua, A. S., Baugh, C. M., Daneshvar, D. H., ... Stern, R. A. (2017). Age of first exposure to American football and long-term neuropsychiatric and cognitive outcomes. *Translational Psychiatry*, 7, e1236. doi:10.1038/tp.2017.197
- Alosco, M. L., Koerte, I. K., Tripodis, Y., Mariani, M., Chua, A. S., Jarnagin, J., ... Stern, R. A. (2017). White matter signal abnormalities in former national football league players. *Alzheimers Dement (Amst)*, 10, 56–65. doi:10.1016/j.dadm.2017.10.003
- Alosco, M. L., Mez, J., Kowall, N. W., Stein, T. D., Goldstein, L. E., Cantu, R. C., ... AC, M. K. (2016). Cognitive reserve as a modifier of clinical expression in chronic traumatic encephalopathy: A preliminary examination. *The Journal of Neuropsychiatry and Clinical Neurosciences*, appineuro-psych16030043. doi:10.1176/appi.neuropsych.16030043
- Alosco, M. L., Tripodis, Y., Jarnagin, J., Baugh, C. M., Martin, B., Chaisson, C. E., ... Stern, R. A. (2017). Repetitive head impact exposure and later-life plasma total tau in former NFL players. *Alzheimers Dement (Amst)*, 7, 33–40. doi:10.1016/j.dadm.201611003
- Armstrong, R. A., McKee, A. C., Stein, T. D., Alvarez, V. E., & Cairns, N. J. (2016). A quantitative study of tau pathology in eleven cases of chronic traumatic encephalopathy. *Neuropathology and Applied Neurobiology*. doi:10.1111/nan.12323
- Asken, B. M., Sullan, M. J., Dekosky, S. T., Jaffee, M. S., & Bauer, R. M. (2017). Research gaps and controversies in chronic traumatic encephalopathy. *JAMA Neurology*, 74(10), 1255–1262. doi:10.1001/jamaneurol.2017.2396

- Barrio, J. R., Small, G. W., Wong, K. P., Huang, S.-C., Liu, J., Merrill, D. A., ... Kepe, V. (2015). In vivo characterization of chronic traumatic encephalopathy using [F-18]FDDNP PET brain imaging. *Proceedings of the National Academy of Sciences of the United States of America*, 112(16), E2039–E2047. doi:10.1073/pnas.1409952112
- Bieniek, K. F., Ross, O. A., Cormier, K. A., Walton, R. L., Soto-Ortolaza, A., Johnston, A. E., ... Dickson, D. W. (2015). Chronic traumatic encephalopathy pathology in a neurodegenerative disorders brain bank. *Acta Neuropathologica*, 130(6), 877–889. doi:10.1007/s00401-015-1502-4
- Cherry, J. D., Stein, T. D., Tripodis, Y., Alvarez, V. E., Huber, B. R., Au, R., ... McKee, A. C. (2017). CCL11 is increased in the CNS in chronic traumatic encephalopathy but not in Alzheimer's disease. *PLoS One*, 12(9), e0185541. doi:10.1371/journal.pone.0185541
- Cherry, J. D., Tripodis, Y., Alvarez, V. E., Huber, B., Kiernan, P. T., Daneshvar, D. H., ... Stein, T. D. (2016). Microglial neuroinflammation contributes to tau accumulation in chronic traumatic encephalopathy. *Acta Neuropathologica Communications*, 4(1), 112. doi:10.1186/s40478-016-0382-8
- Corsellis, J. A., Bruton, C. J., & Freeman-Browne, D. (1973). The aftermath of boxing. *Psychological Medicine*, 3(3), 270–303.
- Coughlin, J. M., Wang, Y., Munro, C. A., Ma, S., Yue, C., Chen, S., ... Pomper, M. G. (2015). Neuroinflammation and brain atrophy in former NFL players: An in vivo multimodal imaging pilot study. *Neurobiology of Disease*, 74, 58–65. doi:10.1016/j.nbd.2014.10.019
- Ford, J. H., Giovanello, K. S., & Guskiewicz, K. M. (2013). Episodic memory in former professional football players with a history of concussion: An event-related functional neuroimaging study. *Journal of Neurotrauma*, 30(20), 1683–1701. doi:10.1089/neu.2012.2535
- Gardner, R. C., Hess, C. P., Brus-Ramer, M., Possin, K. L., Cohn-Sheehy, B. I., Kramer, J. H., ... Rabinovici, G. D. (2016). Cavum septum pellucidum in retired American pro-football players. *Journal of Neurotrauma*, 33(1), 157–161. doi:10.1089/neu.2014.3805
- Goswami, R., Dufort, P., Tartaglia, M. C., Green, R. E., Crawley, A., Tator, C. H., ... Davis, K. D. (2016). Frontotemporal correlates of impulsivity and machine learning in retired professional athletes with a history of multiple concussions. *Brain Structure & Function*, 221(4), 1911–1925. doi:10.1007/s00429-015-1012-0
- Hampshire, A., MacDonald, A., & Owen, A. M. (2013). Hypoconnectivity and hyperfrontality in retired American football players. *Scientific Reports*, 3, 2972. doi:10.1038/srep02972
- Hart, J., Jr., Kraut, M. A., Womack, K. B., Strain, J., Didehbani, N., Bartz, E., ... Cullum, C. M. (2013). Neuroimaging of cognitive dysfunction and depression in aging retired National Football League players: a cross-sectional study. *JAMA Neurology*, 70(3), 326–335. doi:10.1001/2013.jamaneurol.340
- Iverson, G. L., Keene, C. D., Perry, G., & Castellani, R. J. (2017). The need to separate chronic traumatic encephalopathy neuropathology from clinical features. *Journal of Alzheimer's Disease*, 61(1), 17–28. doi:10.3233/jad-170654
- Jordan, B. D. (2013). The clinical spectrum of sport-related traumatic brain injury. *Nature Reviews. Neurology*, 9(4), 222–230. doi:10.1038/nrneurol.2013.33
- Koerte, I. K., Hufschmidt, J., Muehlmann, M., Tripodis, Y., Stamm, J. M., Pasternak, O., ... Shenton, M. E. (2016). Cavum septi pellucidi in symptomatic former professional football players. *Journal of Neurotrauma*, 33(4), 346–353. doi:10.1089/neu.2015.3880
- Lin, A. P., Ramadan, S., Stern, R. A., Box, H. C., Nowinski, C. J., Ross, B. D., & Mountford, C. E. (2015). Changes in the neurochemistry of athletes with repetitive brain trauma: Preliminary results using localized correlated spectroscopy. *Alzheimer's Research & Therapy*, 7(1), 13. doi:10.1186/s13195-015-0094-5
- Ling, H., Morris, H. R., Neal, J. W., Lees, A. J., Hardy, J., Holton, J. L., ... Williams, D. D. (2017). Mixed pathologies including chronic traumatic encephalopathy account for dementia in Retired Association Football (Soccer) players. *Acta Neuropathologica*, 133(3), 337–352. doi:10.1007/s00401-017-1680-3
- Martland, H. S. (1928). Punch drunk. *JAMA*, 91(15), 1103–1107.

- McKee, A. C., Cairns, N. J., Dickson, D. W., Folkerth, R. D., Keene, C. D., Litvan, I., ... Gordon, W. A. (2016). The first NINDS/NIBIB consensus meeting to define neuropathological criteria for the diagnosis of chronic traumatic encephalopathy. *Acta Neuropathologica*, 131(1), 75–86. doi:10.1007/s00401-015-1515-z
- McKee, A. C., Stern, R. A., Nowinski, C. J., Stein, T. D., Daneshvar, D. H., Alvarez, V. E., ... Cantu, R. C. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain*, 136(Pt 1), 43–64. doi:10.1093/brain/aws307
- Mez, J., Daneshvar, D. H., Kiernan, P. T., Abdolmohammadi, B., Alvarez, V. E., Huber, B. R., ... AC, M. K. (2017). Clinicopathological evaluation of chronic traumatic encephalopathy in players of American football. *JAMA*, 318(4), 360–370. doi:10.1001/jama.2017.8334
- Mez, J., Solomon, T. M., Daneshvar, D. H., Murphy, L., Kiernan, P. T., Montenigro, P. H., ... McKee, A. C. (2015). Assessing clinicopathological correlation in chronic traumatic encephalopathy: Rationale and methods for the UNITE study. *Alzheimer's Research & Therapy*, 7(1), 62. doi:10.1186/s13195-015-0148-8
- Millsbaugh, J. A. (1937). Dementia pugilistica. *United States Naval Medicine Bulletin*, 35, 297–303.
- Mitsis, E. M., Riggio, S., Kostakoglu, L., Dickstein, D. L., Machac, J., Delman, B., ... Gandy, S. (2014). Tauopathy PET and amyloid PET in the diagnosis of chronic traumatic encephalopathies: Studies of a retired NFL player and of a man with FTD and a severe head injury. *Translational Psychiatry*, 4, e441. doi:10.1038/tp.2014.91
- Montenigro, P. H., Alosco, M. L., Martin, B. M., Daneshvar, D. H., Mez, J., Chaisson, C. E., ... Tripodis, Y. (2016). Cumulative head impact exposure predicts later-life depression, apathy, executive dysfunction, and cognitive impairment in former high school and college football players. *Journal of Neurotrauma*. doi:10.1089/neu.2016.4413
- Montenigro, P. H., Baugh, C. M., Daneshvar, D. H., Mez, J., Budson, A. E., Au, R., ... Stern, R. A. (2014). Clinical subtypes of chronic traumatic encephalopathy: Literature review and proposed research diagnostic criteria for traumatic encephalopathy syndrome. *Alzheimer's Research & Therapy*, 6(5), 68. doi:10.1186/s13195-014-0068-z
- Olsson, B., Lautner, R., Andreasson, U., Öhrfelt, A., Portelius, E., Bjerke, M., ... Zetterberg, H. (2016). CSF and blood biomarkers for the diagnosis of Alzheimer's disease: A systematic review and meta-analysis. *Lancet Neurology*, 15(7), 673–684. doi:10.1016/S1474-4422(16)00070-3
- Omalu, B. I., DeKosky, S. T., Minster, R. L., Kambh, M. I., Hamilton, R. L., & Wecht, C. H. (2005). Chronic traumatic encephalopathy in a National Football League player. *Neurosurgery*, 57(1), 128–134. discussion 128–134
- Schultz, V., Stern, R. A., Tripodis, Y., Stamm, J., Wrobel, P., Lepage, C., ... Koerte, I. K. (2017). Age at first exposure to repetitive head impacts is associated with smaller thalamic volumes in former professional American football players. *Journal of Neurotrauma*, 34, 1–8. doi:10.1089/neu.2017.5145
- Small, G. W., Kepe, V., Siddarth, P., Ercoli, L. M., Merrill, D. A., Donoghue, N., ... Barrio, J. R. (2013). PET scanning of brain tau in retired national football league players: Preliminary findings. *The American Journal of Geriatric Psychiatry*, 21(2), 138–144. doi:10.1016/j.jagp.2012.11.019
- Stamm, J. M., Bourlas, A. P., Baugh, C. M., Fritts, N. G., Daneshvar, D. H., Martin, B. M., ... Stern, R. A. (2015). Age of first exposure to football and later-life cognitive impairment in former NFL players. *Neurology*, 84(11), 1114–1120. doi:10.1212/WNL.0000000000001358
- Stamm, J. M., Koerte, I. K., Muehlmann, M., Pasternak, O., Bourlas, A. P., Baugh, C. M., ... Shenton, M. E. (2015). Age at first exposure to football is associated with altered corpus callosum white matter microstructure in former professional football players. *Journal of Neurotrauma*, 32(22), 1768–1776. doi:10.1089/neu.2014.3822
- Stein, T. D., Alvarez, V. E., & McKee, A. C. (2015). Concussion in chronic traumatic encephalopathy. *Current Pain and Headache Reports*, 19(10), 47. doi:10.1007/s11916-015-0522-z
- Stein, T. D., Montenigro, P. H., Alvarez, V. E., Xia, W., Crary, J. F., Tripodis, Y., ... McKee, A. C. (2015). Beta-amyloid deposition in chronic traumatic encephalopathy. *Acta Neuropathologica*, 130(1), 21–34. doi:10.1007/s00401-015-1435-y

- Stern, R. A., Daneshvar, D. H., Baugh, C. M., Seichepine, D. R., Montenegro, P. H., Riley, D. O., ... McKee, A. C. (2013). Clinical presentation of chronic traumatic encephalopathy. *Neurology*, 81(13), 1122–1129. doi:10.1212/WNL.0b013e3182a55f7f
- Stern, R. A., Tripodis, Y., Baugh, C. M., Fritts, N. G., Martin, B. M., Chaisson, C., ... Taylor, D. D. (2016). Preliminary study of plasma exosomal tau as a potential biomarker for chronic traumatic encephalopathy. *Journal of Alzheimer's Disease*, 51(4), 1099–1109. doi:10.3233/JAD-151028
- Strain, J., Didehbani, N., Cullum, C. M., Mansinghani, S., Conover, H., Kraut, M. A., ... Womack, K. B. (2013). Depressive symptoms and white matter dysfunction in retired NFL players with concussion history. *Neurology*, 81(1), 25–32. doi:10.1212/WNL.0b013e318299ccf8
- Strain, J. F., Womack, K. B., Didehbani, N., Spence, J. S., Conover, H., Hart, J., ... Cullum, C. M. (2015). Imaging correlates of memory and concussion history in retired National Football League Athletes. *JAMA Neurology*, 72(7), 773–780. doi:10.1001/jamaneurol.2015.0206
- Terry, D. P., Adams, T. E., Ferrara, M. S., & Miller, L. S. (2015). fMRI hypoactivation during verbal learning and memory in former high school football players with multiple concussions. *Archives of Clinical Neuropsychology*, 30(4), 341–355. doi:10.1093/arclin/acv020
- Victoroff, J. (2013). Traumatic encephalopathy: Review and provisional research diagnostic criteria. *NeuroRehabilitation*, 32(2), 211–224. doi:10.3233/NRE-130839

Suggested Reading

- Mahar, I., Alosco, M. L., & McKee, A. C. (2017). Psychiatric phenotypes in chronic traumatic encephalopathy. *Neuroscience and Biobehavioral Reviews*, 83, 622–630. doi:10.1016/j.neubiorev.2017.08.023
- McKee, A. C., Cairns, N. J., Dickson, D. W., Folkerth, R. D., Dirk Keene, C., Litvan, I., ... Bieniek, K. F. (2016). The first NINDS/NIBIB consensus meeting to define neuropathological criteria for the diagnosis of chronic traumatic encephalopathy. *Acta Neuropathologica*, 131(1), 75–86. doi:10.1007/s00401-015-1515-z
- Mez, J., Daneshvar, D. H., Kiernan, P. T., Abdolmohammadi, B., Alvarez, V. E., Huber, B. R., ... McKee, A. C. (2017). Clinicopathological evaluation of chronic traumatic encephalopathy in players of American football. *JAMA*, 318(4), 360–370. doi:10.1001/jama.2017.8334
- Montenegro, P. H., Baugh, C. M., Daneshvar, D. H., Mez, J., Budson, A. E., Au, R., ... Stern, R. A. (2014). Clinical subtypes of chronic traumatic encephalopathy: Literature review and proposed research diagnostic criteria for traumatic encephalopathy syndrome. *Alzheimer's Research & Therapy*, 6(5), 68. doi:10.1186/s13195-014-0068-z
- Stern, R. A., Daneshvar, D. H., Baugh, C. M., Seichepine, D. R., Montenegro, P. H., Riley, D. O., ... McKee, A. C. (2013). Clinical presentation of chronic traumatic encephalopathy. *Neurology*, 81(13), 1122–1129. doi:10.1212/WNL.0b013e3182a55f7f

Neuropsychological Assessment of Cortical and Subcortical Cognitive Phenotypes

Robert H. Paul^{1,2}, Carissa L. Philippi¹, and Gregory Dahl¹

¹*Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA*

²*Missouri Institute of Mental Health, St. Louis, MO, USA*

Introduction

For more than a century, neuropsychological assessment has provided an objective method to quantify brain integrity. This approach overcomes limitations of subjective report of cognitive status, which are vulnerable to bias. The severity of cognitive impairment is often inflated in the context of concurrent mood disruption. By contrast, individuals with neurological brain injury are prone to underestimate the severity of cognitive impairment. Lack of insight into cognitive impairment is frequently associated with Alzheimer's disease (AD), yet the feature is neither universal nor specific to the neuropathogenic mechanisms of AD. For example, recent studies indicate poor self-awareness of cognitive impairment among younger adults infected with human immunodeficiency virus (HIV). The inherent limitations of self-reported cognitive function underscore the importance of objectively defined assessment regardless of the underlying disease or mental health condition.

Anatomical Substrates of Neuropsychological Performance

The field of neuropsychology is based on the central tenet that biology and behavior are intertwined and inseparable. These relationships were established through empirical studies of individuals with natural and iatrogenic brain damage (Kolb & Wishaw, 2015). More recently, neuroimaging using high-resolution magnetic resonance imaging (MRI) and positron emission tomography (PET) allows *in vivo* examination of brain–behavior links. Advanced imaging

techniques such as MRI and PET (among other modalities) have revolutionized the brain sciences, yet neuroimaging studies neither define nor reliably predict functional capacity. Reliable estimates of the functional integrity of the underlying anatomy require a comprehensive approach to cognitive testing using measures sensitive to the primary networks of the brain (Yeo, Krienen, Chee, & Buckner, 2014). Inconsistencies exist in the literature, but most groups agree that these networks can be defined as five to seven relatively unique cognitive domains (attention, information processing speed/psychomotor speed, working memory/executive function, language, learning and memory, visuospatial, and motor). Analyses of the behavioral patterns observed within and across these domains produce rich insights into brain structure and function. This process is referred to as cognitive phenotyping.

Cognitive testing alone cannot reliably determine the etiology of isolated neuronal damage. However, the sensitivity of cognitive phenotyping to detect abnormalities in regional or brain-wide networks is quite good. Neuronal disruption that is largely confined to the cellular layers of the cortical ribbon is expressed behaviorally as deficit driven by the affected cortical region (e.g., language, memory, and motor). By contrast, neuronal damage predominately affecting the subcortical white matter fasciculi or the deep subcortical gray matter nuclei (caudate, globus pallidus, putamen, thalamus) produces a pattern of deficits most consistent with frontal lobe dysfunction (Cummings, 1993). Such anatomical specificity for associated cognitive impairment is most likely to be observed during the early stages of neurodegenerative conditions. By contrast, advanced stages of neurological conditions typically involve both cortical and subcortical brain regions and therefore produce a mixed pattern of cognitive symptoms.

Cortical Versus Subcortical Cognitive Phenotypes

The cortical cognitive phenotype includes amnesic memory loss, apraxia, agnosia, and anomia. This pattern was originally modeled after the prototypical expression of AD. Processing speed, working memory, and executive function are also affected in AD, but the level of impairment is typically less severe than the early and profound impairment in memory retention. Other neurodegenerative conditions that produce a cortical cognitive phenotype include semantic dementia, argyrophilic grain disease, and posterior cortical atrophy (Benson, Davis, & Snyder, 1988; Ferrer, Santpere, & van Leeuwen, 2008; Hodges, Patterson, Oxbury, & Funnell, 1992). The exact nature of the accompanying phenotype differs in these conditions, yet each shares a predilection for early involvement of cortical gray matter regions and a cognitive signature consistent with at least one cortical syndrome. An exception to this model can be seen among individuals with the frontal variant of frontotemporal dementia (FTD). Executive dysfunction is present early in FTD, and the behavioral deficits associated with poor behavioral regulation dominate the clinical presentation. Individuals under 65 with a marked change in personality and self-regulation are likely to meet diagnostic criteria for FTD.

In contrast to the cortical phenotype, subcortical cognitive phenotypes are characterized by abnormalities in attention, working memory, executive function, information processing, and psychomotor speed. This pattern reflects a predilection for disease mechanisms to impact deep subcortical gray matter nuclei (e.g., small vessel ischemia), white matter fasciculi (e.g., multiple sclerosis [MS]), or both gray and white matter regions (HIV) (Cummings, 1993; Rao, 1986; Woods, Moore, Weber, & Grant, 2009). Parkinson's disease, Huntington's disease, and "normal aging" also produce a cognitive pattern consistent with a subcortical profile. Neuropathology may exist in cortical regions (e.g., Parkinson's disease) or in gray-white matter junctions (MS), but the preponderance of cellular damage is concentrated in white

matter tracts and/or the basal ganglia. An interesting example of disconnection is evident among individuals with damage to the cerebellum (e.g., from stroke). The cerebellum is connected to the dorsolateral prefrontal cortex by a long-range fasciculus, and when damaged, affected individuals exhibit significant executive dysfunction (Strick, Dum, & Fiez, 2009). These cases of distant/remote brain damage provide clinical evidence that the behavioral expression of frontal impairment results from damage to the subcortical neural pathways.

Individuals with a subcortical cognitive phenotype often exhibit memory impairment, but the profile differs from what is observed in AD. Subcortical memory dysfunction is characterized by shallow learning and poor delayed recall of newly learned information. The restricted learning curve is a downstream consequence of poor attention, working memory, and/or executive skills needed to organize and learn the information. Long-term free recall is reduced as a function of the poor initial learning, but individuals usually do not exhibit a total loss of information. As such, subcortical etiologies spare recognition memory (discrimination of targets from foils) except when performance is confounded by positive or negative response sets (e.g., cases of stimulus-bound individuals). By contrast, individuals with AD do not benefit from recognition trials because the critical brain structures necessary for encoding and retention are damaged (i.e., medial temporal lobe). The ability to detect these unique memory phenotypes, therefore, is dependent on the specific memory measures included in the test battery. Memory tests that do not include a recognition trial will not differentiate retrieval from retention deficits.

Limitations to Phenotype Modeling

Neuropsychological phenotyping is not a perfect science. As described in the example of memory profiles above, the pattern of results is highly influenced by test selection (sensitivity, coverage), limited time windows to complete testing (e.g., when embedded in a demanding research study), access to appropriate normative data, and availability of alternate and equivalent forms for repeat testing (Kolb & Wishaw, 2015; Lezak, Howieson, Bigler, & Tranel, 2012). Additionally, stimulus confusion resulting from the administration of multiple tests can artificially create behavioral failures due to poor test administration procedures. A related problem is confusion that occurs when language measures are administered during the interval between final learning trials and delayed recall trials on verbal memory tests. These details are not intuitive, yet the quality of data available for phenotyping is intimately linked to sound implementation procedures. Unfortunately, efforts to fully standardize the process (e.g., NIH Toolbox; Gershon et al., 2013) have not been widely adopted, largely because the value of such an approach remains undefined. We expect change in the future, with more ready uptake of standardized cognitive assessments and application of predictive modeling using artificial intelligence algorithms (e.g., machine learning).

Opportunities for Future Research in Cognitive Phenotyping

Neuropsychological tests generate many indices of brain function. Depending on final test selections, the total number of dependent variables obtained from a comprehensive evaluation can easily exceed 50 or more indices. Structural and functional neuroimaging assessments generate many more variables of interest. Aggregating these outcomes into global measures by cognitive domain (e.g., executive function, information processing) or brain region (frontal lobe volume, white matter volume) to reduce the total number of dependent variables reduces

the sensitivity of the outcome measures as areas of relative weakness are masked by relative strengths. An alternative approach is to simply ignore select data outputs based on a priori hypotheses related to regions believed to be relevant to the clinical or research question. This approach avoids reduced and restricted sensitivity due to averaging, but the outcomes are entirely dependent on the accuracy of the a priori hypotheses. It can be argued that this traditional data-reduction approach to clinical neuroscience has contributed to the slow pace of innovation in the field. Thus, a new approach is greatly needed.

Artificial intelligence systems adapted from private industry offer a fresh approach. Rather than intentional omission of potentially important indices of brain function, artificial intelligence incorporates all data input features into models to define the latent variance structure. Groups, including ours, apply these methods to integrate multimodal neuroimaging data, comprehensive neuropsychological performance indices, and laboratory markers, without aggregation of scores by cognitive domains or brain regions and without discarding data based on assumptions. Work conducted to date clearly defines that value of this approach to define the most salient variables that define group classification, as well as inform which variables can be adjusted to reduce risks associated with specific conditions. It is expected that data-driven approaches using artificial intelligence algorithms will transform the field and establish new paths for personalized medicine, inclusive of treatment and cure strategies across a broad spectrum of neurological conditions.

Author Biographies

Robert H. Paul is a board-certified clinical neuropsychologist. He serves as director of the Missouri Institute of Mental Health and professor of psychological sciences at the University of Missouri–St. Louis. His research program is focused on mechanisms of brain dysfunction in health conditions that primarily impact brain structures located deep beneath the surface of the cerebral cortex. He has special interest in clinical neuroscience capacity building in resource-limited environments.

Carissa L. Philippi, PhD, is an assistant professor in the Department of Psychological Sciences at the University of Missouri–St. Louis. Her research takes a dimensional approach to understanding the behavioral (e.g., negative self-related thought) and neural correlates of neuropsychiatric conditions, such as depression. For example, in a recent study, she identified associations between subclinical depression severity and resting-state functional connectivity of the anterior cingulate cortex that mirrored neural circuit dysfunction in major depression.

Gregory Dahl is a graduate research assistant in the Behavioral Neuroscience Program at the University of Missouri–St. Louis. He completed his undergraduate work in Psychology and Philosophy in May of 2015 and began working with client data/statistics for a not-for-profit mental health clinic. Upon entry to graduate school, he began working with Professor Philippi on the cognitive and affective neurobiology of self. His research interests include the neurobiology of memory and the cognitive/behavioral/environmental factors that can affect memory.

References

- Benson, D., Davis, R., & Snyder, B. (1988). Posterior cortical atrophy. *Archives of Neurology*, 45(7), 789–793. doi:10.1001/archneur.1988.00520310107024
- Cummings, J. (1993). Frontal-subcortical circuits and human behavior. *Archives of Neurology*, 50(8), 873–880. doi:10.1001/archneur.1993.00540080076020

- Ferrer, I., Santpere, G., & van Leeuwen, F. W. (2008). Argrophilic grain disease. *Brain*, 131(6), 1416–1432. doi:10.1093/brain/awm305
- Gershon, R. C., Wagster, M. V., Hendrie, H. C., Fox, N. A., Cook, K. F., & Nowinski, C. J. (2013). NIH toolbox for assessment of neurological and behavioral function. *Neurology*, 80(11 Supplement 3), S2–S6. doi:10.1212/WNL.0b013e3182872e5f
- Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992). Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain*, 115(6), 1783–1806. doi:10.1093/brain/115.6.1783
- Kolb, B., & Wishaw, I. (2015). *Fundamentals of human neuropsychology* (7th ed.). New York, NY: Worth Publishers.
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological assessment*. New York, NY: Oxford University Press.
- Rao, S. M. (1986). Neuropsychology of multiple sclerosis: A critical review. *Journal of Clinical and Experimental Neuropsychology*, 8(5), 503–542. doi:10.1080/01688638608405173
- Strick, P. L., Dum, R. P., & Fiez, J. A. (2009). Cerebellum and nonmotor function. *Annual Review of Neuroscience*, 32(1), 413–434. doi:10.1146/annurev.neuro.31.060407.125606
- Woods, S. P., Moore, D. J., Weber, E., & Grant, I. (2009). Cognitive neuropsychology of HIV-associated neurocognitive disorders. *Neuropsychology Review*, 19(2), 152–168. doi:10.1007/s11065-009-9102-5
- Yeo, B. T. T., Krienen, F. M., Chee, M. W. L., & Buckner, R. L. (2014). Estimates of segregation and overlap of functional connectivity networks in the human cerebral cortex. *NeuroImage*, 88, 212–227. doi:10.1016/j.neuroimage.2013.10.046

Mild Traumatic Brain Injury and Posttraumatic Stress Disorder: Diagnostic and Neurobiological Signatures of Comorbidity

Jacob Bolzenius

Missouri Institute of Mental Health, University of Missouri–St. Louis, St. Louis, MO, USA

Traumatic brain injury (TBI) is one of the most common conditions of acquired brain dysfunction, with estimates reporting 1.7 million cases per year in the United States alone (Faul & Coronado, 2015). While treatment for and survivability from more severe TBI has improved markedly over recent decades, mild TBI (mTBI), also commonly described as “concussion,” presents a fairly unique symptom profile and recovery prognosis. Moreover, prevalence of this condition in certain stressful scenarios (e.g., military service, assault, motor vehicle accidents) leaves the individual prone to psychiatric disorders such as posttraumatic stress disorder (PTSD). This commonly comorbid disorder challenges clinical judgment and treatment strategies, particularly as patients progress past the acute injury phase. Optimized methods for identification of mTBI and PTSD, especially when presenting together, have thus been the subject of intense investigation over recent years. This chapter aims to delineate characteristics of both mTBI and PTSD and review evidence regarding the neurobiological bases of these disorders that may guide clinical care decisions.

Diagnosis and Characteristics of mTBI

Severity of TBI is classified as mild, moderate, or severe based on several criteria, including loss of consciousness (LOC), alteration of consciousness or posttraumatic amnesia (PTA), Glasgow Coma Scale (GCS) score, level of care or hospitalization required, and presence of acute neuroimaging abnormalities if available (Department of Veterans Affairs/Department of

Defense, 2016). mTBI is defined as a GCS of 13–15, LOC less than 30 min, and alteration of consciousness or PTA less than 24 hr. Most cases have unremarkable neuroimaging findings (i.e., CT or MRI) and require no significant hospitalization or prolonged care. The accuracy of these criteria and immediate care decisions, however, are largely dependent on self-report as well as information from collateral sources, if present. The most common causes of civilian mTBI include falls and motor vehicle accidents (Bazarian et al., 2009). Among military populations in modern conflicts, blast injuries or blast-related injuries represent the most common cause of mTBI (Helmick et al., 2015; Terrio et al., 2009).

Unlike moderate to severe TBI, which leads to marked and prolonged cognitive deficits, cognitive and behavioral difficulties from mTBI tend to resolve within a couple weeks. Meta-analyses have suggested that reductions in cognitive function are evident in the acute phase of mTBI, particularly in memory, fluency, and processing speed. However, these changes are dependent on the post-injury interval and tend to resolve with time, usually returning to near average within about 90 days (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Frencham, Fox, & Maybery, 2007; Schretlen & Shapiro, 2003). Injury-related litigation has been identified as one key factor to residual or worsening cognitive performance beyond this time point (Belanger et al., 2005). While the majority of individuals with mTBI fully recover, around 10–15% continue to experience symptoms past the 3-month window related to the injury. These chronic symptoms are known as post-concussion syndrome (PCS) (Iverson & Lange, 2011), recently reclassified as *major or mild neurocognitive disorder due to TBI* in the *5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* (American Psychiatric Association, 2013). Symptoms of PCS may take weeks to months to emerge following mTBI and can include a host of secondary effects, including orthopedic pain, headaches, psychiatric conditions, and sensory dysfunction (Leddy, Sandhu, Sodhi, Baker, & Willer, 2012). Treatment options include a variety of pharmacologic, physical, and psychological methods, with evidence supporting the use of early, brief psychological intervention including symptom attribution education, complemented by resumption of physical activity and medication when appropriate (Mittenberg, Canyock, Condit, & Patton, 2010).

Work is ongoing to identify biomarkers that can detect biological correlates of continued cognitive or subjective symptoms in these individuals. Volumetric MRI studies have described cortical thinning (A. P. Michael et al., 2015) and global and focal atrophy to both white and gray matter at 1-year post-injury that relates to cognitive changes and PCS symptoms (Zhou et al., 2013). Diffusion tensor imaging (DTI) has demonstrated sensitivity to sequelae of mTBI and ability to distinguish patients from controls (Hulkower, Poliak, Rosenbaum, Zimmerman, & Lipton, 2013). The advantage of DTI resides in the sensitivity to microstructural detail of neuronal components that are affected by the shearing forces (e.g., traumatic axonal injury) sustained during mTBI, particularly to white matter. As a result, DTI has become the neuroimaging method of choice for examining brain markers of mTBI, especially as this scan sequence is already commonly available on most commercial MRI scanners. Research has suggested that DTI may be more sensitive than cognitive testing to subacute mTBI changes and that these changes normalize longitudinally as injury-related physiological disruption normalizes (Mayer et al., 2010). In individuals with symptoms of PCS, DTI abnormalities in the subacute stage after mTBI have been related to greater white matter structural impairment at 6-month follow-up in patients compared with non-PCS controls (Messé et al., 2012). This is supported by animal research confirming the link between DTI changes and altered neuronal histopathology in mTBI models (Bennett, Mac Donald, & Brody, 2012; Hylin et al., 2013). Additional structural MRI sequences such as susceptibility-weighted imaging (SWI) and fluid-attenuated inversion recovery (FLAIR) provide help detecting

micro-hemorrhages, and together usage of SWI and/or DTI detects mTBI abnormalities in up to one-third of those with negative CT scans on day of injury (Bigler, 2013; Shenton et al., 2012). Functional MRI (fMRI) sequences have also shown promise in differentiating mTBI from controls (Bryer, Medaglia, Rostami, & Hillary, 2013), suggesting that functional network integrity can be leveraged as a marker of both acute and chronic mTBI.

Diagnosis and Characteristics of PTSD

PTSD is a disorder characterized by emotional, behavioral, and cognitive symptoms that develops in response to a severe stressor or threat to life or well-being. Some of the most common clinical assessment tools for PTSD updated for *DSM-5* criteria include the Clinician-Administered PTSD Scale (CAPS-5) (Weathers et al., 2013), the PTSD Symptom Scale—Interview (PSS-I-5) (Foa et al., 2013), and the Structured Clinical Interview for *DSM-5* (SCID-5) (First, Williams, Karg, & Spitzer, 2015). Symptoms of PTSD are grouped diagnostically among several criteria clusters. These include reexperiencing or intrusive thoughts, avoidance behaviors, negative mood or altered cognitive ability, and arousal or reactivity. These diagnostic clusters reflect a change implemented in the *DSM-5*, which separated avoidance and negative alterations in cognition and mood, thus requiring identifiable presence of both of these symptoms for diagnosis. Symptoms can extend well beyond the acute recovery period and lead to continued cognitive difficulties, observable brain abnormalities, and altered quality of life and disability (Seedat, Lochner, Vythilingum, & Stein, 2006). PTSD is especially prevalent among military veterans, presenting in up to 20% of those returning from service (Hoge et al., 2004), and is associated with poor post-deployment health and occupational outcomes compared with non-symptomatic counterparts (Hoge et al., 2008).

While PTSD is generally viewed and treated as a disorder of fear conditioning dysregulation, it is also characterized by changes to objective neuropsychological performance. Individuals with PTSD exhibit greater attention to, and more difficulty disengaging from, emotionally laden stimuli (Christianson, 1992; Paunovi, Lundh, & Ost, 2002; Vythilingam et al., 2007). PTSD has been linked to long-term changes in several cognitive domains such as verbal and visual memory compared with controls (Johnsen & Asbjørnsen, 2008; Vasterling, Aslan, et al., 2018). Further, detriments in areas of executive function are shown to affect memory formation and retrieval for both neutral and emotionally laden content (LaGarde, Doyon, & Brunet, 2010; Williams et al., 2007). In addition to these various types of explicit memory, implicit memory shows differential effects in PTSD, with evidence of facilitated priming for high valence stimuli compared with controls (Kleim, Ehring, & Ehlers, 2011; T. Michael & Ehlers, 2007; T. Michael, Ehlers, & Halligan, 2005). Attention bias to trauma-related words in an emotional Stroop task relates to a diagnosis of PTSD (El Khoury-Malhame et al., 2011) as well as PTSD severity (Fleurkens, Rinck, & van Minnen, 2011). Additionally, these individuals exhibit impaired reward processing as effort expenditure to reach a positive reward varies greatly from patterns demonstrated by controls (Hopper et al., 2007). Thus, there is sufficient evidence substantiating the effects of altered cognitive functions, particularly as they relate to emotionally laden content.

PTSD disrupts normal function of the hypothalamic–pituitary–adrenal axis. These are marked by decreased cortisol and increased levels of corticotropin-releasing hormone that create a sensitized negative feedback inhibitory loop (Sherin & Nemeroff, 2011). To identify the neurocircuitry behind the disorder, prior work has demonstrated links to various markers of brain volume, integrity, and network connectivity. This includes disruptions in frontal

networks necessary for inhibiting vigilance to trauma-related stimuli and extinguishing response patterns, overactive amygdala function that facilitates the learned and habituated aspects of the fear response, and decreased hippocampal activation leading to impaired association with and retention of less emotionally volatile information (Rauch, Shin, & Phelps, 2006). Volumetric MRI studies have related PTSD to smaller volumes in the hippocampus (Smith, 2005; Villarreal et al., 2002), anterior cingulate cortex (Kitayama & Quinn, 2006; Woodward et al., 2006), and amygdala (Karl et al., 2006) compared with healthy controls. DTI research has demonstrated consistent white matter disruptions associated with PTSD, particularly in tracts related to emotional regulation (Long et al., 2013; Schuff et al., 2011). Functional neuroimaging, including PET and fMRI, have also provided evidence for network disruption and altered blood flow patterns in response to emotional stimuli in individuals with PTSD (Bremner et al., 1999; Lanius et al., 2003; Patel, Spreng, Shin, & Girard, 2012). Elevated activation of the amygdala and hippocampus has been observed using fMRI while encoding and recalling negative stimuli in PTSD relative to trauma-exposed non-PTSD individuals (Brohawn, Offringa, Pfaff, Hughes, & Shin, 2010).

Characteristics of Comorbid mTBI and PTSD

Overlapping presentation of symptoms represents one of the chief challenges of comorbid PTSD and mTBI. Clinical determination of differential diagnosis is challenged by the need to rely on self-reported symptom complaints. Acute injury details may be irretrievable due to memory loss related to the traumatic event, making attribution of deficits ambiguous between sequelae of psychological and brain trauma. Both disorders share chronic symptom complaints including insomnia, slowed or foggy thinking, headache, and difficulties with attention, and additional comorbidities may arise including depression, substance use, and somatic problems. Due to variations in injury-related factors such as loss or alteration of consciousness that impact the subjective experience of the event, individuals with mTBI tend to be at higher risk for development of PTSD than more severe head injuries (Klein, Caspi, & Gil, 2016).

Military populations in particular are at elevated risk for exposure to trauma resulting in mTBI and PTSD. A large-scale study demonstrated a threefold increase in the rate of PTSD among veterans exhibiting positive symptoms of TBI compared with those exhibiting no symptoms (Carlson et al., 2010). Studies focusing on mTBI have shown that a significant portion of service members (up to 40% or higher) experiencing symptoms of mTBI also exhibit symptoms of PTSD (Hoge et al., 2008). These elevated rates are likely due to combat environments creating pre-injury states of heightened stress and hypervigilance that expose the individual to PTSD-inducing stimuli prior to onset of brain trauma (Kennedy et al., 2007). Additionally, variations in psychiatric profiles, including higher premorbid PTSD symptoms and lower cognitive function, military combat, and post-deployment stress, predict PTSD development following trauma exposure (Boasso, Steenkamp, Larson, & Litz, 2016; Sørensen, Andersen, Karstoft, & Madsen, 2016). Disentangling these disorder profiles and identifying optimal treatment regimens remain a critical goal, as history of TBI is a prominent risk factor for suicidal ideation (Wisco et al., 2014) and nearly two dozen US veterans die from suicide each day (Department of Veterans Affairs, 2016).

Research shows that presence of PTSD can impact the presentation and severity of the mTBI and vice versa. TBI and PTSD are both independently related to subjective post-injury symptom reporting (Vasterling et al., 2012). Symptoms of PCS in particular overlap notably with those of PTSD; thus treatment of some nonspecific symptoms (e.g., anger, impaired

concentration) (Vasterling, Jacob, & Rasmussen, 2018) may improve outcomes of either condition. In the acute phase, symptoms of PTSD and mTBI (e.g., confusion, disrupted attention, disorientation) can resemble each other, and in the post-acute stage, PCS may be endorsed by individuals with non-trauma disorders (Donnell, Kim, Silva, & Vanderploeg, 2012) or even healthy controls (Iverson & Lange, 2010). Studies have described independent associations between both mTBI and PTSD with PCS, with evidence supporting an additive burden of adversity for those with both mTBI and PTSD (Brenner et al., 2010; Brenner, Vanderploeg, & Terrio, 2009). Data suggest that psychological distress related to PCS and PTSD are predictors of cognitive status regardless of TBI history, but history of mTBI is linked to higher rates of clinical morbidity (Donnelly, Donnelly, Warner, Kittleson, & King, 2017). Further, rates of PTSD are nearly twice as likely one year after mTBI compared with non-TBI civilian trauma (Bryant et al., 2010). Objective cognitive profiles are difficult to ascribe to one single condition, as both affect several of the same domains (e.g., executive function, learning, memory) (Scott et al., 2015), though some research suggests PTSD relates to poorer performance in cognitive domains of reaction time, learning, and memory after adjusting for TBI status (Vasterling et al., 2012). Subjective complaints regarding cognitive function often exceed measurable dysfunction (Spencer, Drag, Walker, & Bieliauskas, 2010; Zatzick et al., 2010).

Few neuroimaging studies have focused on both mTBI and PTSD. One study suggested that PTSD severity was more strongly related to measures of white matter integrity than mTBI, but subclinical symptoms of mTBI moderate the development of PTSD (Bazarian et al., 2013). Research has also shown that PTSD relates to white matter changes using DTI after accounting for mTBI status (Davenport et al., 2016; Schuff et al., 2011). Another study demonstrated greater white matter integrity in PTSD than mTBI, particularly in high-complexity regions (Davenport, Lim, & Sponheim, 2014), supporting the idea of mTBI as a disorder of diffuse brain disruption (Bolzenius et al., 2018; Hu et al., 2016). Other methods, such as single-photon emission computed tomography (SPECT), have revealed greater regional activation in PTSD compared to a mixed-severity TBI group, suggesting that PTSD symptoms in the presence of TBI produce a different signature than when considering either condition alone (Amen et al., 2015). fMRI research has also implicated altered connectivity between prefrontal and hippocampal regions that relates to disruptive reexperiencing of PTSD symptoms. Other studies report altered connectivity between prefrontal and basal ganglia regions, providing a potential neural substrate associated with abnormalities in working memory and maintenance networks that exacerbate PTSD symptoms (Spielberg, McGlinchey, Milberg, & Salat, 2015). Overall, these studies present intriguing interactions between symptom profiles of mTBI and PTSD, but additional research is needed to further tease apart the unique contributions of each condition. Advances in computational data science and predictive modeling algorithms based on artificial intelligence offers the potential to delineate the biology, symptoms, and treatments for PTSD and mTBI as separate entities.

Author Biography

Jacob D. Bolzenius earned a PhD (2014) in Psychology with an emphasis in behavioral neuroscience from the University of Missouri–St. Louis and has held several appointments, currently at the University of Missouri–St. Louis. His research has focused on using novel neuroimaging techniques, including diffusion tensor imaging and volumetric shape analysis, to assess biological correlates of brain disease or insult (e.g., traumatic brain injury, healthy

aging). Currently he is applying diffusion basis spectrum imaging methods to infer markers of cellular inflammation and neuronal integrity associated with delirium in older adults. This research has promise to validate *in vivo* cellular pathology associated with brain disease that can inform the understanding of brain disease progression in later life to apply to prevention and treatment strategies.

References

- Amen, D. G., Raji, C. A., Willeumier, K., Taylor, D., Tarzwell, R., Newberg, A., & Henderson, T. A. (2015). Functional neuroimaging distinguishes posttraumatic stress disorder from traumatic brain injury in focused and large community datasets. *PLoS One*, *10*(7), e0129659.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®), fifth edition*. Washington, DC: Author.
- Bazarian, J. J., Donnelly, K., Peterson, D. R., Warner, G. C., Zhu, T., & Zhong, J. (2013). The relation between posttraumatic stress disorder and mild traumatic brain injury acquired during operations enduring freedom and iraqi freedom. *The Journal of Head Trauma Rehabilitation*, *28*(1), 1–12.
- Bazarian, J. J., McClung, J., Shah, M. N., Cheng, Y. T., Flesher, W., & Kraus, J. (2009). Mild traumatic brain injury in the United States, 1998–2000. *Brain Injury*, *19*(2), 85–91.
- Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K., & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society*, *11*(3), 215–227.
- Bennett, R. E., Mac Donald, C. L., & Brody, D. L. (2012). Diffusion tensor imaging detects axonal injury in a mouse model of repetitive closed-skull traumatic brain injury. *Neuroscience Letters*, *513*(2), 160–165.
- Bigler, E. D. (2013). Neuroimaging biomarkers in mild traumatic brain injury (mTBI). *Neuropsychology Review*, *23*(3), 169–209.
- Boasso, A. M., Steenkamp, M. M., Larson, J. L., & Litz, B. T. (2016). Posttraumatic stress symptoms across the deployment cycle: A latent transition analysis. *Journal of Psychiatric Research*, *83*, 54–60.
- Bolzenius, J. D., Velez, C. S., Lewis, J. D., Bigler, E. D., Wade, B. S. C., Cooper, D. B., ... Tate, D. F. (2018). Diffusion imaging findings in US service members with mild traumatic brain injury and posttraumatic stress disorder. *The Journal of Head Trauma Rehabilitation*, *33*(6), 393–402.
- Bremner, J. D., Narayan, M., Staib, L. H., Southwick, S. M., McGlashan, T., & Charney, D. S. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *The American Journal of Psychiatry*, *156*(11), 1787–1795.
- Brenner, L. A., Terrio, H., Homaifar, B. Y., Gutierrez, P. M., Staves, P. J., Harwood, J. E., ... Warden, D. (2010). Neuropsychological test performance in soldiers with blast-related mild TBI. *Neuropsychology*, *24*(2), 160–167.
- Brenner, L. A., Vanderploeg, R. D., & Terrio, H. (2009). Assessment and diagnosis of mild traumatic brain injury, posttraumatic stress disorder, and other polytrauma conditions: Burden of adversity hypothesis. *Rehabilitation Psychology*, *54*(3), 239–246.
- Brohawn, K. H., Offringa, R., Pfaff, D. L., Hughes, K. C., & Shin, L. M. (2010). The neural correlates of emotional memory in posttraumatic stress disorder. *Biological Psychiatry*, *68*(11), 1023–1030.
- Bryant, R. A., O'Donnell, M. L., Creamer, M., McFarlane, A. C., Clark, C. R., & Silove, D. (2010). The psychiatric sequelae of traumatic injury. *The American Journal of Psychiatry*, *167*(3), 312–320.
- Bryer, E. J., Medaglia, J. D., Rostami, S., & Hillary, F. G. (2013). Neural recruitment after mild traumatic brain injury Is task dependent: A meta-analysis. *Journal of the International Neuropsychological Society*, *19*(7), 751–762.
- Carlson, K. F., Nelson, D., Orazem, R. J., Nugent, S., Cifu, D. X., & Sayer, N. A. (2010). Psychiatric diagnoses among Iraq and Afghanistan war veterans screened for deployment-related traumatic brain injury. *Journal of Traumatic Stress*, *23*(1), 17–24.

- Christianson, S. A. (1992). Emotional stress and eyewitness memory: A critical review. *Psychological Bulletin*, 112(2), 284–309.
- Davenport, N. D., Lamberty, G. J., Nelson, N. W., Lim, K. O., Armstrong, M. T., & Sponheim, S. R. (2016). PTSD confounds detection of compromised cerebral white matter integrity in military veterans reporting a history of mild traumatic brain injury. *Brain Injury*, 30(12), 1491–1500.
- Davenport, N. D., Lim, K. O., & Sponheim, S. R. (2014). White matter abnormalities associated with military PTSD in the context of blast TBI. *Human Brain Mapping*, 36(3), 1053–1064.
- Department of Veterans Affairs. (2016). *Suicide among veterans and other Americans 2001–2014*. Washington, DC: Office of Suicide Prevention.
- Department of Veterans Affairs/Department of Defense. (2016). VA/DoD clinical practice guideline for the management of concussion-mild traumatic brain injury. Retrieved from <https://www.healthquality.va.gov/guidelines/Rehab/mtbi/>.
- Donnell, A. J., Kim, M. S., Silva, M. A., & Vanderploeg, R. D. (2012). Incidence of postconcussion symptoms in psychiatric diagnostic groups, mild traumatic brain injury, and comorbid conditions. *The Clinical Neuropsychologist*, 26(7), 1092–1101.
- Donnelly, K., Donnelly, J. P., Warner, G. C., Kittleson, C. J., & King, P. R. (2017). Longitudinal study of objective and subjective cognitive performance and psychological distress in OEF/OIF Veterans with and without traumatic brain injury. *The Clinical Neuropsychologist*, 3, 1–20.
- El Khoury-Malhame, M., Lanteaume, L., Beetz, E. M., Roques, J., Reynaud, E., Samuelian, J. C., ... Khalfa, S. (2011). Attentional bias in post-traumatic stress disorder diminishes after symptom amelioration. *Behaviour Research and Therapy*, 49(11), 796–801.
- Faul, M., & Coronado, V. (2015). Epidemiology of traumatic brain injury. *Handbook of Clinical Neurology*, 127, 3–13.
- First, M. B., Williams, J. B. W., Karg, R. S., & Spitzer, R. L. (2015). *Structured clinical interview for DSM-5 disorders, clinician version (SCID-5-CV)*. Arlington, VA: American Psychiatric Association.
- Fleurkens, P., Rinck, M., & van Minnen, A. (2011). Specificity and generalization of attentional bias in sexual trauma victims suffering from posttraumatic stress disorder. *Journal of Anxiety Disorders*, 25(6), 783–787.
- Foa, E. B., McLean, C. P., Zang, Y., Zong, J., Rauch, S., Porter, K., ... Kauffman, B. (2013). Psychometric properties of the Posttraumatic Stress Disorder Symptoms Scale Interview for DSM-5 (PSSI-5). *Psychological Assessment*, 28, 1159–1165.
- Frencham, K. A. R., Fox, A. M., & Maybery, M. T. (2007). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology*, 27(3), 334–351.
- Helmick, K. M., Spells, C. A., Malik, S. Z., Davies, C. A., Marion, D. W., & Hinds, S. R. (2015). Traumatic brain injury in the US military: epidemiology and key clinical and research programs. *Brain Imaging and Behavior*, 9(3), 358–366.
- Hoge, C. W., Castro, C. A., Messer, S. C., McGurk, D., Cotting, D. I., & Koffman, R. L. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *NEJM*, 351, 13–22.
- Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. soldiers returning from Iraq. *NEJM*, 358(5), 453–463.
- Hopper, J. W., Pitman, R. K., Su, Z., Heyman, G. M., Lasko, N. B., Macklin, M. L., ... Elman, I. (2007). Probing reward function in posttraumatic stress disorder: Expectancy and satisfaction with monetary gains and losses. *Journal of Psychiatric Research*, 42(10), 802–807.
- Hu, H., Zhou, Y., Wang, Q., Su, S., Qiu, Y., Ge, J., ... Xiao, Z. (2016). Association of abnormal white matter integrity in the acute phase of motor vehicle accidents with post-traumatic stress disorder. *Journal of Affective Disorders*, 190, 714–722.
- Hulkower, M. B., Poliak, D. B., Rosenbaum, S. B., Zimmerman, M. E., & Lipton, M. L. (2013). A decade of DTI in traumatic brain injury: 10 Years and 100 articles later. *American Journal of Neuroradiology*, 34(11), 2064–2074.

- Hylin, M. J., Orsi, S. A., Zhao, J., Bockhorst, K., Perez, A., Moore, A. N., & Dash, P. K. (2013). Behavioral and histopathological alterations resulting from mild fluid percussion injury. *Journal of Neurotrauma*, 30(9), 702–715.
- Iverson, G. L., & Lange, R. T. (2010). Examination of “postconcussion-like” symptoms in a healthy sample. *Applied Neuropsychology*, 10(3), 137–144.
- Iverson, G. L., & Lange, R. T. (2011). Post-concussion syndrome. In M. Schoenberg & J. Scott (Eds.), *The little black book of neuropsychology* (pp. 745–764). Boston, MA: Springer.
- Johnsen, G. E., & Asbjørnsen, A. E. (2008). Consistent impaired verbal memory in PTSD: A meta-analysis. *Journal of Affective Disorders*, 111(1), 74–82.
- Karl, A., Schaefer, M., Malta, L. S., Dörfel, D., Rohleder, N., & Werner, A. (2006). A meta-analysis of structural brain abnormalities in PTSD. *Neuroscience and Biobehavioral Reviews*, 30(7), 1004–1031.
- Kennedy, J. E., Jaffee, M. S., Leskin, G. A., Stokes, J. W., Leal, F. O., & Fitzpatrick, P. J. (2007). Posttraumatic stress disorder and posttraumatic stress disorder-like symptoms and mild traumatic brain injury. *Journal of Rehabilitation Research and Development*, 44(7), 895–920.
- Kitayama, N., & Quinn, S. (2006). Smaller volume of anterior cingulate cortex in abuse-related post-traumatic stress disorder. *Journal of Affective Disorders*, 90(2-3), 171–174.
- Kleim, B., Ehring, T., & Ehlers, A. (2011). Perceptual processing advantages for trauma-related visual cues in post-traumatic stress disorder. *Psychological Medicine*, 42(1), 173–181.
- Klein, E., Caspi, Y., & Gil, S. (2016). The relation between memory of the traumatic event and PTSD: Evidence from studies of traumatic brain injury. *The Canadian Journal of Psychiatry*, 48(1), 28–33.
- LaGarde, G., Doyon, J., & Brunet, A. (2010). Memory and executive dysfunctions associated with acute posttraumatic stress disorder. *Psychiatry Research*, 177(1), 144–149.
- Lanius, R. A., Williamson, P. C., Hopper, J., Densmore, M., Boksman, K., Gupta, M. A., ... Menon, R. S. (2003). Recall of emotional states in posttraumatic stress disorder: An fMRI investigation. *Biological Psychiatry*, 53(3), 204–210.
- Leddy, J. J., Sandhu, H., Sodhi, V., Baker, J. G., & Willer, B. (2012). Rehabilitation of concussion and post-concussion syndrome. *Sports Health*, 4(2), 147–154.
- Long, Z., Duan, X., Xie, B., Du, H., Li, R., Xu, Q., ... Chen, H. (2013). Altered brain structural connectivity in post-traumatic stress disorder: A diffusion tensor imaging tractography study. *Journal of Affective Disorders*, 150(3), 798–806.
- Mayer, A. R., Ling, J., Mannell, M. V., Gasparovic, C., Phillips, J. P., Doezema, D., ... Yeo, R. A. (2010). A prospective diffusion tensor imaging study in mild traumatic brain injury. *Neurology*, 74(8), 643–650.
- Messé, A., Caplain, S., Péligrini-Issac, M., Blanche, S., Montreuil, M., Lévy, R., ... Benali, H. (2012). Structural integrity and postconcussion syndrome in mild traumatic brain injury patients. *Brain Imaging and Behavior*, 6(2), 283–292.
- Michael, A. P., Stout, J., Roskos, P. T., Bolzenius, J., Gfeller, J., Mogul, D., & Bucholz, R. (2015). Evaluation of cortical thickness after traumatic brain injury in military veterans. *Journal of Neurotrauma*, 32(22), 1751–1758.
- Michael, T., & Ehlers, A. (2007). Enhanced perceptual priming for neutral stimuli occurring in a traumatic context: Two experimental investigations. *Behaviour Research and Therapy*, 45(2), 341–358.
- Michael, T., Ehlers, A., & Halligan, S. L. (2005). Enhanced priming for trauma-related material in post-traumatic stress disorder. *Emotion (Washington, DC)*, 5(1), 103–112.
- Mittenberg, W., Canyock, E. M., Condit, D., & Patton, C. (2010). Treatment of post-concussion syndrome following mild head injury. *Journal of Clinical and Experimental Neuropsychology*, 23(6), 829–836.
- Patel, R., Spreng, R. N., Shin, L. M., & Girard, T. A. (2012). Neurocircuitry models of posttraumatic stress disorder and beyond: A meta-analysis of functional neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, 36(9), 2130–2142.

- Paunovi, N., Lundh, L. G., & Ost, L. G. (2002). Attentional and memory bias for emotional information in crime victims with acute posttraumatic stress disorder (PTSD). *Journal of Anxiety Disorders*, 16(6), 675–692.
- Rauch, S. L., Shin, L. M., & Phelps, E. A. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: Human neuroimaging research—Past, present, and future. *Biological Psychiatry*, 60(4), 376–382.
- Schretlen, D. J., & Shapiro, A. M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, 15(4), 341–349.
- Schuff, N., Zhang, Y., Zhan, W., Lenoci, M., Ching, C., Boreta, L., ... Neylan, T. C. (2011). Patterns of altered cortical perfusion and diminished subcortical integrity in posttraumatic stress disorder: An MRI study. *NeuroImage*, 54(S1), S62–S68.
- Scott, J. C., Matt, G. E., Wrocklage, K. M., Crnich, C., Jordan, J., Southwick, S. M., ... Schweinsburg, B. C. (2015). A quantitative meta-analysis of neurocognitive functioning in posttraumatic stress disorder. *Psychological Bulletin*, 141(1), 105–140.
- Seedat, S., Lochner, C., Vythilingum, B., & Stein, D. J. (2006). Disability and quality of life in post-traumatic stress disorder. *Pharmacoeconomics*, 24(10), 989–998.
- Shenton, M. E., Hamoda, H. M., Schneiderman, J. S., Bouix, S., Pasternak, O., Rath, Y., ... Zafonte, R. (2012). A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging and Behavior*, 6(2), 137–192.
- Sherin, J. E., & Nemeroff, C. B. (2011). Post-traumatic stress disorder: The neurobiological impact of psychological trauma. *Dialogues in Clinical Neuroscience*, 13(3), 263–278.
- Smith, M. E. (2005). Bilateral hippocampal volume reduction in adults with post-traumatic stress disorder: A meta-analysis of structural MRI studies. *Hippocampus*, 15(6), 798–807.
- Sørensen, H. J., Andersen, S. B., Karstoft, K.-I., & Madsen, T. (2016). The influence of pre-deployment cognitive ability on post-traumatic stress disorder symptoms and trajectories: The Danish USPER follow-up study of Afghanistan veterans. *Journal of Affective Disorders*, 196, 148–153.
- Spencer, R. J., Drag, L. L., Walker, S. J., & Bieliauskas, L. A. (2010). Self-reported cognitive symptoms following mild traumatic brain injury are poorly associated with neuropsychological performance in OIF/OEF veterans. *Journal of Rehabilitation Research and Development*, 47(6), 521–530.
- Spielberg, J. M., McGlinchey, R. E., Milberg, W. P., & Salat, D. H. (2015). Brain network disturbance related to posttraumatic stress and traumatic brain injury in veterans. *Biological Psychiatry*, 78(3), 210–216.
- Terrio, H., Brenner, L. A., Ivins, B. J., Cho, J. M., Helmick, K., Schwab, K., ... Warden, D. (2009). Traumatic brain injury screening: Preliminary findings in a US army brigade combat team. *The Journal of Head Trauma Rehabilitation*, 24(1), 14–23.
- Vasterling, J. J., Aslan, M., Lee, L. O., Proctor, S. P., Ko, J., Jacob, S., & Concato, J. (2018). Longitudinal associations among posttraumatic stress disorder symptoms, traumatic brain injury, and neurocognitive functioning in army soldiers deployed to the Iraq war. *Journal of the International Neuropsychological Society*, 24(4), 311–323.
- Vasterling, J. J., Brailey, K., Proctor, S. P., Kane, R., Heeren, T., & Franz, M. (2012). Neuropsychological outcomes of mild traumatic brain injury, post-traumatic stress disorder and depression in Iraq-deployed US Army soldiers. *The British Journal of Psychiatry*, 201(3), 186–192.
- Vasterling, J. J., Jacob, S. N., & Rasmussen, A. (2018). Traumatic brain injury and posttraumatic stress disorder: Conceptual, diagnostic, and therapeutic considerations in the context of co-occurrence. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 30(2), 91–100.
- Villareal, G., Hamilton, D. A., Petropoulos, H., Driscoll, I., Rowland, L. M., Griego, J. A., ... Brooks, W. M. (2002). Reduced hippocampal volume and total white matter volume in posttraumatic stress disorder. *Biological Psychiatry*, 52(2), 119–125.
- Vythilingam, M., Blair, K. S., McCaffrey, D., Scaramozza, M., Jones, M., Nakic, M., ... Blair, R. J. (2007). Biased emotional attention in post-traumatic stress disorder: A help as well as a hindrance? *Psychological Medicine*, 37(10), 1445–1455.

- Weathers, F. W., Blake, D. D., Schnurr, P. P., Kaloupek, D. G., Marx, B. P., Keane, T. M. (2013). The clinician-administered PTSD scale for DSM-5 (CAPS-5). Interview available from the National Center for PTSD at www.ptsd.va.gov
- Williams, J. M., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin*, 133(1), 122–148.
- Wisco, B. E., Marx, B. P., Holowka, D. W., Vasterling, J. J., Han, S. C., Chen, M. S., ... Keane, T. M. (2014). Traumatic brain injury, PTSD, and current suicidal ideation among Iraq and Afghanistan U.S. veterans. *Journal of Traumatic Stress*, 27(2), 244–248.
- Woodward, S. H., Kaloupek, D. G., Streeter, C. C., Martinez, C., Schaer, M., & Eliez, S. (2006). Decreased anterior cingulate volume in combat-related PTSD. *Biological Psychiatry*, 59(7), 582–587.
- Zatzick, D. F., Rivara, F. P., Jurkovich, G. J., Hoge, C. W., Wang, J., Fan, M.-Y., ... Mackenzie, E. J. (2010). Multisite investigation of traumatic brain injuries, posttraumatic stress disorder, and self-reported health and cognitive impairments. *Archives of General Psychiatry*, 67(12), 1291–1300.
- Zhou, Y., Kierans, A., Kenul, D., Ge, Y., Rath, J., Reaume, J., ... Lui, Y. W. (2013). Mild traumatic brain injury: Longitudinal regional brain volume changes. *Radiology*, 267(3), 880–890.

Caloric Restriction, Cognitive Function, and Brain Health

Patrick J. Smith

*Department of Psychiatry and Behavioral Sciences, Duke University Medical Center,
Durham, NC, USA*

Introduction

An increasingly large body of evidence suggests that lifestyle factors, including diet and exercise, play an important role in either mitigating or accelerating the neuropathological processes, leading to the development of Alzheimer's disease (AD) and other forms of late life dementia (Solfrizzi & Panza, 2014). Although previously conceived of as a disease developing in the later stages of life, AD in particular has been found to develop over the course of decades (Jack et al., 2010), emphasizing the importance of chronic lifestyle practices on the progression of subtle, insidious neuropathological changes.

Among the lifestyle characteristics examined, physical activity and dietary practices have been most extensively studied (Scarmeas et al., 2009). Although many studies have examined patterns of dietary behavior, such as the Mediterranean and Dietary Approaches to Stop Hypertension (DASH) diet, caloric restriction has consistently been an area of interest, owing to a large body of animal evidence demonstrating that it is the only known intervention to consistently increase lifespan across species. In addition, several lines of evidence among humans suggest that more limited intake of energy may confer reduced risk of dementia. For example, multiple studies have suggested that lower weight in midlife is associated with substantially lower risk of AD and other forms of dementia. Similarly, several randomized trials have provided evidence, albeit preliminary, that intentional caloric restriction may improve cognitive performance (Witte, Fobker, Gellner, Knecht, & Floel, 2009) and improve neuroimaging markers of brain health (Espeland et al., 2016). Nevertheless, multiple studies have suggested that lower weight, particularly underweight, is associated with worse outcomes among older adults, in contrast to the protective effects demonstrated in midlife.

Epidemiological Data

Accumulating evidence suggests that lower levels of caloric intake may be associated with improved cognitive performance and reduced rates of dementia. Although several recent examinations have been conducted in humans, this relationship has been studied extensively among animals. Despite relatively limited evidence in humans linking caloric restriction directly to brain outcomes, multiple, indirect lines of evidence support the notion that restricted energy intake may have salubrious effects. For example, multiple studies have demonstrated that being overweight or obese, particularly in midlife, is a risk factor for dementia (Kivipelto et al., 2006). Gustafson, Rothenberg, Blennow, Steen, and Skoog (2003) found that every 1.0 increase in BMI at age 70 was associated with a 36% increased risk of AD over an 18-year follow-up of nondemented, Swedish women. Similar results were found in a sample of Japanese men, with every 2.9 point increase in BMI conferring a 21% increased risk of vascular dementia (VaD) (Kalmijn et al., 2000). Although genetic factors no doubt play a prominent role in the development of AD, twin studies nevertheless demonstrate a similar relationship, with midlife obesity conferring more than a threefold increased risk of dementia (Xu et al., 2011). Although the mechanisms linking obesity to dementia are likely multifactorial (Craft, 2009), recent studies have suggested that the presence of obesity in midlife is associated with neuropathological correlates of dementia, including neurofibrillary tangles (Chuang et al., 2016) and total brain volume loss (Debetto et al., 2011). Despite these findings, it must also be noted that not all studies have shown an association between obesity and increased risk of dementia, a phenomenon that has come to be referred to as the “obesity paradox” (Driscoll et al., 2016).

Independent of studies examining obesity, a separate line of evidence suggests that more modest caloric intake is associated with improved longevity and that excess energy intake is associated with adverse cognitive outcomes. For example, among nearly 1,000 participants in the Washington Heights-Inwood Columbia Aging project, Luchsinger, Tang, Shea, and Mayeux (2002) found that greater caloric intake was associated with increased risk of AD, particularly among individuals with APOE-4 genotype. A well-known association has also been documented between chronic, markedly diminished caloric intake and improved longevity among Okinawans, who have a high prevalence of centenarians and relatively low rates of dementia (Willcox et al., 2007). In an archival analysis of centenarian Okinawans, participants even reported a 10.9% energy deficit during their 30s, a pattern that was maintained over subsequent decades of life.

Randomized Trials

Relatively few randomized trials have directly tested the impact of caloric restriction in human participants. In one of the most positive findings reported, Witte et al. (2009) examined the effects of caloric restriction on cognitive performance among 50 healthy, elderly subjects at either normal weight or mildly overweight. Participants were randomly assigned to either caloric restriction, increased intake of unsaturated fatty acids (UFAs), or a control condition. Following 3 months of treatment, individuals in the caloric restriction group exhibited increased verbal memory scores, and these improvements were correlated with decreased levels of fasting insulin and C-reactive protein. Notably, these results were strongest among subjects with the best adherence. Serum levels of brain-derived neurotrophic factor (BDNF), a growth factor thought to mediate the effects of aerobic fitness on brain function, were also assessed in this trial, but were not altered with treatment and were not associated with alterations in cognitive performance.

Martin et al. (2007) conducted a similar study among 48 overweight adults, aged 25–50 years. Participants were randomly assigned to one of four groups: caloric restriction, caloric restriction and exercise, a low-calorie diet condition, or to a control group (weight maintenance). At baseline, 3 months, and again at 6 months, cognitive tests were administered assessing verbal and visual memory, as well as concentration/attention. Overall, cognitive function changed very little across any group, and no consistent pattern of change emerged. In addition, changes in cognitive performance were not related to changes in caloric intake (indexed by daily energy deficit) or weight loss.

Several other randomized trials provide preliminary evidence that caloric restriction may confer cognitive and/or brain benefits in various patient populations. Horie et al. (2016) found that a 12-month nutritional counseling intervention among obese individuals with mild cognitive impairment was successful in improving cognitive performance across multiple subtests. Cognitive improvements were associated with changes in insulin resistance, CRP, leptin, and intake of energy, fat, and carbohydrates.

We previously examined this relationship in a sample of 120 middle-aged adults with hypertension and overweight or obesity (Smith et al., 2010). Participants were randomized for 4 months to a one of three groups: (a) a multicomponent weight loss intervention consisting of aerobic exercise and the DASH diet with caloric restriction, (b) the DASH diet without weight loss, and (c) a usual care control group. Results suggested that combined weight loss group showed improvements on several tests of executive function, learning, and memory, as well as psychomotor speed, with the DASH diet group alone showing more modest benefits on one measure of psychomotor speed alone compared with controls.

In one of the longest randomized trials to examined caloric restriction, Espeland et al. (2016) examined the potential persistent effects of the Action for Health in Diabetes clinical trial 10 years after randomization. The trial was designed to promote caloric restriction among adults with diabetes. In order to examine this relationship, the authors conducted a per protocol analysis, incorporating only individuals who exhibited at least modest weight loss. At the time of the 10-year follow-up, participants in the intervention group exhibited 28% lower white matter volume compared with control participants, who underwent a diabetes support and education program. In contrast, improvements in cognitive performance were relatively modest, only being observed on one subtest of processing speed.

In a relatively small trial, Prehn et al. (2017) examined changes in brain structure and function among 37 obese, postmenopausal women, randomized to an intensive 12-week low-calorie diet or a control group. In a *per protocol* analysis, the authors excluded individuals in the intervention group who did not lose at least 10% body weight, as well as participants in the control group who lost more than 5% of body weight. Participants in the intervention group exhibited improved memory performance paralleled by increased gray matter volume in the inferior frontal gyrus and hippocampus, as well as augmented hippocampal resting-state functional connectivity to parietal areas. The effects appeared to be specific to transient negative energy balance and were not detected after subsequent weight maintenance.

An overlapping but conceptually distinct question is whether weight loss is associated with improvements in neurocognitive performance. Although many studies achieve weight loss through a combination of exercise and dietary modification, more modest weight loss may be achieved through exercise alone. In addition, some studies have examined the impact of weight loss through bariatric surgery and its impact on cognitive change, although these studies are beyond the scope of the present review. In one of the few systematic reviews to examine intentional weight loss and cognitive function changes, Siervo et al. (2011) combined all randomized trials examining weight loss and cognitive function changes, including data from 12 studies, seven of which incorporated a control group in the study design. Of note, although the

majority of studies included were behavioral weight loss interventions, gastric bypass studies were also included. The authors found that intentional weight loss was associated with small improvements in memory and attention/executive functioning across a heterogeneous and group of trials, both in terms of intervention, sample, and cognitive assessment. Despite these positive findings, several quasi-randomized (Bryan & Tiggemann, 2001) and randomized (Martin et al., 2007) trials have failed to find cognitive benefits with caloric restriction.

Whole Diet Interventions

While only a small number of studies has examined caloric restriction and cognitive function specifically, it should be noted that a growing body of evidence suggests that overall healthier dietary patterns, which often include modest caloric reductions, have shown preliminary evidence of mitigating cognitive decline (Martinez-Lapiscina, Clavero, Toledo, Estruch, et al., 2013; Smith et al., 2010). As noted above, our study examining the DASH diet, with and without caloric restriction and weight loss, was associated with modest improvements in cognitive performance, which were associated with weight loss and improved fitness (Smith et al., 2010). In addition, results from a single site among the larger multi-site PREMEDI study demonstrated that participants randomized to receive the MeDI diet exhibited better cognitive performance than controls (Martinez-Lapiscina, Clavero, Toledo, Estruch, et al., 2013) and these benefits were maintained among some participants, who showed a reduced risk of mild cognitive impairment during subsequent follow-ups (Martinez-Lapiscina, Clavero, Toledo, San Julián, et al., 2013). These studies have provided preliminary evidence that intentional modification of dietary intake may confer cognitive improvements (Smith & Blumenthal, 2010), a question that is now being examined in several ongoing randomized trials.

Mechanisms

There are several mechanisms linking caloric restriction to cognitive and brain health. Although the majority of existing evidence has been derived from animal models of neurodegenerative illnesses, a growing body of evidence in humans has extended these findings, providing preliminary evidence linking underlying changes in neuroinflammation, cerebrovascular health, and neural signaling processes to cognitive performance (Murphy, Dias, & Thuret, 2014).

Neuroinflammation

A growing body of evidence suggests that neuroinflammation and microglial activation in response to injury may play an important role in the development of late life cognitive dysfunction and AD (Grammas, 2011). Caloric restriction and exercise-induced weight loss appear to improve peripheral inflammatory markers, and animal models of caloric restriction suggest that reduced caloric intake may mitigate amyloid burden in mouse models of AD. Moreover, increasing evidence suggests that neuroinflammatory cytokines may be an important regulator of neuronal plasticity, synaptogenesis, and neurogenesis, suggesting that basal elevations of neuroinflammation may counteract neuronal proliferation associated with behavior modification.

Cerebrovascular Health

As noted earlier, obesity and associated vascular comorbidities are associated with substantially increased risk of stroke, cerebrovascular burden, and AD in later life. Not surprisingly, preliminary evidence suggests that subtle MRI evidence of microvascular dysfunction is evident even among younger obese samples (Guo, Bakshi, & Lin, 2015) and that weight loss may improve microvascular function (Haltia et al., 2007) particularly among diabetic patients (Espeland et al., 2016). Moreover, microvascular function has been shown to mediate the association between dietary intake and cognitive performance in some samples of older adults (Gu et al., 2016), underscoring its potential importance among individuals engaging in chronic caloric restriction.

Neurohumoral/Growth Factor Modulation

Although few studies have examined it directly, preliminary evidence suggests that caloric restriction may increase expression of growth factors known to be important for neural plasticity and neurogenesis, most notably BDNF. Animal models of caloric restriction have suggested an upregulation of BDNF following reduced caloric intake, and, although few human studies have examined BDNF changes following caloric restriction (Witte et al., 2009), emerging evidence from other behavioral interventions suggests that BDNF expression may play an important role in neurogenesis, particularly in perihippocampal areas that are seminal in AD progression. Indeed, animal models have demonstrated that BDNF expression following caloric restriction is associated with enhanced hippocampal neurogenesis. In one of the few human studies to examine this, Araya, Orellana, and Espinoza (2008) found that only 3 months of caloric restriction enhanced BDNF expression among insulin-resistant overweight and obese subjects, suggesting that BDNF may be possible with dietary modification.

A closely related neural mechanism is enhanced neurohumoral responsivity, particularly enhanced insulin sensitivity and insulin-like growth factor (IGF-1) expression, which have been implicated as important components of the neuropathological processes underlying dementia, with particularly detrimental effects to the hippocampus. Insulin and IGF-1 appear to act as neurotrophins by inhibiting apoptosis, and insulin resistance has been increasingly recognized as a risk factor for AD, with novel treatments currently being designed to enhance cerebral glucose utilization. In one of the longest randomized trials ever to examine this question, Colman et al. (2014) examined the long-term effects of caloric restriction vs. ad libitum eating patterns in a sample of 30 adult, male, rhesus monkeys. The monkeys were followed for 20 years, and detailed accounting of chronic disease development and causes of death were examined. In their study, not only did monkeys randomized to caloric restriction live longer, but also they exhibited better glucose homeostasis, lower incidence of cancer and diabetes, and exhibited more preserved brain volumes compared with the control monkeys, particularly gray matter volumes in the frontal lobes.

Oxidative Stress

One of the most enduring theories linking caloric restriction to successful aging is its presumed amelioration of oxidative stress, which remains a central mechanism of age-related decline. Indeed, genes facilitating oxidative stress responses and DNA repair are preferentially

upregulated in the aging prefrontal cortex (Lu et al., 2004), implicating a central role in cognitive decline. Numerous animal studies have demonstrated that calorically limited mouse samples exhibit improved longevity and lower rates of oxidative damage, as evidenced by reduced accumulation of oxidatively damaged proteins, lipids, and DNA (Finkel & Holbrook, 2000). Although very few human studies have examined alterations in oxidative stress directly, Harvie et al. (2011) found that a 6-month randomized trial comparing caloric restriction with intermittent fasting yielded comparable improvements in markers of oxidative stress.

A related line of inquiry examines the impact of caloric restriction on sirtuins (Bishop, Lu, & Yankner, 2010), a family of proteins involved in cellular response to environmental and dietary stress. Specifically, these NAD-dependent deacetylases and monoadenosine diphosphate-ribosyl transferases influence the regulation of lipid and glucose metabolism, DNA repair, and insulin secretion and control cell survival. Although beyond the scope of the present review, sirtuins appear to be an important molecular mechanism linking caloric restriction to improved longevity (Mair & Dillin, 2008).

Future Directions

The existing body of evidence linking caloric restriction to cognitive function provides compelling preliminary data suggesting that limited energy intake may confer neuroprotective effects. Despite the available evidence, there are strikingly few large, well-controlled randomized controlled testing the benefits of caloric restriction among overweight or obese adults, and the vast majority of mechanistic data have been conducted in non-human samples, limiting the ability to draw conclusions regarding the putative neurobiological links between diet and brain function. Larger randomized trials examining caloric restriction among obese adults are needed to gain a more comprehensive understanding of the potential impact of dietary strategies to mitigate cognitive decline. Future studies could also benefit from delineating the unique effects of weight loss through caloric restriction with and without exercise, as many of the available studies utilized both treatment modalities to achieve larger treatment improvements in weight. A related, understudied aspect of caloric restriction is the potential benefit of intermittent fasting on cognitive and brain outcomes, which has been shown to produce comparable weight loss benefits when compared with traditional caloric restriction (Davis et al., 2016), reduces cardiovascular risk factors, is associated with reduced risk of age-related cognitive deficits, and may be easier for patients to adhere to. Finally, future randomized trials would benefit from careful assessment of mechanisms linking caloric restriction to cognitive function, including neuroimaging assessments of structural and functional outcomes, markers of neuroinflammation, neurotrophic expression, and oxidative stress.

Author Biography

Dr. Patrick J. Smith is an assistant professor in the Department of Psychiatry and Behavioral Sciences at Duke University Medical Center. Dr. Smith obtained his PhD in clinical psychology from Duke University with an emphasis in health and neuropsychology, as well as an MPH in biostatistics from the University of North Carolina at Chapel Hill. His research examines the impact of lifestyle modification, including aerobic exercise and dietary modification, on cardiovascular and cognitive outcomes.

References

- Araya, A. V., Orellana, X., & Espinoza, J. (2008). Evaluation of the effect of caloric restriction on serum BDNF in overweight and obese subjects: Preliminary evidences. *Endocrine*, 33(3), 300–304.
- Bishop, N. A., Lu, T., & Yankner, B. A. (2010). Neural mechanisms of ageing and cognitive decline. *Nature*, 464(7288), 529–535.
- Bryan, J., & Tiggemann, M. (2001). The effect of weight-loss dieting on cognitive performance and psychological well-being in overweight women. *Appetite*, 36(2), 147–156.
- Chuang, Y. F., An, Y., Bilgel, M., Wong, D. F., Troncoso, J. C., O'Brien, R. J., ... Thambisetty, M. (2016). Midlife adiposity predicts earlier onset of Alzheimer's dementia, neuropathology and presymptomatic cerebral amyloid accumulation. *Molecular Psychiatry*, 21(7), 910–915.
- Colman, R. J., Beasley, T. M., Kemnitz, J. W., Johnson, S. C., Weindruch, R., & Anderson, R. M. (2014). Caloric restriction reduces age-related and all-cause mortality in rhesus monkeys. *Nature Communications*, 5, 3557.
- Craft, S. (2009). The role of metabolic disorders in Alzheimer disease and vascular dementia: Two roads converged. *Archives of Neurology*, 66(3), 300–305.
- Davis, C. S., Clarke, R. E., Coulter, S. N., Rounsefell, K. N., Walker, R. E., Rauch, C. E., ... Ryan, L. (2016). Intermittent energy restriction and weight loss: A systematic review. *European Journal of Clinical Nutrition*, 70(3), 292–299.
- Debette, S., Seshadri, S., Beiser, A., Au, R., Himali, J. J., Palumbo, C., ... DeCarli, C. (2011). Midlife vascular risk factor exposure accelerates structural brain aging and cognitive decline. *Neurology*, 77(5), 461–468.
- Driscoll, I., Gaussoin, S. A., Wassertheil-Smoller, S., Limacher, M., Casanova, R., Yaffe, K., ... Espeland, M. A. (2016). Obesity and structural brain integrity in older women: The Women's Health Initiative Magnetic Resonance Imaging Study. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, 71(9), 1216–1222.
- Espeland, M. A., Erickson, K., Neiberg, R. H., Jakicic, J. M., Wadden, T. A., Wing, R. R., ... Bryan, R. N. (2016). Brain and white matter hyperintensity volumes after 10 years of random assignment to lifestyle intervention. *Diabetes Care*, 39(5), 764–771.
- Finkel, T., & Holbrook, N. J. (2000). Oxidants, oxidative stress and the biology of ageing. *Nature*, 408(6809), 239–247.
- Grammas, P. (2011). Neurovascular dysfunction, inflammation and endothelial activation: Implications for the pathogenesis of Alzheimer's disease. *Journal of Neuroinflammation*, 8, 26.
- Gu, Y., Vorburger, R. S., Gazes, Y., Habeck, C. G., Stern, Y., Luchsinger, J. A., ... Brickman, A. M. (2016). White matter integrity as a mediator in the relationship between dietary nutrients and cognition in the elderly. *Annals of Neurology*, 79(6), 1014–1025.
- Guo, J., Bakshi, V., & Lin, A. L. (2015). Early shifts of brain metabolism by caloric restriction preserve white matter integrity and long-term memory in aging mice. *Frontiers in Aging Neuroscience*, 7, 213.
- Gustafson, D., Rothenberg, E., Blennow, K., Steen, B., & Skoog, I. (2003). An 18-year follow-up of overweight and risk of Alzheimer disease. *Archives of Internal Medicine*, 163(13), 1524–1528.
- Haltia, L. T., Viljanen, A., Parkkola, R., Kempainen, N., Rinne, J. O., Nuutila, P., & Kaasinen, V. (2007). Brain white matter expansion in human obesity and the recovering effect of dieting. *The Journal of Clinical Endocrinology and Metabolism*, 92(8), 3278–3284.
- Harvie, M. N., Pegington, M., Mattson, M. P., Frystyk, J., Dillon, B., Evans, G., ... Cutler, R. G. (2011). The effects of intermittent or continuous energy restriction on weight loss and metabolic disease risk markers: A randomized trial in young overweight women. *International Journal of Obesity*, 35(5), 714–727.
- Horie, N. C., Serrao, V. T., Simon, S. S., Gascon, M. R. P., Santos, A. X. D., Zambone, M. A., ... Cercato, C. (2016). Cognitive effects of intentional weight loss in elderly obese individuals with mild cognitive impairment. *The Journal of Clinical Endocrinology and Metabolism*, 101(3), 1104–1112.
- Jack, C. R., Jr., Knopman, D. S., Jagust, W. J., Shaw, L. M., Aisen, P. S., Weiner, M. W., ... Trojanowski, J. Q. (2010). Hypothetical model of dynamic biomarkers of the Alzheimer's pathological cascade. *Lancet Neurology*, 9(1), 119–128.

- Kalmijn, S., Foley, D., White, L., Burchfiel, C. M., Curb, J. D., Petrovitch, H., ... Launer, L. J. (2000). Metabolic cardiovascular syndrome and risk of dementia in Japanese-American elderly men. The Honolulu-Asia aging study. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 20(10), 2255–2260.
- Kivipelto, M., Ngandu, T., Laatikainen, T., Winblad, B., Soininen, H., & Tuomilehto, J. (2006). Risk score for the prediction of dementia risk in 20 years among middle aged people: A longitudinal, population-based study. *Lancet Neurology*, 5(9), 735–741.
- Lu, T., Pan, Y., Kao, S. Y., Li, C., Kohane, I., Chan, J., & Yankner, B. A. (2004). Gene regulation and DNA damage in the ageing human brain. *Nature*, 429(6994), 883–891.
- Luchsinger, J. A., Tang, M. X., Shea, S., & Mayeux, R. (2002). Caloric intake and the risk of Alzheimer disease. *Archives of Neurology*, 59(8), 1258–1263.
- Mair, W., & Dillin, A. (2008). Aging and survival: The genetics of life span extension by dietary restriction. *Annual Review of Biochemistry*, 77, 727–754.
- Martin, C. K., Anton, S. D., Han, H., York-Crowe, E., Redman, L. M., Ravussin, E., & Williamson, D. A. (2007). Examination of cognitive function during six months of calorie restriction: Results of a randomized controlled trial. *Rejuvenation Research*, 10(2), 179–190.
- Martinez-Lapiscina, E. H., Clavero, P., Toledo, E., Estruch, R., Salas-Salvadó, J., & Julián, B. S. (2013). Mediterranean diet improves cognition: The PREDIMED-NAVARRA randomised trial. *Journal of Neurology, Neurosurgery, and Psychiatry*, 84(2), 1318–1325.
- Martinez-Lapiscina, E. H., Clavero, P., Toledo, E., Julián, B. S., Sanchez-Tainta, A., Corella, D., ... Martínez-Gonzalez, M. Á. (2013). Virgin olive oil supplementation and long-term cognition: The PREDIMED-NAVARRA randomized, trial. *The Journal of Nutrition, Health & Aging*, 17(6), 544–552.
- Murphy, T., Dias, G. P., & Thuret, S. (2014). Effects of diet on brain plasticity in animal and human studies: Mind the gap. *Neural Plasticity*, 2014, 563160.
- Prehn, K., Jumpertz von Schwartzberg, R., Mai, K., Zeitz, U., Witte, A. V., Hampel, D., ... Flöel, A. (2017). Caloric restriction in older adults-differential effects of weight loss and reduced weight on brain structure and function. *Cerebral Cortex*, 27(3), 1765–1778.
- Scarmeas, N., Luchsinger, J. A., Schupf, N., Brickman, A. M., Cosentino, S., Tang, M. X., & Stern, Y. (2009). Physical activity, diet, and risk of Alzheimer disease. *JAMA: The Journal of the American Medical Association*, 302(6), 627–637.
- Siervo, M., Arnold, R., Wells, J. C., Tagliabue, A., Colantuoni, A., Albanese, E., ... Stephan, B. C. M. (2011). Intentional weight loss in overweight and obese individuals and cognitive function: A systematic review and meta-analysis. *Obesity Reviews*, 12(11), 968–983.
- Smith, P. J., & Blumenthal, J. A. (2010). Diet and neurocognition: Review of evidence and methodological considerations. *Current Aging Science*, 3(1), 57–66.
- Smith, P. J., Blumenthal, J. A., Babyak, M. A., Craighead, L., Welsh-Bohmer, K. A., Browndyke, J. N., ... Sherwood, A. (2010). Effects of the dietary approaches to stop hypertension diet, exercise, and caloric restriction on neurocognition in overweight adults with high blood pressure. *Hypertension*, 55(6), 1331–1338.
- Solfrizzi, V., & Panza, F. (2014). Mediterranean diet and cognitive decline. A lesson from the whole-diet approach: What challenges lie ahead? *Journal of Alzheimer's Disease*, 39(2), 283–286.
- Willcox, B. J., Willcox, D. C., Todoriki, H., Fujiyoshi, A., Yano, K., He, Q., ... Suzuki, M. (2007). Caloric restriction, the traditional Okinawan diet, and healthy aging: The diet of the world's longest-lived people and its potential impact on morbidity and life span. *Annals of the New York Academy of Sciences*, 1114, 434–455.
- Witte, A. V., Fobker, M., Gellner, R., Knecht, S., & Floel, A. (2009). Caloric restriction improves memory in elderly humans. *Proceedings of the National Academy of Sciences of the United States of America*, 106(4), 1255–1260.
- Xu, W. L., Atti, A. R., Gatz, M., Pedersen, N. L., Johansson, B., & Fratiglioni, L. (2011). Midlife overweight and obesity increase late-life dementia risk: A population-based twin study. *Neurology*, 76(18), 1568–1574.

Psychosocial: Substance Abuse—Street Drugs

J. Megan Ross, Jacqueline C. Duperrouzel,
Ileana Pacheco-Colón, and Raul Gonzalez

Department of Psychology, Florida International University, Miami, FL, USA

Introduction

Drug use is a significant public health problem that impacts millions of individuals worldwide. In the United States, approximately 27 million individuals (12 years old and older) reported consumption of an illicit drug in the past 30 days—this translates to approximately 1 in 10 people in 2014. The percentage of individuals who have used an illicit drug in the past 30 days has increased each year since 2002 (Substance Abuse and Mental Health Services Administration [SAMHSA], 2015). Drugs of abuse impact neurotransmission, often influence neuropsychological performance, and confer risk of addiction. It has been well documented that substance use and abuse negatively impacts neuropsychological functioning. Since a review of every drug of abuse is beyond the scope of this chapter, we will focus on the most commonly used illegal psychoactive drugs. Whenever possible, this chapter will rely on findings from large-scale studies, review papers, and meta-analyses, as well as any pertinent novel, recent, and high impact research findings.

Before undertaking a review of the literature on neuropsychological consequences of substance use, there are several methodological challenges and limitations inherent in such work that are worth considering. One such pervasive challenge is heterogeneity of the participant samples. Participants within and across studies often have diverse patterns of substance use, vary on whether or not they meet criteria for a substance use disorder, often use multiple substances, and vary in prevalence of other comorbid conditions often common among substance users (e.g., psychiatric disorders, traumatic brain injury, HIV). Characteristics of substance users can vary on an array of domains that include length of abstinence, number and severity of polysubstance use, amount and frequency of use, and diagnosis and severity of

substance use disorders. For example, the neuropsychological effects of a particular drug may dissipate after a prolonged period of abstinence (e.g., cannabis; Schreiner & Dunn, 2012), or neuropsychological effects become more prominent after a prolonged period of abstinence (e.g., cocaine; Woicik et al., 2009). Perhaps one of the most intractable challenges to date is definitively determining whether neuropsychological impairments are a result of a substance use disorder or if these impairments precede a substance use disorder. Quasi-experimental designs are the only ethical research methods to use when studying the effects of chronic substance use on neuropsychological functioning in humans. As such, longitudinal designs and twin studies are some of the strongest research designs to assess whether neuropsychological deficits result from or predate substance use. With these issues in mind, we will focus on meta-analyses, longitudinal studies, twin studies, and recent novel studies whenever possible.

Benzodiazepines

Benzodiazepines (BZD) are one of the most widely used therapeutic drugs to treat anxiety and sleep disorders, with an estimated 3% of individuals endorsing prescribed lifetime use in 2014 (SAMHSA, 2015). Among high school seniors, use of Xanax (the most common nonmedically abused BZD) and perceived BZD availability have decreased over the past decade (SAMHSA, 2015).

Introduced during the 1960s, BZD became favored over prior sedatives and barbiturates because of their improved safety and low risk of lethality. BZD produces anxiolytic, anticonvulsant, sedative-hypnotic, and anesthetic effects, leading it to become synonymous with terms such as sedatives and tranquilizers. Created with the intention for only short-term use, BZD physiological and pharmacological dependence, withdrawal, and negative side effects are common even at low therapeutic doses. BZDs exert their effects on the central nervous system (CNS) by facilitating the rapid binding of GABA to its receptors with high concentrations in the amygdala, frontal cortex, insula, hippocampus, and cerebellum (Sieghart, 1994). Acute subjective effects of BZD administration include sedation, slowing, relaxation, and anterograde amnesia, which are directly associated with neuropsychological functioning (Buffett-Jerrott & Stewart, 2002).

Acutely, BZDs have been found to elicit deficits in many cognitive domains including processing speed, explicit and implicit memory, attention, and executive functions (Buffett-Jerrott & Stewart, 2002). However, differences in BZD chemical potency, bioavailability, and half-lives make it difficult to generalize results across individual studies.

To examine long-term BZD use effects, meta-analyses have provided greater insight on how cognitive functioning is influenced by prolonged use. Barker, Greenwood, Jackson, and Crowe (2004a, 2004b) evaluated cognitive performance in 12 neuropsychological domains and found moderate to large effects across all domains that included sensory processing, nonverbal memory, speed of processing, attention/concentration, general intelligence, working memory, psychomotor speed, visuospatial, problem solving, verbal memory, motor control/performance, and verbal reasoning in 13 studies.

Additionally, Barker and colleagues (2004a) conducted another meta-analysis to assess whether neuropsychological functioning in long-term users improves after BZD cessation. Results revealed small to moderate effect sizes in 5 of 11 domains, indicating an improvement in visuospatial, attention/concentration, general intelligence, psychomotor speed, and nonverbal memory performance. However, despite improvements, small to large negative effects were observed in verbal memory, psychomotor speed, speed of processing, motor

control, visuospatial, general intelligence, attention/concentration, and nonverbal memory compared with controls, despite discontinued BZD use for 6 months.

Similarly, BZD half-life metabolism has been found to negatively influence cognition. In a single study conducted by Helmes and Ostbye (2015), BZD half-life classification (i.e., short, intermediate, and long acting) was associated with differences in neuropsychological performance. Specifically, intermediate BZDs were associated with poorer performance in judgment and language comprehension and long-acting BZDs with poorer language comprehension and verbal short-term memory. These results suggest that larger cognitive deficits are observed in BZDs with longer half-lives, but no deficits are observed in shorter-acting BZDs. Despite specific cognitive impairments associated with BZD use, risk of global cognitive decline and incident dementia is only slightly higher compared with nonusers (Gray et al., 2016). When comparing BZD users, a slight increase in individual risk was only associated with low BZD exposure and not with higher levels of exposure. These results further suggest that the risk for developing more severe neurocognitive disorders such as Alzheimer's or dementia is only slightly elevated for those using low-dose BZDs (Gray et al., 2016).

Overall, evidence supports that acute and prolonged BZD use affects neurocognitive performance persistently. Despite evidence suggesting discontinuation of BZD use improves cognitive performance, residual effects have been observed and need to be further explored.

Cannabis

Cannabis is the most commonly used illicit drug (per federal regulations) in the United States with over 45% of Americans reporting lifetime use in 2014 (SAMHSA, 2015). With growing efforts over the last decade to legalize cannabis for recreational and medicinal use, risk perception has steadily declined among users and nonusers (Volkow, Baler, Compton, & Weiss, 2014). Due to its widespread use among various ages, cannabis and its neuropsychological effects have been avidly researched and debated.

The genus *Cannabis* contains three subspecies, *Cannabis sativa*, *Cannabis indica*, and *Cannabis ruderalis*, all which contain over 400 compounds influencing its analgesic, sedative, and stimulatory properties. In 1964, delta-9-tetrahydrocannabinol (Δ^9 -THC) was discovered to be the primary psychoactive constituent of cannabis, promoting interest in research and leading to the discovery of the endogenous cannabinoids. The discovery of the first cannabinoid receptor (CB1) in the mammalian brain in 1988 and the second (CB2) in the 1990s has led to a better understanding of how THC exerts its psychoactive effects by binding to CB1 receptors. CB1 receptors are located throughout the body with the densest concentrations located in the CNS. They are most abundant in the hippocampus, amygdala, basal ganglia, and cerebellum, structures known to influence learning, memory, reasoning, and emotional processing (Glass, Faull, & Dragunow, 1997), but can be found throughout cortex.

The psychoactive effects of cannabis on the CNS vary depending on route of administration and dose. The most common method of use, smoking, results in peak plasma concentrations within 3–10 min, and psychoactive effects lasting approximately 2–3 hr. Acute intoxication has been shown to affect neuropsychological functioning; however, results examining specific cognitive domains and age ranges provide varying results. In addition, many studies have provided evidence that regular and long-term cannabis use results in neurocognitive functioning deficits, even after the acute effects of the drug have subsided.

Due to heterogeneity in study design and limitations in methodology, single studies and research reviews provide a fragmented picture of cannabis' effects. To control for these

limitations, the utilization of meta-analytic techniques has allowed for consensus among findings. Grant, Gonzalez, Carey, Natarajan, and Wolfson (2003) examined the non-acute (i.e., residual, long term, permanent) effects of cannabis use in 11 studies and were the first to highlight small observable effect sizes providing evidence for subtle overall cognitive differences in heavy cannabis users compared with nonusers. Schreiner and Dunn (2012) replicated these results and extended them with numerous additional studies published since the original meta-analysis. They found positive effects globally and in six cognitive domains including abstraction/executive, attention, forgetting/retrieval, learning, motor, and verbal language, indicating deficits after acute cannabis intoxication (i.e., minimum 25-day abstinence). Further examining prolonged cannabis abstinence effects, Schreiner and Dunn (2012) found no significant effects of cognitive impairment after 6 months of discontinued use, demonstrating impairments may only be limited to recent cessation influenced by THC metabolite presence.

However, there is ongoing debate on the various factors that may influence neuropsychological performance in cannabis users. Two that have received significant attention are sex and age of initial use. Sex differences observed in episodic memory and age of cannabis use initiation suggest that females may be at higher risk for developing neurocognitive vulnerabilities and maintaining use compared with males (Crane, Schuster, Mermelstein, & Gonzalez, 2015). Similarly, an earlier age of cannabis use onset has been postulated by others to result in worse neurocognitive functioning. Strong support for this idea emerged recently from the largest longitudinal study to date assessing the relationship between cannabis use and neurocognitive functioning (Meier et al., 2012). Importantly, baseline assessments of neurocognitive functioning were available for all participants prior to cannabis use onset. The authors found that neuropsychological decline was associated with persistent cannabis use, only when persistent cannabis use began before age 18. However, studies of monozygotic and dizygotic twin pairs have reported no neurocognitive differences between twin pairs discordant for history of cannabis use (Jackson et al., 2016).

Recently neuroimaging techniques including functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) have been used to further delineate the neurobiological effects of cannabis use. In a systematic review conducted by Martin-Santos et al. (2010), acute and chronic cannabis exposure was associated with decreased blood flow during resting global acquisition in prefrontal cortex (PFC) and anterior cingulate cortex (ACC) compared with nonusers. Similarly, in a review of 43 structural and fMRI studies, the authors concluded that THC exposure was associated with reduced hippocampal volume and lower prefrontal cortical, cerebellar, and striatal blood oxygen level-dependent (BOLD) signal compared with controls during cognitive tasks (Batalla et al., 2013). These findings indicate a possible inefficient neural network influenced by cannabis use, despite comparable task outcomes with nonusers. Finally, evidence from recent neuroimaging studies is suggesting altered reward processing and motivation among regular cannabis users (e.g., Volkow et al., 2014).

The preponderance of evidence demonstrates that acute cannabis use on neuropsychological functioning influences cognitive domains including learning and memory. Deficits are also observed among current heavy cannabis users when not intoxicated with less frequent users displaying minimal or no deficits, although there is ongoing debate on the extent of these deficits in this population. While current neuropsychological results have suggested cannabis use impairments may dissipate over time, future neuroimaging studies and further examining prolonged neural changes after cessation need to be explored to support prior evidence.

Cocaine

Cocaine is a psychoactive alkaloid that is found in the leaves of the coca bush, native to Southwest America. In 2013, cultivation of the coca bush was at its lowest in the last three decades, creating a decrease in the availability of cocaine. In 2014, approximately 4.3 million American adults reported using cocaine in the past year, and 1.5 million reported using cocaine in the past month (SAMHSA, 2015). Typically, cocaine is consumed in one of two forms, which are cocaine hydrochloride, a powdered form of cocaine that is usually ingested intranasally, or crack cocaine, a free-base form of cocaine that is typically smoked. It is important to note that cocaine can cause ischemic and hemorrhagic cerebrovascular damage that can lead to neuropsychological deficits (Levine et al., 1990).

Similarly to other psychostimulants, acute effects of cocaine on neuropsychological functioning results in better attention (Johnson et al., 1998), speed of information processing (Higgins et al., 1990), and inhibitory control (Fillmore, Rush, & Hays, 2006). However, cocaine use can cause long-term neuronal and neuropsychological damage. Brain imaging studies suggest cocaine-dependent individuals have lower gray matter volumes in the premotor cortex, orbitofrontal cortex, temporal cortex, thalamus, and cerebellum compared with healthy controls. These deficits were associated with poorer performance on tasks of executive function and fine motor performance (Sim et al., 2007). Ersche, Williams, Robins, and Bullmore (2013) conducted a voxel-wise meta-analysis of MRI studies to determine structural brain abnormalities among individuals addicted to stimulants (i.e., cocaine, amphetamines, or methamphetamines). Although the meta-analysis included individuals addicted to cocaine, amphetamines, or methamphetamines, a majority of the studies ($n = 10$) included only participants who were addicted to cocaine. The meta-analysis contained 16 studies composed of 494 stimulant-dependent individuals compared with 428 healthy controls. Stimulant-addicted individuals had significant gray matter volume decreases in five specific regions: the insula, ventromedial PFC, inferior frontal gyrus, pregenual anterior cingulate gyrus, and anterior thalamus. The regions with gray matter volume decreases are primarily involved with executive function. These findings generally support research that suggests executive function deficits among cocaine-dependent individuals.

A qualitative review conducted by Jovanovski, Erb, and Zakzanis (2005) of the neuropsychological effects of cocaine use compared the effect sizes across studies to determine the most consistent neuropsychological deficits. Across 15 studies that included 481 cocaine users and 586 controls, the most consistent deficit observed among cocaine users was in attention and executive functioning. Attention deficits had the largest effect sizes followed by working memory, visual memory, and executive functioning. Small effect sizes were observed for deficits in language function (including verbal fluency and sensory-perceptual function). It must be noted that the cocaine-dependent individuals included in this meta-analysis have a wide range of number of days of abstinence (0–1,075).

One notable study conducted by Woicik and colleagues (2009) documented differences in neuropsychological performance among individuals with current cocaine use disorders and matched healthy controls in measures of attention, executive function, and verbal memory. However, these deficits were more pronounced in the individuals with cocaine use disorders who have a negative urine status for cocaine. These findings suggest that neuropsychological deficits for cocaine-dependent individuals may manifest after a longer period of abstinence, but more work is needed to understand the underlying cause for this observed effect.

Currently, the evidence generally suggests that the largest deficits associated with cocaine use are in the areas of attention and executive functioning, while small effects have been

observed in memory and language functioning. These findings are supported by neuroimaging studies that have observed decreased gray matter volume primarily in the PFC. However, as documented by Woicik and colleagues (2009), these findings may be more pronounced after a longer period of abstinence from cocaine use. Future studies that evaluate the association of cocaine use and neuropsychological functioning may benefit from also examining cocaine-dependent individuals who have been abstinent from cocaine use for an extended period of time.

MDMA (Ecstasy)

3,4-Methylenedioxymethamphetamine (MDMA), or “ecstasy,” has a similar chemical structure to both stimulants and hallucinogens and has been classified as an “entactogen.” In the United States, 6.6% of Americans ages 12 and older have used ecstasy at least once in their lifetime (SAMHSA, 2015). A significant challenge to the study of the neuropsychological effects of MDMA is that ecstasy pills are expected to contain MDMA; however this is not always the case. Many ecstasy pills (also known as molly, candy, and rolling) contain other psychoactive substances or may not contain any MDMA at all. Specifically, out of 1,214 tablets sold as ecstasy, only 39% of the tablets contained pure MDMA, 46% did not contain any MDMA at all, and the remaining 15% contained both MDMA and other substances (e.g., caffeine, dextromethorphan, methylenedioxyamphetamine, methylenedioxyethylamphetamine, and methamphetamine; Tanner-Smith, 2006).

Attention performance has been measured during acute intoxication of MDMA and has yielded mixed results. Studies suggest no impairment in attention (Vollenweider, Gamma, Liechti, & Huber, 1998), though one study found that acute intoxication of MDMA can result in deficits in divided attention and tracking performance (Kuypers, Wingen, Samyn, Limbert, & Ramaekers, 2007). Impairments in memory performance have also been reported; however, these impairments appear to return to baseline levels after intoxication (Kuypers & Ramaekers, 2005). Kuypers, Wingen, Heinecke, Formisano, and Ramaekers (2011) designed a study to determine the neural substrates associated with memory impairment during acute intoxication of MDMA via MRI. Results suggest that impairments with memory encoding may be due to the influence of MDMA on middle frontal gyrus functioning.

Several meta-analyses have been conducted that address the neuropsychological sequelae of MDMA. Verbaten (2003) examined only studies with between-group designs between 1975 and 2002. This meta-analysis contained 14 studies, and the only inclusion criteria for the meta-analysis were that participants had to be abstinent from MDMA and other drugs for at least one week prior to the assessment. Results suggest that MDMA users performed significantly worse on measures of short-term memory, long-term memory, and attention. Kalechstein, De La Garza, Mahoney, Fantegrossi, and Newton (2007) conducted a similar meta-analysis of studies conducted until 2004, with lenient inclusion criteria (i.e., any studies that evaluated neuropsychological effects of MDMA with matched controls) resulting in 23 studies and strict inclusion criteria (i.e., matched controls on demographic and premorbid factors and MDMA users were abstinent at evaluation and not treatment seeking) resulting in 11 studies. MDMA users performed significantly worse in all domains in both conditions. Other meta-analyses have reached similar conclusions, with small to moderate effect sizes across all domains and a negative association between performance and length of MDMA use (Zakzanis, Campbell, & Jovanovski, 2007). Recently, a meta-analysis by Murphy et al. (2012) was conducted to determine the effects of MDMA on visuospatial memory. Liberal criteria for

inclusion in the meta-analysis were applied (i.e., any study that used visuospatial memory tasks and participants who were abstinent during the evaluation), yielding 52 studies. Moderate effect sizes were observed for MDMA users performing significantly worse on recall or recognition of spatial distribution of stimulus elements, recognition of figures, and reproduction or production of figures.

Notable prospective studies have been recently conducted that included only MDMA-naïve participants at baseline and followed the participants to determine the effects of MDMA after initiation of use. The study included 149 participants who had never used MDMA at baseline—109 participants were administered the 1-year follow-up. During the 1-year follow-up period, 43 subjects did not use any illicit substances, while 23 participants used MDMA more than 10 times. The remaining subjects, who used MDMA more than once but less than 10 times, were not included in the analysis. Change scores were compared between MDMA users and nonusers. MDMA users performed significantly worse on immediate and delayed recall of visual paired associations; however, there was no other area of cognitive decline (Wagner, Becker, Koester, Gouzoulis-Mayfrank, & Daumann, 2013). Using the same sample at a 2-year follow-up, immediate and delayed recall of visual paired associates declined significantly among MDMA users, and no significant decline was observed in other areas of neuropsychological functioning (Wagner et al., 2015).

Generally, the evidence supports that MDMA may cause decline in visual memory and learning impairment among users. Although notable, the small sample sizes among some studies limit the generalizability of these findings. A larger sample size may reveal small or moderate changes in cognitive domains other than visual memory and learning. Future studies in this area would therefore benefit from larger sample sizes. Additionally, meta-analyses suggest cognitive decline in several domains, though these findings are divergent among prospective studies.

Methamphetamine

Methamphetamine (e.g., ice, crank, crystal meth) is a potent and highly addictive stimulant of the CNS, the effects of which can last up to 12 hr. Methamphetamine can be consumed in a variety of ways, including snorting, smoking, and injection. Recent national estimates indicate that 1.3 million people (0.5% of the population) reported using methamphetamine in the past year and 569,000 (0.2%) reported using it in the past month, figures similar to those from previous years (SAMHSA, 2015).

Methamphetamine's lipophilicity allows it to cross the blood–brain barrier faster and thus penetrate the CNS to a greater extent than its parent compound amphetamine. Methamphetamine stimulates the release of dopamine while simultaneously blocking, to some extent, synaptic reuptake of dopamine, both of which result in increased levels of dopamine in reward regions of the brain.

The acute effects of methamphetamine may include autonomic arousal, tachycardia, suppression of appetite, euphoria, feelings of increased physical and mental capacity, elevated self-esteem, increased libido, insomnia, and irritability. Administration of moderate doses of methamphetamine can lead to enhanced attention and concentration (Johnson et al., 2005).

Long-term use induces neurotoxicity in several neurotransmitter systems, particularly the nigrostriatal dopaminergic pathways, thus altering the function of prefrontal–striatal circuits. A meta-analysis, which included 18 studies and a total of 487 participants with lifetime methamphetamine abuse/dependence and 464 healthy controls, revealed that chronic

methamphetamine users showed impaired neuropsychological performance in the domains of learning, executive functions, memory, speed of information processing, and motor skills relative to healthy controls, which is likely related to the dysfunction in the neural circuits underlying these processes (Scott et al., 2007). These effect sizes were broadly in the medium range, such that methamphetamine users performed worse than controls in measures of attention/working memory, visuoconstruction, and language (Scott et al., 2007).

Neuroimaging studies have found a range of structural and metabolic abnormalities in the brains of chronic methamphetamine abusers. Structurally, methamphetamine abusers showed enlarged striatal volumes, suggesting an initial compensatory (inflammatory) response (Chang, Alicata, Ernst, & Volkow, 2007). However, a study found that greater usage ultimately led to a decrease in striatal volume, associated with greater cognitive impairment (Chang et al., 2005). Smaller hippocampal volumes have also been linked to poorer episodic memory performance among chronic users (Thompson et al., 2004). PET studies have found reduced dopamine transporter (DAT) density as well as reduced D₂ dopamine receptors in the striatum of methamphetamine users (Chang et al., 2007). Though changes in DAT density subside with prolonged abstinence, the psychomotor and learning impairments associated with them do not, which suggests either insufficient recovery or the involvement of other unidentified neural networks (Chang et al., 2007). Furthermore, H¹ magnetic resonance spectroscopy studies have revealed lower concentrations of the metabolite *N*-acetylaspartate (NAA), a marker for neuronal integrity and density, and creatine, which reflects cellular energy metabolism, in the basal ganglia of individuals who recently abstained from chronic methamphetamine use (Chang et al., 2007). The etiology and consequences of these abnormalities are not yet clear.

Studies have employed fMRI techniques to detect differences in the brain activity of methamphetamine abusers, which mostly involve hypoactivity of frontal areas. Notably, Salo, Fassbender, Buonocore, and Ursu (2013) found that methamphetamine abusers showed little to no activation in the dorsolateral PFC relative to controls during a variant of the Stroop task that measured trial-to-trial reaction time adjustments. PFC activity was negatively correlated with reaction time adjustments, such that less activity was related to longer reaction times and thus worse performance (Salo et al., 2013). These findings suggest a failure to adapt a behavioral response based on prior experience, which is a hallmark behavior of addiction (Salo et al., 2013). Hypoactivity of frontal and other areas in methamphetamine users has also been linked to deficits in socioemotional processing (Payer et al., 2008) and decision making (Paulus, Hozack, Frank, Brown, & Schuckit, 2003).

Together, these findings suggest that long-term methamphetamine abuse leads to learning and memory impairments, as well as executive dysfunction. Neuroimaging findings suggest that these deficits are linked to abnormalities in prefrontal–striatal circuits, most notably abnormalities in gray matter volumes of subcortical structures and hypoactivity of frontal areas. Further research is needed to determine whether these impairments will improve with prolonged abstinence.

Opiates

Opiate is extracted from the seeds of the opium poppy plant, with majority of the illegal opiates being grown in Afghanistan. Opioids are the synthetic form of opiates. Approximately 4.8 million Americans reported using heroin in their lifetime during 2014, a figure that has remained steady since 2013 (SAMHSA, 2015). Prescription opioid addiction, such as Vicodin

and Oxycontin, has become a national crisis in the United States due to the increases in opioid abuse and overdose deaths. The number of deaths related to opioid abuse has almost tripled since 2010 (Hedegaard, Chen, & Warner, 2015).

Neuroimaging studies have documented detrimental effects of opiate use on brain structure and function. General cortical atrophy and enlargement of external and internal CSF spaces (Pezawas et al., 1998) have been shown among individuals addicted to opiates. Specifically, decreases in gray matter density have been observed in the prefrontal, anterior cingulate, insular, and temporal cortices among opiate abusers compared with healthy controls (Lyoo et al., 2006).

Gruber, Silveri, and Yurgelun-Todd (2007) reviewed the neuropsychological consequences of opiate use. Results suggest that neuropsychological deficits exist both acutely and residually in measures of attention, concentration, recall, visuospatial, and psychomotor speed. Executive function appears to suffer the greatest impact from opiate use. A recent meta-analysis on the neuropsychological effects of opiate use revealed that the most robust impairments were in measures of verbal working memory, risk-taking (i.e., delay discounting and decision making), and verbal fluency. This review analyzed 20 studies with 767 chronic opioid users and 15,196 healthy controls compared on 7 neuropsychological domains: short-term verbal working memory, short-term visuospatial working memory, long-term memory, attention, cognitive impulsivity, non-planning impulsivity, and cognitive flexibility (Baldacchino, Balfour, Passetti, Humphris, & Matthews, 2012).

Research on opioid addiction has focused on the neuropsychological impairments associated with methadone maintenance therapy (MMT), the most commonly used form of replacement therapy for opioid dependence. Neuropsychological deficits have been observed among patients on MMT including attention, working memory, memory, psychomotor speed, problem solving, and decision making (Darke, Sims, McDonald, & Wickes, 2000; Mintzer & Stitzer, 2002). When comparing patients on MMT to abstinent heroin abusers, patients on MMT had significantly poorer performance on measures of processing speed, visuospatial perception, cognitive flexibility, working memory, and logical reasoning (Verdejo, Toribio, Orozco, Puente, & Pérez-García, 2005).

These findings overwhelmingly suggest that there are neuropsychological impairments associated with opioid dependence. These cognitive impairments include verbal working memory, risk-taking, and verbal fluency. In addition, patients on MMT may experience cognitive impairments in processing speed, visuospatial perception, cognitive flexibility, working memory, and logical reasoning.

Discussion

Substance use is associated with numerous negative outcomes, including social, emotional, and economic consequences that make it a significant public health problem. The use of neuropsychological tools in the context of substance use disorders has helped us to gain a better understanding of how critical cognitive processes are affected by substances of abuse, acutely and in the longer term. This chapter presents a complex picture of consequences from use of various substances. The heterogeneity in findings is likely due to a variety of issues, including each drug's own unique pharmacological properties as well as the characteristics of participant samples. For example, did the study include individuals recently out of recovery, a community sample of users, individuals that used a certain amount of a drug, or individuals that met criteria for a substance use disorder? Regardless, as has been described in this chapter, adverse effects

on neurocognitive functioning are common consequence of substance use. Indeed, our review suggests that substance abuse is generally associated with neuropsychological deficits. However, as noted, these deficits vary across substances, neurocognitive domains, pattern, severity, and duration. Collectively, in most cases, more severe, frequent, and symptomatic substance use (e.g., a diagnosable substance use disorder) is associated with poorer neurocognitive outcomes. Recovery of neurocognitive functioning with abstinence is a more complex issue. Whereas abstinence from some substances may result in normalization of neurocognitive functioning over time (e.g., memory functioning among cannabis users), other deficits seem to persist or progressively worsen despite abstinence with other substances (e.g., cocaine and BZD).

Another important issue to consider, which often looms in the background and has received progressively more attention in recent years, is whether any of the neurocognitive deficits observed among substance users may have predated use; rather than a consequence of use, they may serve as a risk factor for addiction. From a theoretical standpoint, measures of inhibitory control, decision making, and other aspects of executive functioning are plausible candidates for neurocognitive deficits that may predate heavy use. However, without prospective longitudinal studies that adequately assess neurocognitive functioning before and after initiation of substance use, this issue cannot be adequately resolved. Although such studies have been conducted by several research groups or among specific cohorts (e.g., the Dunedin cohort; CEDAR; Pittsburgh Youth Study), many of these were not specifically designed to examine changes in neurocognitive functioning over time. The newly launched Adolescent Brain Cognitive Development (ABCD) project aims to examine neurocognitive abilities and brain structure and function, alongside many other relevant factors, among a representative sample of 10,000 children starting at ages 9–10 and throughout adolescence and early adulthood. Substance use characteristics will also be carefully assessed in this project to provide a clearer picture of how the brain looks and functions prior to and after initiation of substance use and how it continues to change among those that use relative to those that do not. In the process, the results of this project will address many of these lingering issues.

Despite dramatic advances in the scientific study of the neuropsychology of substance use, there are plenty of areas in which future studies could help elucidate discrepant findings and further our understanding of substance use disorders. As noted, prospective longitudinal studies with large sample sizes will continue to move the field forward. Similarly, more sophisticated meta-analyses that carefully consider how heterogeneity of samples and specific substance use characteristics influence findings will be valuable to the scientific literature.

Author Biographies

J. Megan Ross graduated from Florida International University with a doctorate in clinical psychology. She is currently a postdoctoral fellow at the Institute for Behavioral Genetics at the University of Colorado, Boulder. Her research focuses on predictors and moderators of risk-taking behavior among adolescents.

Jacqueline C. Duperrouzel graduated with a BS in Psychology from the Florida State University and received her MS in Clinical Psychology from Florida International University (FIU). She is currently a graduate student at FIU pursuing a PhD in Clinical Science, where her current research interests focus on examining the neuropsychological effects of early-onset cannabis use and factors that contribute to substance use disorder development.

Ileana Pacheco-Colón graduated with a BS in Psychology from Brown University. She is currently a graduate student pursuing a PhD in Clinical Psychology at Florida International

University, where her research focuses on the effects of adolescent cannabis use on neuropsychological functioning and motivation.

Raul Gonzalez is professor of psychology, psychiatry, and immunology at Florida International University (FIU). He graduated with his PhD in Clinical Psychology from the San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology, with a specialization in clinical neuropsychology. He is the director of the Substance Use and HIV Neuropsychology and affiliated with the Center for Children and Families at FIU. His research broadly examines risks for and consequences of substance use, with a focus on cannabis.

References

- Baldacchino, A., Balfour, D., Passetti, F., Humphris, G., & Matthews, K. (2012). Neuropsychological consequences of chronic opioid use: A quantitative review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 36(9), 2056–2068.
- Barker, M. J., Greenwood, K. M., Jackson, M., & Crowe, S. F. (2004a). Cognitive effects of long-term benzodiazepine use. *CNS Drugs*, 18(1), 37–48.
- Barker, M. J., Greenwood, K. M., Jackson, M., & Crowe, S. F. (2004b). Persistence of cognitive effects after withdrawal from long-term benzodiazepine use: A meta analysis. *Archives of Clinical Neuropsychology*, 19, 437–454.
- Batalla, A., Bhattacharyya, S., Yücel, M., Fusar-Poli, P., Crippa, J. A., Nogué, S., ... Martin-Santos, R. (2013). Structural and functional imaging studies in chronic cannabis users: A systematic review of adolescent and adult findings. *PLoS One*, 8(2), e55821.
- Buffett-Jerrott, S. E., & Stewart, S. H. (2002). Cognitive and sedative effects of benzodiazepine use. *Current Pharmaceutical Design*, 8(1), 45–58.
- Chang, L., Alicata, D., Ernst, T., & Volkow, N. (2007). Structural and metabolic brain changes in the striatum associated with methamphetamine abuse. *Addiction*, 102(1), 16–32.
- Chang, L., Cloak, C., Patterson, K., Grob, C., Miller, E. N., & Ernst, T. (2005). Enlarged striatum in abstinent methamphetamine abusers: A possible compensatory response. *Biological Psychiatry*, 57(9), 967–974.
- Crane, N. A., Schuster, R. M., Mermelstein, R. J., & Gonzalez, R. (2015). Neuropsychological sex differences associated with age of initiated use among young adult cannabis users. *Journal of Clinical and Experimental Neuropsychology*, 23(2), 117–137.
- Darke, S., Sims, J., McDonald, S., & Wickes, W. (2000). Cognitive impairment among methadone maintenance patients. *Addiction*, 95(5), 687–695.
- Ersche, K. D., Williams, G. B., Robbins, T. W., & Bullmore, E. T. (2013). Meta-analysis of structural brain abnormalities associated with stimulant drug dependence and neuroimaging of addiction vulnerability and resilience. *Current Opinion in Neurobiology*, 23(4), 615–624.
- Fillmore, M. T., Rush, C. R., & Hays, L. (2006). Acute effects of cocaine in two models of inhibitory control: Implications of non-linear dose effects. *Addiction*, 101(9), 1323–1332.
- Glass, M., Faull, R. L. M., & Dragunow, M. (1997). Cannabinoid receptors in the human brain: A detailed anatomical and quantitative autoradiographic study in the fetal, neonatal and adult human brain. *Neuroscience*, 77(2), 299–318.
- Grant, I., Gonzalez, R., Carey, C. L., Natarajan, L., & Wolfson, T. (2003). Non-acute (residual) neurocognitive effects of cannabis use: A meta-analytic study. *Journal of the International Neuropsychological Society*, 9(5), 679–689.
- Gray, S. L., Dublin, S., Yu, O., Walker, R., Anderson, M., Hubbard, R. A., ... Larson, E. B. (2016). Benzodiazepine use and risk of incident dementia or cognitive decline: Prospective population based study. *British Medical Journal*, 352.
- Gruber, S. A., Silveri, M. M., & Yurgelun-Todd, D. A. (2007). Neuropsychological consequences of opiate use. *Neuropsychology Review*, 17(3), 299–315.

- Hedegaard, H., Chen, L. H., & Warner, M. (2015). *Drug-poisoning deaths involving heroin: United States, 2000–2013*. US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.
- Helmes, E., & Ostbye, T. (2015). Associations between benzodiazepine use and neuropsychological test scores in older adults. *Canadian Journal on Aging, 34*(2), 207–214.
- Higgins, S. T., Bickel, W. K., Hughes, J. R., Lynn, M., Capeless, M. A., & Fenwick, J. W. (1990). Effects of intranasal cocaine on human learning, performance and physiology. *Psychopharmacology, 102*(4), 451–458.
- Jackson, N. J., Isen, J. D., Khoddam, R., Irons, D., Tuvblad, C., Iacono, W. G., & Baker, L. A. (2016). Impact of adolescent marijuana use on intelligence: Results from two longitudinal twin studies. *Proceedings of the National Academy of Sciences of the United States of America, 113*(5), E500–E508.
- Johnson, B., Overton, D., Wells, L., Kenny, P., Abramson, D., Dhoother, S., ... Bordnick, P. (1998). Effects of acute intravenous cocaine on cardiovascular function, human learning, and performance in cocaine addicts. *Psychiatry Research, 77*(1), 35–42.
- Johnson, B. A., Roache, J. D., Ait-Daoud, N., Wallace, C., Wells, L. T., & Wang, Y. (2005). Effects of isradipine on methamphetamine-induced changes in attentional and perceptual-motor skills of cognition. *Psychopharmacology, 178*(2–3), 296–302.
- Jovanovski, D., Erb, S., & Zakzanis, K. K. (2005). Neurocognitive deficits in cocaine users: A quantitative review of the evidence. *Journal of Clinical and Experimental Neuropsychology, 27*(2), 189–204.
- Kalechstein, A. D., De La Garza, R., II, Mahoney, J. J., III, Fantegrossi, W. E., & Newton, T. F. (2007). MDMA use and neurocognition: A meta-analytic review. *Psychopharmacology, 189*(4), 531–537.
- Kuypers, K. P., & Ramaekers, J. G. (2005). Transient memory impairment after acute dose of 75mg 3,4-methylene-dioxymethamphetamine. *Journal of Psychopharmacology, 19*(6), 633–639.
- Kuypers, K. P., Wingen, M., Heinecke, A., Formisano, E., & Ramaekers, J. G. (2011). MDMA intoxication and verbal memory performance: a placebo-controlled pharmaco-MRI study. *Journal of Psychopharmacology, 25*(8), 1053–1061.
- Kuypers, K. P., Wingen, M., Samyn, N., Limbert, N., & Ramaekers, J. G. (2007). Acute effects of nocturnal doses of MDMA on measures of impulsivity and psychomotor performance throughout the night. *Psychopharmacology, 192*(1), 111–119.
- Levine, S. R., Brust, J. C., Futrell, N., Ho, K.-L., Blake, D., Millikan, C. H., ... Selwa, J. F. (1990). Cerebrovascular complications of the use of the crack form of alkaloidal cocaine. *New England Journal of Medicine, 323*(11), 699–704.
- Lyoo, I. K., Pollack, M. H., Silveri, M. M., Ahn, K. H., Diaz, C. I., Hwang, J., ... Renshaw, P. F. (2006). Prefrontal and temporal gray matter density decreases in opiate dependence. *Psychopharmacology, 184*(2), 139–144.
- Martin-Santos, R., Fagundo, A. B., Crippa, J. A., Atakan, Z., Bhattacharyya, S., Allen, P., ... McGuire, P. (2010). Neuroimaging in cannabis use: A systematic review of the literature. *Psychological Medicine, 40*(03), 383–398.
- Meier, M. H., Caspi, A., Ambler, A., Harrington, H., Houts, R., Keefe, R. S., & Moffitt, T. E. (2012). Persistent cannabis users show neuropsychological decline from childhood to midlife. *Proceedings of the National Academy of Sciences of the United States of America, 109*(40), E2657–E2664.
- Mintzer, M. Z., & Stitzer, M. L. (2002). Cognitive impairment in methadone maintenance patients. *Drug and Alcohol Dependence, 67*(1), 41–51.
- Murphy, P. N., Bruno, R., Ryland, I., Wareing, M., Fisk, J. E., Montgomery, C., & Hilton, J. (2012). The effects of ‘ecstasy’ (MDMA) on visuospatial memory performance: Findings from a systematic review with meta-analyses. *Human Psychopharmacology: Clinical and Experimental, 27*(2), 113–138.
- Paulus, M. P., Hozack, N., Frank, L., Brown, G. G., & Schuckit, M. A. (2003). Decision making by methamphetamine-dependent subjects is associated with error-rate-independent decrease in prefrontal and parietal activation. *Biological Psychiatry, 53*(1), 65–74.
- Payer, D. E., Lieberman, M. D., Monterosso, J. R., Xu, J., Fong, T. W., & London, E. D. (2008). Differences in cortical activity between methamphetamine-dependent and healthy individuals performing a facial affect matching task. *Drug and Alcohol Dependence, 93*(1), 93–102.

- Pezawas, L. M., Fischer, G., Diamant, K., Schneider, C., Schindler, S. D., Thurnher, M., ... Kasper, S. (1998). Cerebral CT findings in male opioid-dependent patients: Stereological, planimetric and linear measurements. *Psychiatry Research: Neuroimaging*, 83(3), 139–147.
- Salo, R., Fassbender, C., Buonocore, M. H., & Ursu, S. (2013). Behavioral regulation in methamphetamine abusers: An fMRI study. *Psychiatry Research: Neuroimaging*, 211(3), 234–238.
- Schreiner, A. M., & Dunn, M. E. (2012). Residual effects of cannabis use on neurocognitive performance after prolonged abstinence: A meta-analysis. *Experimental and Clinical Psychopharmacology*, 20(5), 420.
- Scott, J. C., Woods, S. P., Matt, G. E., Meyer, R. A., Heaton, R. K., Atkinson, J. H., & Grant, I. (2007). Neurocognitive effects of methamphetamine: A critical review and meta-analysis. *Neuropsychology Review*, 17(3), 275–297.
- Sieghart, W. (1994). Pharmacology of benzodiazepine receptors: An update. *Journal of Psychiatry and Neuroscience*, 19(1), 24–29.
- Sim, M. E., Lyoo, I. K., Streeter, C. C., Covell, J., Sarid-Segal, O., Ciraulo, D. A., ... Renshaw, P. F. (2007). Cerebellar gray matter volume correlates with duration of cocaine use in cocaine-dependent subjects. *Neuropsychopharmacology*, 32(10), 2229–2237.
- Substance Abuse and Mental Health Services Administration. (2015). *2014 National survey on drug use and health: Detailed tables*. Rockville, MD: Center for Behavioral Health Statistics and Quality.
- Tanner-Smith, E. E. (2006). Pharmacological content of tablets sold as “ecstasy”: Results from an online testing service. *Drug and Alcohol Dependence*, 83(3), 247–254.
- Thompson, P. M., Hayashi, K. M., Simon, S. L., Geaga, J. A., Hong, M. S., Sui, Y., ... London, E. D. (2004). Structural abnormalities in the brains of human subjects who use methamphetamine. *The Journal of Neuroscience*, 24(26), 6028–6036.
- Verbaten, M. N. (2003). Specific memory deficits in ecstasy users? The results of a meta-analysis. *Human Psychopharmacology: Clinical and Experimental*, 18(4), 281–290.
- Verdejo, A., Toribio, I., Orozco, C., Puente, K. L., & Pérez-García, M. (2005). Neuropsychological functioning in methadone maintenance patients versus abstinent heroin abusers. *Drug and Alcohol Dependence*, 78(3), 283–288.
- Volkow, N. D., Baler, R. D., Compton, W. M., & Weiss, S. R. (2014). Adverse health effects of marijuana use. *New England Journal of Medicine*, 370(23), 2219–2227.
- Volkow, N. D., Wang, G. J., Telang, F., Fowler, J. S., Alexoff, D., Logan, J., ... Tomasi, D. (2014). Decreased dopamine brain reactivity in marijuana abusers is associated with negative emotionality and addiction severity. *Proceedings of the National Academy of Sciences of the United States of America*, 111(30), E3149–E3156.
- Vollenweider, F. X., Gamma, A., Liechti, M., & Huber, T. (1998). Psychological and cardiovascular effects and short-term sequelae of MDMA (“ecstasy”) in MDMA-naïve healthy volunteers. *Neuropsychopharmacology*, 19(4), 241–251.
- Wagner, D., Becker, B., Koester, P., Gouzoulis-Mayfrank, E., & Daumann, J. (2013). A prospective study of learning, memory, and executive function in new MDMA users. *Addiction*, 108(1), 136–145.
- Wagner, D., Tkotz, S., Koester, P., Becker, B., Gouzoulis-mayfrank, E., & Daumann, J. (2015). Learning, memory, and executive function in new MDMA users: A two-year follow-up study. *Frontiers in Neuroscience*, 9, 445.
- Woicik, P. A., Moeller, S. J., Alia-Klein, N., Maloney, T., Lukasik, T. M., Yeliosof, O., ... Goldstein, R. Z. (2009). The neuropsychology of cocaine addiction: Recent cocaine use masks impairment. *Neuropsychopharmacology*, 34(5), 1112–1122.
- Zakzanis, K. K., Campbell, Z., & Jovanovski, D. (2007). The neuropsychology of ecstasy (MDMA) use: A quantitative review. *Human Psychopharmacology: Clinical and Experimental*, 22(7), 427–435.

The Effects of Prenatal Stress on Offspring Development

Megan R. Gunnar and Colleen Doyle

*Institute of Child Development, College of Education and Human Development,
University of Minnesota, Minneapolis, MN, USA*

Introduction

In recent decades, research from a wide range of disciplines has shown prenatal development provides a foundation for learning and health throughout the lifespan. A growing body of work suggests that variation in the prenatal environment can produce individual differences and program regulatory systems that subsequently influence developmental organization and outcomes in infancy, childhood, and adulthood. This research provides evidence of long-term educational, behavioral, and psychological sequelae of *prenatal stress* (PS). In this work, prenatal stress is often used as an umbrella term for maternal experiences that include physiological and psychological responses to perceived threat or challenge, as well as negative mood states (frustration, anxiety, depression). The central hypothesis guiding PS research is that maternal experiences influence the *in utero* environment, alter the fetus's developing central nervous system (CNS), and subsequently set probabilistic parameters for future brain and behavior development. Although epidemiological, correlational, and quasi-experimental research with human subjects supports this *fetal origins hypothesis*, confounding variables prevent these studies from providing definitive proof. Instead, strong causal evidence for PS effects comes primarily from animal experiments. This area of research underscores the importance of delineating the effects and mechanisms of stress during the prenatal period in order to provide the optimal foundation for child development.

The goals of this article are to (a) outline the essential features of normal prenatal brain development in humans, (b) review the primary methods for studying brain development, (c) summarize findings on the effects and mechanisms of PS, and (d) consider factors that might buffer against PS and promote resilience. Finally, conclusions are discussed in the context of implications for efforts to support the health of pregnant women and their children.

Prenatal Brain Development

Brain development unfolds through a series of complex and dynamic processes involving the interaction of genes and the environment (see Stiles, 2008). Human prenatal CNS development is organized into the *embryonic period* (conception—gestational week [GW] 8) and the *fetal period* (GW9—birth). The first step in embryonic brain development is the emergence of stem cells called *neural progenitor cells* at GW2. Through the process of *gastrulation*, neural progenitors become capable of producing all types of cells for the brain and CNS. Following their transformation, the region of neural progenitor cells is called the *neural plate*. During GW3, the first brain structure, the *neural tube*, begins to form. Through the process of *neurulation*, ridges rise along the sides of the neural plate, arch together, and fuse. Before closure of the tube, the anterior end expands to form three primary *brain vesicles* that are precursors of the forebrain, midbrain, and hindbrain. By GW8, these subdivide to form five secondary brain vesicles that are the precursors of the medulla, pons, midbrain, diencephalon, and telencephalon. From GW4–8 the embryo grows in size tenfold. During this time of rapid development, *neurogenesis*, or neuron production, begins. Between GW6 and GW20, the brain produces about 250,000 cells per minute.

At the start of the fetal period, neuron production begins to drive dramatic changes in the brain's gross morphology. The smooth structure of the immature brain is transformed as gyral and sulcal folding emerge. Between GW8 and GW22, the longitudinal fissure that separates the right and left hemispheres develops. Additional primary sulci form between GW14 and GW26, secondary sulci emerge between GW30 and GW35, and the development of tertiary sulci extends from GW36 into the postnatal period. These foldings allow the fetal brain to dramatically increase cortical surface area. By GW20, most of the billions of neurons in the human brain have been produced. *Neural migration* ensues in two main waves, with peaks at GW8–10 and GW12. Through migration, neurons radially form the six-layered *neocortex* in an “inside out” fashion, so that deeper layers are populated and mature first, and more superficial layers are populated and mature last. *Cajal–Retzius cells* control the positioning of neurons by producing a molecule, *reelin*, that signals neurons to stop migrating. Around GW16 neurons reach their final position and begin extending axons and dendrites, which allow them to communicate with other neurons via *synapses*. Few synapses are formed until GW28, when the pace accelerates to approximately 40,000 synapses per minute. Through synaptic communication neurons establish functional neural circuits that mediate sensory and motor processing and underlie behavior. Around GW32–34, *myelination* begins. This involves the production of the fatty sheath that insulates nerve fibers and allows for cells to transmit information faster.

Along with cell proliferation, naturally occurring cell death plays an essential role in CNS development. *Necrotic cell death* eliminates cells that are damaged due to injury, and *apoptosis* eliminates cells that initiate an intrinsic suicide program. Since neurons that form connections with other neurons are more likely to survive, apoptosis is hypothesized to regulate the establishment of neural circuits. Apoptosis also eliminates cells like neural progenitor cells once their transitory functions are complete. During prenatal development normative cell death eliminates 50% or more of neurons.

Brain maturation begins a new phase at birth characterized by exuberant connectivity and refinement of neural circuitry. Between birth and the third year of life, gray matter increases from 50% of adult brain volume to 90%, and white matter myelination progresses to an adult-like pattern. Finally, *synaptic pruning* begins refining neural connectivity via plasticity processes that are influenced by the child's experience and environment (Greenough, Black, & Wallace, 1987).

Methods of Studying Fetal Brain Development

Animal models

Because experimental studies of typical and atypical fetal brain development are neither easy nor ethical with human subjects, animal models provide an essential tool for understanding PS effects. Animal experiments allow researchers to systematically manipulate the type, timing, and intensity of the stressor and test for differential effects on offspring development at the behavioral, anatomical, and cellular level of the brain. Stimuli used to stress the mothers include random light or noise, restraint, or removal from the home cage. Additionally, animal models provide an opportunity to tease apart pre- and postnatal influences on offspring development through cross-fostering designs. Although animal studies have used many mammals, the majority have been conducted on rodents. As a result, researchers often grapple with issues of translation when interpreting findings (Gottlieb & Lickliter, 2004).

Fetal physiological monitoring

For over seven decades researchers have studied indices of fetal physiological functioning, including fetal heart rate (FHR), fetal heart rate variability (FHRV), fetal movement (FM), and “coupling,” or the association between FHRV and FM (see DiPietro, Costigan, & Voegtline, 2015). Over the course of pregnancy, these indices change in a predictable way that is reflective of brain maturation. This is because the heart is innervated by fibers from both branches of the autonomic nervous system, which interact to regulate FHR and FHRV; sympathetic innervation increases FHR, while parasympathetic innervation reduces FHR. Early in gestation, FHR is predominately under the control of the sympathetic nervous system. As the CNS develops, the maturation of the parasympathetic branch allows progressive activity of the vagus nerve to slow the heart down and modulate FHR in coordination with motor functioning. As a result, over the course of pregnancy, FHR decreases and FHRV and cardiac-motor coupling increases. Researchers often use fetal physiological monitoring to characterize the adaptation and individual developmental trajectory of the CNS in the fetus. Typically, 30–50 min of fetal monitoring using electrodes applied to the mother’s stomach is required to detect within-individual stability and measure variations that emerge during fetal rest-activity cycles. There is evidence of rank-order stability of FHRV over gestation, suggesting continuity in individual differences in brain development is detectable during pregnancy. Additionally, by comparing typically progressing pregnancies with pregnancies characterized by intrauterine growth restriction and other conditions, researchers have identified deviations in fetal indices that reflect these conditions. Similarly, researchers have used fetal indices to examine possible deviations in fetal CNS development that are associated with PS effects. Finally, differences in fetal indices, independent of maternal factors, have been used to predict offspring behavioral and psychological development.

Fetal MRI

Since the development of magnetic resonance imaging (MRI) in the 1980s, researchers have used this technology to study fetal brain development (see Prayer et al., 2006). MRI uses a powerful magnetic field (MF) and radiofrequency (RF) pulse to align magnetically active nuclei (i.e., protons) and then knock the nuclei out of alignment. As the nuclei recover their alignment, they emit a radio signal that is converted into an image. Although MRI can generate detailed images depicting fetal brain anatomy, until recently it was not widely used due to safety concerns. Unlike other imaging methods, MRI does not involve radiation, which can directly kill cells or cause genetic mutations. Pregnant women cannot be scanned if they have

metal devices or implants. However, primary safety concerns for the fetus include risk due to exposure to MF or RF pulse or to increased heat or noise caused by the MRI. In addition to animal studies examining these concerns, several follow-up studies of children studied with fetal MRI have been carried out. Although no significant adverse effects have been discovered, researchers typically do not use MRI to study normal brain development until after GW18, when the major steps of organ production and development have been completed.

Currently, the most significant challenge to using MRI to study brain development is fetal motion, which can cause ghosting or blurring of the acquired MRI image. In life-threatening situations, the use of a sedative can reduce fetal motion; however, this is not a practical approach for general use. Instead, the ongoing development of faster imaging and motion correction techniques are essential to advancing fetal MRI methodology. Currently, research using this approach is in the nascent stage. Because of the rapid pace of fetal brain maturation, changes that can be detected with fetal MRI occur on the order of weeks or even days. As a result, it is difficult to study fetal brain development at a precise developmental stage. Therefore, in order to fully leverage fetal MRI to study how PS may alter brain development, an increased understanding of temporal and regional variability of events in normal brain maturation is needed.

Effects of Prenatal Stress on Offspring Brain and Behavior Development

The delineation of PS effects depends on the definition of stress itself (see Gunnar & Davis, 2013). *Stress* is conceptualized as a state of disharmony or threatened homeostasis that sets in motion physiological and behavioral responses to reestablish homeostasis. *Distress* is a negative psychological response to the perception of threatening, difficult, or stressful experiences that can include a variety of cognitive and affective states, such as uncertainty, frustration, anxiety, and depression. Thus PS is equated with negative emotional states. This is problematic for several reasons. First, PS can involve physical challenges that do not involve negative emotions, such as the pregnant woman doing physically demanding work that activates stress hormones. Second, not all emotionally negative states activate stress biology (Hennessy & Levine, 1979), suggesting that associations between mood dysregulation and fetal outcomes may involve biology beyond activation of stress-mediating systems. Third, because it is the interpretation of the event that mediates the impact of objective conditions on stress biology, when PS is defined by the event (i.e., exposure to a natural disaster), there can be a wide variety of individual responses.

Notably, several researchers have shown PS is neither specific to pregnancy nor stress. Correlations among self-report measures of stress and symptoms of depression and anxiety are high during pregnancy and remain stable through the first 2 years after delivery. These results suggest that measures of PS may gauge factors like personality traits, coping skills, and social support. Additionally, many environmental stressors, such as low socioeconomic status, marital/family conflict, and paternal death or incarceration, are likely to persist after pregnancy. Since these factors can influence maternal caregiving, investigators must measure and control for postnatal levels of stress in order to differentiate pre- and postnatal influences on child development.

To test for PS effects on offspring brain and behavior development, investigators have looked for converging evidence from animal and human studies. Here we summarize results in both domains of research.

Animal studies

Results from animal models show offspring exposed to PS exhibit heightened anxiety and fear of novelty, diminished exploration and social interaction, deficits in attention, learning and

memory, delays in motor development, and disturbances in motor behavior (stereotyped, non-directed motor behavior). These findings have been widely replicated in rats and rhesus monkeys using different stress paradigms. Also, PS alterations to offspring behavior have been associated with changes in brain development, including alterations to the hippocampus and amygdala, two areas of the brain that support learning, memory, and emotion regulation. These alterations include reduced neurogenesis, synaptic density, and dendritic length and complexity. Additionally, research shows PS alters the neurodevelopmental trajectory of the amygdala, such that animals exposed to PS exhibit initially reduced and subsequently increased amygdala volume. Offspring exposed to stress at the end of gestation also shows hypermyelination. Finally, animal experiments show that PS induces alterations to hypothalamic–pituitary–adrenocortical (HPA) axis functioning including elevated basal corticosterone levels, heightened HPA axis reactivity to acute stress, and decreased negative feedback of the HPA axis. These physiological alterations are associated with decreased binding capacity of both mineralocorticoid and glucocorticoid receptors in the brain.

Additionally, animal models suggest some sex-specific effects of PS. While males exposed to PS show deficits on tasks involving learning and spatial working memory, there are conflicting reports of learning deficits and learning enhancements for females. Male offspring with learning deficits show decreased neurogenesis in the dentate gyrus of the hippocampus, while female offspring with increased anxiety show decreased neurogenesis in the ventral hippocampus.

Most research on stress posits that mild forms of stress can have a positive effect on development and the organism's ability to respond to stress in the future. Similarly, animal models show differential effects of PS depending on exposure level. Although daily maternal restraint of 1 hr will reduce dendritic length and complexity in the brains of adult male rat offspring, restraint of only 30 min will increase dendritic length and complexity. There are no known equivalent studies with adult female rats. Other studies have shown enhanced learning ability of both male and female offspring following mild PS. Additionally, animal models show that fetuses are affected differently when subjected to a random versus predictable stress protocol. In response to a random stressor, glucocorticoid levels increased in maternal and fetal blood after each stress exposure. In contrast, levels no longer increased by the third day of a predictable stress protocol. These results suggest researchers should assess whether animals (and correspondingly, humans) adapt to certain types of PS experiences.

Animal designs that cross-foster pups from a stressed mother onto a control mother (and vice versa) have shown independent effects of pre- and postnatal influences on offspring development. Results from these studies suggest that chronic, unpredictable stress at the end of pregnancy can increase anxious behavior of mothers until after pups are weaned. During this time, persisting PS effects can decrease maternal caretaking behavior and increase glucocorticoids passed to offspring through milk. After cross-fostering, PS pups reared by control mothers showed reduced anxiety, and control pups reared by stressed mothers showed increased anxiety. Additionally, male PS rats reared by control mothers showed normalized HPA axis functioning. However, rearing by a control mother did not ameliorate learning deficits in PS male offspring. These results suggest that PS effects on learning may be prenatally mediated.

Quasi-experimental human studies

Prospective studies involving pregnant women who experienced natural or man-made disasters have found persisting effects on child outcomes attributed to PS. Notable programs of research have followed children whose mothers experienced an ice storm, hurricane and tropical storms, major floods and earthquakes, or an act of terrorism or war. These studies have attempted to

disentangle the effects of objective stressors (i.e., days without power), subjective reactions (e.g., distress, perceived stress), and physiological stress reactivity (e.g., cortisol over 24 hr). Additionally, retrospective studies have examined PS effects in children whose mothers experienced disasters like the Dutch Hunger Winter of 1944–1945.

Results from these quasi-experimental studies have linked PS to increased prevalence rates of neurodevelopmental disorders (attention-deficit hyperactivity disorder, autism, schizophrenia), psychiatric disorders (depression, anxiety), difficult temperament, increased behavior problems, and deficits in attention, language, and motor development. Elevated subjective distress (and not objective stress) was associated with increased *internalizing problems* (anxiety, depression, social withdrawal) that remained stable from ages 4–11 years old. High subjective distress was also associated with increased *externalizing problems* (aggression, hyperactivity) that diminished over development relative to published norms. Although exposure to severe stress during an ice storm was associated with IQ, memory, and language deficits at 2, 5, 8, and 11 years old, moderate levels of stress were associated with enhanced cognitive abilities. Recent findings have shown brain differences might be associated with PS and/or behavioral alterations in these cohorts. Objective but not subjective PS predicted smaller right hippocampus volumes in males at age 11; no effects of PS were detected for females. In comparison to age-matched unexposed controls, PS exposure related to the Dutch Hunger Winter was associated with smaller brain volume in males but not females at age 68. Altogether, findings from quasi-experimental studies echo results of animal models showing PS effects may depend on stressor type, timing, and intensity, as well as offspring sex.

Correlational human studies

Early correlational research on PS effects focused on offspring *birth outcomes* (gestational age at delivery, birth weight, Apgar scores) that are thought to reflect adverse *in utero* exposures and fetal development. This work led to a widely replicated finding linking PS to shorter gestation and low birth weight. Although the broader interest of this research was the possible developmental sequelae of birth outcomes, initially very few longitudinal, prospective datasets were available to assess behavioral and psychological functioning beyond the neonatal period. Today, several datasets allow researchers to examine long-term outcomes. Although mounting correlational evidence suggests that PS is associated with persisting educational, behavioral, and psychological problems, caution is required in assuming causation.

Results of correlational human research on PS effects are varied. However, major findings that have been replicated by several different investigators may be organized and interpreted within the developmental psychopathology principles of equifinality and multifinality (Cicchetti & Rogosch, 1996). *Equifinality* suggests that a single developmental outcome (i.e., ADHD) may be influenced by many different factors. Accordingly, findings have linked a single type of offspring outcome (inattention/hyperactivity) to multiple types of PS (elevated levels of maternal stress, depression, anxiety, trait anxiety, and pregnancy-specific anxiety). Other outcomes that have been independently linked to these five types of PS include difficult temperament, increased negative behavioral reactivity, and increased anxiety. Also, lower verbal IQ has been independently associated with both elevated maternal anxiety and depression. In contrast, the principle of *multifinality* suggests that one developmental factor can lead to many different outcomes. Accordingly, high maternal anxiety has been associated with different offspring outcomes, including different types of internalizing and externalizing problems, and poor performance on executive function and decision-making tasks.

Correlational research findings also suggest that offspring sex and intensity and timing of PS exposure may induce different effects on development, some of which may be positive. Overall,

PS findings suggest males may be more vulnerable to adverse *in utero* effects, with female outcomes being more variable. However, some reports contradict this. Although elevated maternal anxiety and cortisol early in pregnancy have been associated with deficits in infant cognition, moderate and high levels of PS late in pregnancy have been associated with enhanced infant cognitive maturation and abilities. Additionally, the timing of PS exposure has been related to alterations in offspring brain structure and connectivity development. Studies assessing PS effects in middle childhood have linked reductions in offspring gray matter volume to high levels of PS in the second (but not first or third) trimesters, including maternal anxiety and depression. Also, high levels of maternal stressful life events in the second trimester have been associated with precocious white matter tract development in the *uncinate fasciculus*, an area of the brain linked to behavioral disorders, including antisocial behavior and autistic spectrum conditions. Some investigators have interpreted this neurodevelopmental profile of reduced gray matter and precocious white matter as indicative of aberrant accelerated brain maturation induced by PS. Finally, correlational research has shown an association between PS and altered offspring amygdala development. Increased maternal prenatal cortisol levels have been associated with increased offspring amygdala volume at age 10. Also, elevated maternal depressive symptoms have been associated with atypical amygdala functional connectivity in 6-month olds that is consistent with patterns observed in adolescents and adults with major depressive disorder.

Correlational studies that have measured PS in the pre- and postnatal periods show that although many effects of PS are attenuated after controlling for postpartum measures, PS is independently associated with adverse child outcomes. Fetal studies provide additional, proximal evidence supporting putative effects of PS on offspring CNS development. Human and animal research has shown rapid fetal response to experimental manipulations designed to change maternal psychological state (both for induced stress and induced relaxation). Further, within the context of expectable prenatal CNS development (decreased FHR, increased FHRV, and cardiac-motor coupling), maternal distress (anxiety, depression, and negative mood) has been associated with variations in fetal indices and their rate of change over the course of gestation. Several studies suggest that PS is associated with an accelerated pace of fetal CNS maturation and more mature neural integration at birth.

It is possible that maternal self-report of PS and offspring outcomes in the pre- and postnatal period reflect a shared genetic contribution, instead of a causal influence of the prenatal environment. Comparisons between children conceived via *in vitro* fertilization using embryos from the mother and those conceived with donor embryos have been used to tease apart genetic and putative PS effects on *in utero* development (Rice et al., 2010). Results suggest that offspring inattention/hyperactivity may be due to a shared inheritance of characteristics, rather than PS effects on the *in utero* environment. In contrast, lower birth weight, shortened gestation, and offspring antisocial behavior were linked to PS in related and unrelated dyads, suggesting an *in utero* environment effect. PS was also linked to offspring anxiety; however this association was not significant after accounting for postnatal measures of maternal anxiety and depression. These findings underscore the importance of considering confounding genetic and postnatal factors in PS research.

Mechanisms of PS Effects

Despite strong evidence of PS effects from animal models, and compelling results from human studies, our understanding of the biological mediators of PS is incomplete. Research examining possible mechanisms of PS has focused on the endocrine, immune, and vascular systems, which play a major role in the response and adaptation to stress.

Maternal endocrine system

The maternal HPA axis has been the most widely investigated mediator of PS effects. PS is hypothesized to produce alterations in offspring brain and behavior development through the action of glucocorticoids. In human studies, however, few data show the HPA axis is the smoking gun behind PS effects. As previously reviewed, measures of maternal HPA axis functioning and PS or distress measures have independently been associated with offspring outcomes. However, with few exceptions researchers have failed to find an association between maternal stress hormones and measures of maternal distress in human studies (see Beijers, Buitelaar, & de Weerth, 2014).

Animal experiments, however, do show stress-induced maternal glucocorticoids mediate some offspring outcomes. By adrenalectomizing pregnant rats prior to the initiation of stress, and injecting them with either saline or maintenance levels of glucocorticoids, researchers were able to block glucocorticoid production in response to stress. Stress exposure in the absence of stress hormones prevented alterations typically observed in offspring, including increased anxiety, learning deficits, and HPA dysregulation. Moreover, when investigators administered additional glucocorticoids to adrenalectomized pregnant rats to simulate a stress response, they were able to restore increased anxiety and HPA dysregulation in offspring. However, this manipulation did not restore offspring learning deficits. These findings suggest that glucocorticoids mediate *some* PS effects seen in animal models and that other biological mechanisms likely play a role.

Accordingly, animal and human studies suggest that maternal thyroid hormone (TH) may partially mediate PS effects. TH is a biologically plausible mechanism for PS effects because TH deficiency is associated with mood disturbance, and maternal TH also helps to regulate multiple prenatal neurodevelopment processes, including neurogenesis, neuronal migration, axonal and dendritic growth, synaptogenesis, and myelination. Insufficient maternal TH levels, especially early in development when the fetus relies entirely on maternal TH production, are associated with alterations in offspring brain development and persisting behavioral deficits. Research has shown children of women with untreated TH deficiency at the beginning of the second trimester exhibit lower IQ and deficits in language, attention, and learning that echo PS effects seen in animal and human studies.

Maternal immune system

Recent research has highlighted a complex interplay between the maternal HPA axis and immune system and suggested that maternal inflammation during pregnancy is a likely mechanism of PS effects. Maternal inflammation may alter offspring development through small immune signaling proteins called *cytokines*. Animal models show that cytokines are released into the blood in response to infection, immune activation, and stress. Additionally, animal and human studies have shown that PS is positively correlated with higher circulating levels of proinflammatory cytokines and that cytokines in maternal circulation can cross the placenta and access the fetal brain. Research suggests cytokine signaling pathways may help regulate offspring neurodevelopmental events including neuronal differentiation and migration and axon and synapse formation. Animal and human studies have linked many types of infections and pathogens to offspring outcomes, prompting the hypothesis that maternal immune activation may be a common mechanism underlying these effects. Additionally, rodent models that use agonists of innate immune system receptors to stimulate a proinflammatory cascade of cytokines in the maternal immune system (in the absence of an actual pathogen) have provided evidence that maternal immune activation and not direct fetal infection alters offspring development. Recent research suggests the balance of maternal pro- and anti-inflammatory cytokine

production may influence offspring outcomes; manipulating the maternal immune system to overexpress IL-10, an anti-inflammatory cytokine prevented behavioral abnormalities in offspring.

Maternal vascular system

Although there is no direct vascular connection between mother and offspring, maternal autonomic responses (heart rate, blood pressure) and hemodynamics (circulation of blood) influence the offspring via the placenta. Animal research has shown that PS releases adrenaline and noradrenaline into circulation, which can reduce placental blood flow and fetal oxygenation. Through prolonged *hypoxia*, or oxygen deficiency, PS can alter offspring brain and behavior development. Human studies suggest that due to the vasculature of the developing brain, the areas most vulnerable to hypoxic injury are subcortical structures, including the amygdala, hippocampus, thalamus, and hypothalamus. Furthermore, research shows these tissues may be most vulnerable to hypoxia prior to GW24. Hypoxia has been associated with offspring outcomes linked to measures of PS and distress, such as learning and behavioral problems, as well as peripheral nerve damage, hearing deficits, and seizures. Altogether, these findings suggest that maternal vascular mechanisms may be most likely to mediate PS effects on development in the first and second trimester of pregnancy.

The placenta

As previously indicated, the “maternal–placental–fetal unit” is a complex, dynamic system that facilitates the exchange of hormones, cytokines, oxygen, and nutrients between mother and offspring. As a result, the placenta is a likely mediator of PS effects. The placenta is the first fetal organ to develop, and for the majority of pregnancy, it is approximately one cell thick. During a normally progressing pregnancy, only a small amount of maternal stress hormones reach the fetal brain. Approximately 90% of circulating corticosteroids are sequestered by a corticosteroid-binding globulin (CBG) before they access the fetus, and most of the remaining 10% is converted to inactive metabolites by the placental enzyme 11 β -hydroxy steroid de-hydrogenase-2 (11 β -HSD-2). However, these dynamics shift at the end of gestation, allowing a surge in fetal cortisol levels to drive maturation of the fetal lungs and possibly help initiate labor. Research suggests that chronic PS reduces the level of CBG in rodents and downregulates the activity of 11 β -HSD-2 in rodents and humans. Thus, altered placental functioning as a result of PS may play a role in offspring outcomes by allowing elevated levels of glucocorticoids to cross the placental barrier.

Additionally, the placenta plays a fundamental role in organizing the development of the fetal CNS through the production of placental corticotrophin-releasing hormone (pCRH). pCRH has been shown to regulate the maturation of the fetal HPA axis and other systems and modify the timing of labor onset and delivery. Maternal cortisol stimulates pCRH production, which in turn stimulates cortisol production. This positive feedback loop can progressively amplify pCRH and cortisol production during pregnancy. Thus, research suggests pCRH trajectory (i.e., rate of production acceleration), rather than absolute concentration of pCRH, may be an important indicator of PS alterations to fetal development. Notably, because the placenta produces pCRH in primates, but not other mammals, researchers are unable to study this mechanism in rodent models.

An emerging body of work has suggested that placental factors may mediate sex-specific PS-related offspring outcomes. As a result of sex-specific differences in the structure and function of the placenta, research suggests that female fetuses may make multiple adaptations in response to an adverse *in utero* environment, while males may show fewer signs of *in utero*

adaptation (see Clifton, 2010). These findings align with results from animal and human literature showing that males may be more vulnerable to adverse PS-related outcomes, while females show a more varied response.

Offspring regulatory systems

Animal and human studies suggest that PS disrupts normal development of homeostatic regulatory systems in the offspring, leading to persisting dysregulation of the offspring's own endocrine, autonomic, and immune systems. This dysregulation is hypothesized to subsequently play a role in inducing adverse behavioral outcomes. Findings from animal work support this hypothesis. Mechanistically, PS has been shown to alter the number of glucocorticoid receptors in the offspring hippocampus, an area important in terminating the stress response. Rodent and nonhuman primates exposed to PS show a heightened, prolonged elevation of corticosterone in response to stress, and several investigators have replicated this finding in humans. Similar "programming" effects have been reported for the immune system. Injections of IL-6 into pregnant mice induced chronically elevated levels of IL-6 in adult offspring. Additionally, high levels of objective PS during an ice storm were associated with an immune profile biased toward a proinflammatory response in 13-year-old children.

Toward Resilience and Intervention

Not all women and children who experience high stress during pregnancy show biological evidence of this stress. Multiple factors may influence maternal and offspring susceptibility to stress effects, including genetic predispositions, individual differences in personality and temperament, and lifestyle factors. Research suggests that variation in the interpretation of a stimulus as stressful depends on the characteristics of the individual experiencing the stimulus. Accordingly, researchers interested in PS effects are beginning to assess maternal personality traits, as well as individual differences in maternal threat perception and reaction. Additionally, research suggests that environment and lifestyle factors also contribute to maternal resilience. Social support has been shown to "buffer" or reduce levels of self-reported stress during pregnancy. Lifestyle factors like exercise have been shown to promote resilience and healthy outcomes for pregnant women and their children. Notably, research also shows that postnatal caregiving experiences can play an important role in reducing or reversing PS effects. As reviewed here, fostering prenatally stressed (PS) pups onto control mothers after birth has been shown to prevent adverse offspring outcomes.

Conclusions

For many generations, the human brain was considered to be a *tabula rasa*, or blank slate, at birth. However, a growing body of research suggests brain and behavior development are already underway by delivery. Undoubtedly, animal models and other recently developed methods have been crucial to opening a new window into early brain growth that previously has been inaccessible. Our growing understanding of both typical and atypical fetal brain development has provided an important foundation for research investigating the "fetal origins" hypothesis that maternal health, emotions, and stress levels have long-term effects on offspring outcomes. Research shows that PS interferes with normal CNS development and induces brain perturbations that result in persisting effects on offspring functioning. Affected regions of the brain identified in this literature include the amygdala, hippocampus, and

uncinate fasciculus. Additionally, PS is associated with reduced gray matter volume and hypermyelination. Central findings on offspring behavioral outcomes have associated PS with increased offspring anxiety, fear, and inhibition in the face of novelty, as well as deficits in attention, learning, memory, and motor maturation. Importantly, moderate PS has been associated with enhanced cognitive and motor maturation and ability level. Besides PS intensity, research shows that PS effects may depend on stress type, timing of exposure, and offspring sex. Although results are varied, findings suggest that PS exposure in early or mid-gestation is associated with increased risk for adverse offspring outcomes. It is possible the offspring is more vulnerable during early gestation when disruption to the rapidly developing architecture of the brain may have cascading effects. Currently, there is no unifying definition of PS, and many investigators confound PS with distress. This may help explain why human studies have had difficulty detecting a biological mediator of PS effects. However, research suggests that major regulatory systems that play a role in the adaptation and response to stress, including the maternal endocrine, immune, and vascular system, are likely mechanisms of PS effects.

Maternal experiences during the pregnancy have profound effects on development, which are not yet well understood by researchers, clinicians, and policy makers. The growing PS literature may identify new opportunities to support the health and resilience of women and their children. However, scientists have the duty to deliver findings to women in a clear way that does not promote scaremongering. Importantly, correlational research may be suggestive but is *not* strong evidence of a causal relationship. Based on currently available evidence, advisory panels such as the March of Dimes recommend that stress during pregnancy should be reduced, if possible. This might be accomplished by helping pregnant women build a strong support network, form a plan to cope with anticipated challenges, practice stress reduction techniques (mediation, yoga), and maintain healthy habits (sleep, exercise, nutrition). As PS research shows, supporting the health of women during pregnancy can help promote the optimal development of their children over the lifespan.

Author Biographies

Megan R. Gunnar is a regents professor and Distinguished McKnight University professor at the University of Minnesota. She is the director and chair of the Institute of Child Development, the interim director of the Center for Early Education and Development, and the associate director of the Center for Neurobehavioral Development. She received her PhD in Developmental Psychology at Stanford University and then completed a postdoctoral fellowship in stress neurobiology at Stanford Medical School. In 1979 she came to the U of M as an assistant professor moving through the ranks to full professor by 1990. Professor Gunnar has spent her career studying how stress affects human brain and behavioral development and the processes that help children regulate stress. She is the recipient of lifetime achievement awards from the American Psychological Association, Division 7 Developmental Psychology, and the Society for Research in Child Development and a lifetime mentor award from the Association for Psychological Science. Nationally she is a member of the Harvard National Scientific Council on the Developing Child that translates developmental science into language that communicates with policy makers. Internationally she is a member of the Canadian Institute for Advanced Research's Program on Child and Brain Development, a group working on how early experiences "gets under the skin" to influence lifelong health and well-being. In addition, she chairs the Research Advisory Council for the Minnesota Children's Museum and is a consultant on stress and development for the Greater Minneapolis Crisis Nursery.

Colleen Doyle is a doctoral student and National Science Foundation Graduate Research Fellow at the University of Minnesota, Institute of Child Development. Colleen received her undergraduate education at the University of Chicago and earned a Masters of Fine Arts in poetry at Boston University, before switching careers to examine the human experience empirically. Prior to beginning graduate work in developmental psychology, Colleen worked as a research assistant with Dr. Catherine Monk at Columbia University Medical Center studying the effects of fetal programming. Currently, Colleen's research interests focus on understanding how *in utero* developmental processes influence fetal central nervous system maturation, infant brain and behavioral development, and health and well-being throughout the lifespan.

References

- Beijers, R., Buitelaar, J. K., & de Weerth, C. (2014). Mechanisms underlying the effects of prenatal psychosocial stress on child outcomes: Beyond the HPA axis. *European Child & Adolescent Psychiatry*, 23(10), 943–956.
- Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, 8(04), 597–600.
- Clifton, V. L. (2010). Review: Sex and the human placenta: Mediating differential strategies of fetal growth and survival. *Placenta*, 31(Suppl), S33–S39.
- DiPietro, J. A., Costigan, K. A., & Voegtline, K. M. (2015). *Studies in fetal behavior: Revisited, renewed, and reimagined. Monographs of the Society for Research in Child Development*, 80(3), 1–94. doi:10.1111/mono.v80.3
- Gottlieb, G., & Lickliter, R. (2004). The various roles of animal models in understanding human development. *Social Development*, 13(2), 311–325.
- Greenough, W. T., Black, J. E., & Wallace, C. S. (1987). Experience and brain development. *Child Development*, 58(3), 539–559.
- Gunnar, M. R., & Davis, E. P. (2013). The effects of stress on early brain and behavioral development. In J. L. R. Rubenstein & P. Rakic (Eds.), *Comprehensive developmental neuroscience: Neural circuit development and function in the healthy and diseased brain* (pp. 447–465). Amsterdam, the Netherlands: Academic Press.
- Hennessy, J. W., & Levine, S. (1979). Stress, arousal, and the pituitary-adrenal system: A psychoendocrine hypothesis. *Progress in Psychobiology and Physiological Psychology*, 8, 133–178.
- Prayer, D., Kasprian, G., Krampl, E., Ulm, B., Witzani, L., Prayer, L., & Brugger, P. C. (2006). MRI of normal fetal brain development. *European Journal of Radiology*, 57(2), 199–216.
- Rice, F., Harold, G. T., Boivin, J., Van den Bree, M., Hay, D. F., & Thapar, A. (2010). The links between prenatal stress and offspring development and psychopathology: Disentangling environmental and inherited influences. *Psychological Medicine*, 40(02), 335–345.
- Stiles, J. (2008). *The fundamentals of brain development: Integrating nature and nurture*. Cambridge, MA: Harvard University Press.

Psychophysiology of Traumatic Stress and Posttraumatic Stress Disorder

Michael G. Griffin, Brittany F. Goodman,
Rebecca E. Chesher, and Natalia M. Kecala

*Department of Psychological Sciences, Center for Trauma Recovery,
University of Missouri–St. Louis, St. Louis, MO, USA*

Diagnostic criteria for posttraumatic stress disorder (PTSD) were introduced in third edition of Diagnostic and Statistical Manual for Mental Disorders (DSM-III) (American Psychiatric Association [APA], 1980). Of course, scientific studies of trauma reactions extend back to Janet's work at the turn of the last century and Kardiner's (1941) description of "physioneurosis" among veterans of World War I. Seminal studies targeting psychophysiological biomarkers of traumatic stress reactions served a key role in mitigating initial skepticism from clinicians, scientists, and the community that questioned the validity of PTSD as a distinct diagnostic entity (Yehuda & McFarlane, 1995).

The DSM-III diagnostic criteria included three symptom clusters composed of reexperiencing, avoidance, and hyperarousal symptoms. Most of the biological research into the disorder was focused on the hyperarousal symptoms, such as exaggerated startle and hyperreactivity to trauma cues. Historically, the DSM diagnostic system has defined physiological reactivity in PTSD in broad terms, such as "heightened startle reactivity" and "increased physiological reactivity to trauma-related cues or reminders of the traumatic event."

The current conceptualization of PTSD as detailed in the DSM-5 (APA, 2013) includes four clusters: (a) intrusion or reexperiencing symptoms, such as intrusive thoughts or memories, nightmares, or flashbacks to the traumatic experience; (b) avoidance symptoms, which include avoidance of thoughts, feelings, people, or situations connected to the trauma; (c) negative mood and cognitions arising from the traumatic experience, including negative self-appraisal, self-blame, shame, detachment from others, and a loss of interest in previously enjoyable activities; and (d) hyperarousal symptoms, such as hypervigilance to trauma cues, exaggerated startle response, irritability and anger, and difficulties with sleep.

The current conceptualization of PTSD is highly informed by results from structured, empirical investigations of psychophysiological alterations that follow a traumatic event (Pole, 2007). However, a disproportionate number of these studies only included male combat veterans, the results of which do not readily generalize to females or other trauma populations. This trend is beginning to shift, with new studies aiming to establish explanatory models of PTSD from cardiac activity (heart rate [HR], heart rate variability [HRV]), sweat gland activity (skin conductance [SC]), and facial electromyogram (EMG) activity of facial muscles as indices of negative emotional states (e.g., frontalis or corrugator) or startle response (orbicularis oculi).

Broadly, the early psychophysiological studies of PTSD utilized one or more of the following paradigms: (a) resting physiological state, (b) responses to standard trauma cues (e.g., sound of bombs exploding, gunfire, or video of car crashes) or personalized trauma cues (e.g., scripts of the individual's traumatic event), or (c) startle reactivity to sudden loud auditory stimuli (typically pure tones).

The large collection of early work by Pitman, Orr, and colleagues contributed significantly to the knowledge base of psychophysiological trauma reactions in all three domains. Of special note was their work on the reactions to personalized trauma cues that was derived from Lang's bio-information theory of emotion networks (Lang, 1979). Specifically, this led to the development of an auditory script-driven imagery paradigm of trauma and neutral cues that were recorded and played back in the psychophysiological laboratory while measurement of autonomic nervous system reactivity was recorded (e.g., Pitman, Orr, Forgue, de Jong, & Claiborn, 1987).

An important meta-analysis of the early psychophysiological research (Pole, 2007) reported significantly larger effect sizes for higher physiological reactivity when comparing individuals with PTSD to trauma-exposed participants without PTSD. Additionally, a psychophysiological signature emerged, characterized by the absence of SC habituation to startling tones and a facial EMG pattern of negative emotional state in response to idiographic trauma cues. Notably, HR reactivity was the most consistent indicator of PTSD hyperreactivity, consistent with observations that elevated HR immediately following a traumatic event predicts the risk of PTSD (e.g., Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2011).

Recent Developments in the Psychophysiology of Traumatic Stress and PTSD

Recent studies have focused on the role of the parasympathetic nervous system in PTSD. The vast majority of prior studies focused on the sympathetic nervous system, with little attention directed at the parasympathetic system, largely due to the insensitivity of measures (e.g., SC and HR) to parasympathetic tone and drive. However, it is now possible to quantify parasympathetic nervous system activity through measures such as HRV (Task Force of the European Society of Cardiology and the North American Society for Pacing and Electrophysiology, 1996). Innovative work by Porges (2011) provides a theoretic framework (i.e., polyvagal theory) for this line of inquiry. A second area of current interest is fear processing and the elucidation of brain fear circuits that drive PTSD symptoms. These research directions emphasize brain mechanisms that align with the National Institute of Mental Health initiative "Research Domain Criteria (RDoC)" to delineate linkages between biological factors, neural circuits, and clinical phenotypes (Insel *et al.*, 2010).

The Polyvagal Theory and Traumatic Stress

Based on phylogenetic development of the nervous system, the polyvagal theory suggests that an individual's reaction to a challenge or life threat depends on activation of the underlying system. Two separate vagal systems have been identified: the unmyelinated vagus that is phylogenetically older and originates in the dorsal motor nucleus and the myelinated vagus that originates in the nucleus ambiguus (NA). According to this theory, the sympathetic nervous system activates the "fight-or-flight" response. The older vagal system is associated with immobilization or freezing behavior, while the newer pathway is linked to social aspects of survival and control of HR. Output from this system follows a respiratory rhythm that allows for noninvasive monitoring of respiratory sinus arrhythmia (RSA) as a measure of HRV secondary to breathing dynamics.

The polyvagal theory suggests that the activation of these systems follows the Jacksonian principle of dissolution, such that when an organism is faced with a challenge, the phylogenetically newest system is engaged first, followed by the older system if needed to secure safety. Neuroception, the unconscious detection of threat through temporal brain regions, determines which system is to be engaged. Consistent with this theory, trauma may induce a deficit in neuroception, which leads to incorrectly interpreting a safe situation as dangerous. Trauma survivors may then rely on the sympathetic nervous system more readily or have a lowered threshold for engagement of the sympathetic nervous system. Future RSA-targeted studies will help inform the role of neuroception in PTSD.

The polyvagal theory describes a harmonized synchrony between the sympathetic and parasympathetic systems. Specifically, the myelinated vagus serves as a "vagal break" on HR via the SA node. The myelinated vagus maintains HR at levels that are optimal for restoration and regrowth. When faced with a challenge, the vagal brake is released allowing for increased HR and mobilization of needed resources. Prolonged threat activates the sympathetic nervous system via the adrenal system. Persistence of the threat leads to freezing, fainting, or dissociative behaviors governed by the unmyelinated vagal system (Porges, 2011).

Faulty neuroception may lead to chronic activation of sympathetic drive and maladaptive social behavior. Preliminary evidence suggests engagement of the myelinated vagal system via music therapy leads to better regulation of the viscera and an increase spontaneous social behavior (Porges, 2011).

Specific findings examining alterations in the parasympathetic nervous system show that individuals with PTSD exhibit decreased HRV during trauma recall (Keary, Hughes, & Palmieri, 2009) at baseline, and during affective induction (Hauschildt, Peters, Moritz, & Jelinek, 2011). Further, there is evidence for higher basal HR with PTSD that may be driven by a reduction in parasympathetic input (Hopper, Spinazzola, Simpson, & van der Kolk, 2006). While promising, additional studies of the polyvagal theory and the role of altered vagal/parasympathetic responses are needed.

Traumatic Stress and the Psychophysiology of Fear Processing

Theoretical models of fear processing and PTSD emphasize the role of the amygdala (Norrholm & Jovanovic, 2018). A traumatic event represents an unconditioned stimulus that elicits an unconditioned fear response, driven by activation of the sympathetic nervous system. While some traumatic events are repetitive, a single event is sufficient to elicit a

conditioned physiological and emotional fear response even in the context of overt safety. For example, a combat trauma survivor may have an extreme physiological reaction to fireworks, despite the absence of an imminent threat or danger.

Psychophysiology and neuroimaging research demonstrate heightened amygdala activation in response to trauma cues in PTSD (Shin, Rauch, & Pitman, 2006). In addition, hyporeactivity in the medial prefrontal cortex (mPFC) inhibits amygdala activation in participants with PTSD, along with smaller volume and decreased functionality of the hippocampus (Shin et al., 2006). Without inhibition from the mPFC, hyperactivity in the amygdala creates the fear and anxiety symptoms in PTSD (Davis, 1992).

The “learning” conceptualization of PTSD proposes that the conditioned response is reinforced by avoidance of trauma cues, preventing extinction through operant conditioning. There is little empirical research to support avoidance as a mechanism inhibiting extinction in PTSD (Lissek & van Meurs, 2015). However, extinction learning in PTSD is not retained, as measured using physiological reactivity on the day following extinction learning. This deficit in retention corresponds to reactivity in the mPFC (Lissek & van Meurs, 2015).

Non-associative learning theories have also been suggested as a means to explain hyperarousal symptoms in PTSD. Individuals with PTSD exhibit poor habituation to repeated stimuli such as loud tones (Pole, 2007). There is also evidence of sensitization in PTSD with enhanced responses following repeated activation, for example, with greater HR responses to repeated loud tones and greater amygdala activation while viewing fearful or neutral faces (Lissek & van Meurs, 2015).

Future Directions

Two developing areas for psychophysiological investigation in PTSD are (a) reorientation toward mechanisms of physiological recovery (allostasis) (Ottaviani, 2018) and away from an exclusive focus on reactivity. Recently, Goodman and Griffin (2018) used HR recovery to significantly improve the ability to predict the risk for developing PTSD in a longitudinal design and (b) the use of psychophysiological indices as biomarkers of fear extinction in treatment studies. Clinical trials using a virtual reality environment (Diemer, Muhlberger, Pauli, & Zwanzger, 2014) and startle measurement demonstrate the high sensitivity of psychophysiological measures to treatment response (Griffin, Resick, & Galovski, 2012; Robinson-Andrew et al., 2014). Pretreatment psychophysiological reactivity also predicts posttreatment outcome (Norrholm et al., 2016). Finally, measurement and biofeedback using HRV is an effective treatment option for PTSD (Tan, Dao, Farmer, Sutherland, & Gevirtz, 2011; Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009).

Similarly, targeted training to manage visceral activity elicited by a visual threat allows for the creation of new associations and realignment of fear network response (Norrholm & Jovanoovic, 2018). Application of these strategic interventions represents a potentially transformative focus of new clinical research studies.

Author Biographies

Michael G. Griffin, PhD, has research interests in the area of traumatic stress and posttraumatic stress disorder (PTSD) and biological alterations associated with the disorder. He is interested in studying psychophysiological and psychoendocrine changes in sexual and physical assault survivors.

Brittany F. Goodman, MA, is a doctoral candidate with research interests related to risk factors for PTSD development and testing the polyvagal theory in trauma populations.

Rebecca E. Chesher, MA, is a doctoral candidate with research interests in PTSD and understanding psychological and physiological factors that occur with the dysregulation of sleep.

Natalia M. Kecala, MA, has research interests in peritraumatic reactions in PTSD and how maladaptive coping strategies may be a risk factor for the disorder.

References

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Arlington, VA: American Psychiatric Publishing.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Bryant, R. A., Creamer, M., O'Donnell, M., Silove, D., & McFarlane, A. C. (2011). Heart rate after trauma and the specificity of fear circuitry disorders. *Psychological Medicine*, 41(12), 2573–2580. doi:10.1017/s0033291711000948
- Davis, M. (1992). The role of the amygdala in fear-potentiated startle: Implications for animal models of anxiety. *Trends in Pharmacological Sciences*, 13, 35–41.
- Diemer, J., Muhlberger, A., Pauli, P., & Zwanzger, P. (2014). Virtual reality exposure in anxiety disorders: Impact on psychophysiological reactivity. *World Journal of Biological Psychiatry*, 15, 427–442.
- Goodman, B. F., & Griffin, M. G. (2018). Prospectively predicting PTSD status with heart rate reactivity and recovery in interpersonal violence survivors. *Psychiatry Research*, 259, 270–276. doi:10.1016/j.psychres.2017.10.036
- Griffin, M. G., Resick, P. A., & Galovski, T. E. (2012). Does physiologic response to loud tones change following cognitive-behavioral treatment for posttraumatic stress disorder? *Journal of Traumatic Stress*, 25, 25–32.
- Hauschildt, M., Peters, M. J. V., Moritz, S., & Jelinek, L. (2011). Heart rate variability in response to affective scenes in posttraumatic stress disorder. *Biological Psychology*, 88, 215–222.
- Hopper, J. W., Spinazzola, J., Simpson, W. B., & van der Kolk, B. A. (2006). Preliminary evidence of parasympathetic influence on basal heart rate in posttraumatic stress disorder. *Journal of Psychosomatic Research*, 60, 83–90.
- Insel, T. R., Cuthbert, B. N., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., ... Wang, P. (2010). Research Domain Criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, 167, 748–751. doi:10.1176/appi.ajp.2010.09091379
- Kardiner, A. (1941). *The traumatic neuroses of war*. New York, NY: Paul Hoeber.
- Keary, T. A., Hughes, J. W., & Palmieri, P. A. (2009). Women with posttraumatic stress disorder have larger decreases in heart rate variability during stress tasks. *International Journal of Psychophysiology*, 73, 257–264.
- Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology*, 16, 495–512.
- Lissek, S., & van Meurs, B. (2015). Learning models of PTSD: Theoretical accounts and psychobiological evidence. *International Journal of Psychophysiology*, 98(3, Part 2), 594–605. doi:10.1016/j.ijpsycho.2014.11.006
- Norrholm, S. D., & Jovanovic, T. (2018). Fear processing, psychophysiology, and PTSD. *Harvard Review of Psychiatry*, 26, 129–141. doi:10.1097/HRP.0000000000000189
- Norrholm, S. D., Jovanovic, T., Gerardi, M., Breazeale, K. G., Price, M., Davis, M., ... Tuerk, P. W. (2016). Baseline psychophysiological and cortisol reactivity as a predictor of PTSD treatment outcome in virtual reality exposure therapy. *Behaviour Research and Therapy*, 82, 28–37.
- Ottaviani, C. (2018). Brain-heart interactions in perseverative cognition. *Psychophysiology*, 55, e13082. doi:10.1111/psyp.13082

- Pitman, R. K., Orr, S. P., Forgue, D. F., de Jong, J. B., & Claiborn, J. M. (1987). Psychophysiological assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry*, 44, 970–975. doi:10.1001/archpsyc.1987.01800230050009
- Pole, N. (2007). The psychophysiology of posttraumatic stress disorder: A meta-analysis. *Psychological Bulletin*, 133(5), 725–746. doi:10.1037/0033-2909.133.5.725
- Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation*. New York, NY: W.W. Norton & Company, Inc.
- Robinson-Andrew, E. J., Duval, E. R., Nelson, C. B., Echiverri-Cohen, A., Giardino, N., Defever, A., ... Rauch, S. A. M. (2014). Changes in trauma-potentiated startle with treatment of posttraumatic stress disorder in combat veterans. *Journal of Anxiety Disorders*, 28, 358–362.
- Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences*, 1071, 67–79. doi:10.1196/annals.1364.007
- Tan, G., Dao, T. K., Farmer, L., Sutherland, R. J., & Gevirtz, R. (2011). Heart rate variability (HRV) and posttraumatic stress disorder (PTSD): A pilot study. *Applied Psychophysiology and Biofeedback*, 36, 27–35.
- Task Force of the European Society of Cardiology and the North American Society for Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation*, 17, 151–178. doi:10.1111/j.1542-474X.1996.tb00275.x
- Yehuda, R., & McFarlane, A. C. (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis. *American Journal of Psychiatry*, 152, 1705–1713.
- Zucker, T. L., Samuelson, K. W., Muench, F., Greenberg, M. A., & Gevirtz, R. N. (2009). The effects of respiratory sinus arrhythmia biofeedback on heart rate variability and posttraumatic stress disorder symptoms: A pilot study. *Applied Psychophysiology and Biofeedback*, 34, 134–156.

Suggested Reading

- Ney, L. J., Wade, M., Reynolds, A., Zuj, D. V., Dymond, S., Matthews, A., & Felmingham, K. L. (2018). Critical evaluation of current data analysis strategies for psychophysiological measures of fear conditioning and extinction in humans. *International Journal of Psychophysiology*, 134, 95–107.
- Pineles, S. L., & Orr, S. P. (2018). The psychophysiology of PTSD. In C. B. Nemeroff & C. R. Marmar (Eds.), *Post-traumatic stress disorder*. Oxford University Press.
- Pitman, R. K., Rasmusson, A. M., Koenen, K. C., Shin, L. M., Orr, S. P., Gilbertson, M. W., ... Liberzon, I. (2012). Biological studies of post-traumatic stress disorder. *Nature Reviews. Neuroscience*, 13, 769–787.
- Porges, S. W. (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, 42, 123–146.

Cognitive Reserve

Sarah A. Cooley

*Department of Neurology, School of Medicine, Washington University in Saint Louis,
St. Louis, MO, USA*

Introduction

The relationship between neuropathology and clinical presentation is imperfect. An often-cited postmortem study of older individuals reported substantial Alzheimer's pathology in the brain tissue of individuals who did not have a corresponding degree of cognitive impairment prior to death (Katzman et al., 1988). The concept of cognitive reserve (CR) was introduced as an important explanation for this disconnect in pre- and postmortem disease phenotypes. The following chapter reviews the theoretical basis of CR and putative neural mechanisms underlying the construct. Potential pathways to enhance CR throughout the lifespan are discussed.

Brain Reserve Versus Cognitive Reserve

Katzman et al. (1988) reported greater brain weight and neuronal cell counts in cognitively normal individuals with neuropathological evidence of Alzheimer's disease (AD) compared with cognitively impaired individuals with a similar degree of neuropathology. This observation became the basis for the "passive" model of reserve, otherwise known as brain reserve (Satz, 1993). The brain reserve model highlights the importance of premorbid brain structure integrity, which naturally varies across individuals. Those with higher reserve (more robust neuronal integrity) prior to an injury or onset of disease are less likely to express symptoms compared with individuals with lower reserve. The brain reserve model is often referenced as a quantitative model because biological measures of brain integrity are summed to calculate a quantitative index. For example, neuronal/synapse count (Wilson et al., 2013), head circumference (Perneckzy et al., 2010), and brain/intracranial volume (ICV) (Sumowski et al., 2013) have been utilized as markers of brain reserve. Not all studies report significant associations

between these markers and clinical outcomes (Jenkins, Fox, Rossor, Harvey, & Rossor, 2000; Tate et al., 2011), raising concern that additional factors contribute to individual outcomes despite similar levels of neuropathology.

CR was proposed as an alternative model to brain reserve. CR is described as an “active” model that emphasizes brain function (versus brain structure) and dynamic versus static mechanisms (Stern, 2002). Similar to brain reserve, a person with high CR would be expected to perform better on testing or have a better clinical outcome compared with an individual with lower CR at any given level of neuropathology. Importantly, when compared with those with low CR, individuals with higher CR experience a much faster disease progression after the expression of initial symptoms. These results suggest that a critical threshold governs disease manifestation, such that clinical evidence of severe cognitive dysfunction or dementia is delayed until the threshold of pathology has been met (Hall et al., 2009).

CR is quantified using proxy measures including education (Stern et al., 1994), occupational attainment (Stern et al., 1994), premorbid IQ (Armstrong et al., 2012), literacy (Manly, Schupf, Tang, & Stern, 2005), leisure activities (Scarmeas, Levy, Tang, & Stern, 2001), social networks (Bennett, Schneider, Tang, Arnold, & Wilson, 2006), and bilingualism (Gollan, Salmon, Montoya, & Galasko, 2011). Nucci, Mapelli, and Mondini (2012) demonstrated additional gain using an aggregate measure of multiple CR proxies, referred to as the Cognitive Reserve Index.

Evidence for CR

Stern et al. (1994) conducted one of the first studies of CR and risk of AD in a large group of older adults. Results from this study indicated that individuals with low CR (<8 years of education) had a 2.2 times higher risk for developing dementia over a 4-year period compared with those with high CR (≥8 years of education). Additionally, individuals with lower occupational attainment (e.g., skilled trade, office worker) were more than twice as likely to develop AD over the same four-year period compared with those with higher occupational attainment (e.g., manager, technical professional). A follow-up study reported that individuals who participated in more frequent leisure activities (high CR) had a 38% lower risk of developing dementia compared with those engaged in fewer leisure activities (Scarmeas et al., 2001). Perhaps most importantly, late life modifications to CR lower the risk of subsequent dementia, suggesting neuronal plasticity across the older adult age range (Valenzuela & Sachdev, 2006).

Higher CR is associated with better cognitive health in normal aging (Baker et al., 2017), AD (Scarmeas et al., 2003) and numerous neurological conditions including cerebrovascular disease (Brickman et al., 2009; Lane, Paul, Moser, Fletcher, & Cohen, 2011), human immunodeficiency virus (HIV) (Foley et al., 2012), and Parkinson’s disease (Hindle, Martyr, & Clare, 2014). Other studies report the beneficial effects of higher CR on cognitive outcomes in traumatic brain injury (Kesler, Adams, Blasey, & Bigler, 2003), multiple sclerosis (Nunnari et al., 2016), Lewy body disease (Perneckzy et al., 2008), hepatitis C (Bieliauskas et al., 2007), and frontotemporal lobar degeneration (Placek et al., 2016).

Neural Mechanisms of CR

The literature has utilized two broad terms (neural reserve and neural compensation) to describe the underlying neural mechanisms of CR (Stern, 2017). *Neural reserve* refers to the interindividual variability in brain regions or networks that contribute to cognitive performance

and the resistance of these networks to neuropathology. *Neural compensation* refers to the recruitment of additional/alternative brain regions or networks to perform a cognitive task among individuals with brain degeneration.

Neural reserve encompasses the efficiency of a brain region or network needed to successfully perform a given task. Capacity refers to the maximum degree to which a brain network can be activated to perform a cognitive task, particularly as tasks become increasingly more challenging. Accordingly, a person with higher capacity is more likely to perform a difficult, resource-demanding cognitive task than someone with lower capacity. Neuroimaging studies of neural reserve, CR, and cognitive performance suggest that these concepts apply most readily to young adults with intact brain networks and limited neuropathology (for reviews see Anthony & Lin, 2018; Steffener & Stern, 2012; Stern, 2017).

Neural compensation is typically studied within the context of age-related changes in brain networks as well as pathological (e.g., AD) changes in the brain. Under the neural compensation model, once a primary network reaches capacity or loses function, an alternate (or “compensatory”) network is activated to facilitate task completion. Accuracy, however, is not guaranteed. For example, the older adults who engage compensatory networks to perform a task are more likely to exhibit worse overall performance compared with older adults who did not require engagement of brain regions beyond the primary network (Zarahn, Rakitin, Abela, Flynn, & Stern, 2007). Both neural reserve and neural compensation are associated with CR (Habeck et al., 2003).

In terms of anatomical substrates, Stern et al. (2008) identified the superior frontal gyrus as key to CR-related benefits. Neural correlates of CR also include the medial temporal lobe regions (e.g., medial temporal gyrus, parahippocampal gyrus), which correspond with higher CR in a manner suggesting neural reserve in younger individuals, while the neural correlates of neural compensation in aging (independent of AD) include frontal regions (e.g., superior frontal gyrus, inferior frontal gyrus) (Anthony & Lin, 2018). Additionally, resting-state functional MRI reveals that activity in the default mode network and dorsal attention network correlates with CR in older adults (Arenaza-Urquijo et al., 2013; Bastin et al., 2012).

Cognitive Reserve Interventions

Life experiences have a greater impact on CR compared with brain reserve. For example, Richards and Sacker (2003) reported strong associations between CR and adult occupational attainment. Other studies suggest that participation in mentally stimulating social or leisure activities (e.g., knitting, gardening) relates to lower risks of dementia in older adults (Scarmeas et al., 2001). One study estimated a decline by 0.18 years for every day of self-reported participation in cognitively stimulating leisure activities in community-dwelling older adults (Hall et al., 2009). These results suggest that mental stimulation increases CR and provides protection against cognitive decline, even when implemented in late adulthood. Preliminary evidence indicates these effects may even extend to individuals with mild to moderate AD (Mondini et al., 2016).

Conclusions

CR is a protective mechanism against brain injury. Individuals with higher CR are more likely to exhibit better cognitive performance and lower risk of expressing clinically relevant symptoms of brain dysfunction. Additionally, the neuroimaging literature supports a neural

basis for CR with the identification of specific regions and networks that associate with proxies of CR, such as education and occupational attainment. Future research is needed to examine the long-term outcomes of interventions designed to bolster CR throughout the lifespan.

Author Biographies

Sarah A. Cooley, PhD, is a research data analyst in the Department of Neurology at Washington University in St. Louis School of Medicine. Her research interests include the neuropsychology of aging in HIV, specifically examining changes in cognitive functioning in older HIV-positive individuals compared with normal aging, as well as the neuroimaging correlates of these cognitive changes.

References

- Anthony, M., & Lin, F. (2018). A systematic review for functional neuroimaging studies of cognitive reserve across the cognitive aging spectrum. *Archives of Clinical Neuropsychology*, 33(8), 937–948. <https://doi.org/10.1093/arclin/acx125>
- Arenaza-Urquijo, E. M., Landeau, B., La Joie, R., Mevel, K., Mézenge, F., Perrotin, A., ... Chételat, G. (2013). Relationships between years of education and gray matter volume, metabolism, and functional connectivity in healthy elders. *NeuroImage*, 83, 450–457.
- Armstrong, M. J., Naglie, G., Duff-Canning, S., Meaney, C., Gill, D., Eslinger, P. J., ... Marras, C. (2012). Roles of education and IQ in cognitive reserve in Parkinson's disease—Mild cognitive impairment. *Dementia and Geriatric Cognitive Disorders Extra*, 2, 343–352.
- Baker, L. M., Laidlaw, D. H., Cabeen, R., Akbudak, E., Contruo, T. E., Correia, S., ... Paul, R. H. (2017). Cognitive reserve moderates the relationship between neuropsychological performance and white matter fiber bundle length in healthy older adults. *Brain Imaging and Behavior*, 11(3), 632–639.
- Bastin, C., Yakushev, I., Bahri, M. A., Fellgiebel, A., Eustache, F., Landeau, B., ... Salmon, E. (2012). Cognitive reserve impacts on inter-individual variability in resting-state cerebral metabolism in normal aging. *NeuroImage*, 63(2), 713–722.
- Bennett, D. A., Schneider, J. A., Tang, Y., Arnold, S. E., & Wilson, R. S. (2006). The effect of social networks on the relation between Alzheimer's disease pathology and level of cognitive function in old people: A longitudinal cohort study. *The Lancet Neurology*, 5(5), 406–412.
- Bieliauskas, L. A., Back-Madruga, C., Lindsay, K. L., Wright, E. C., Kronfol, Z., Lok, A. S. F., & Fontana, R. J. (2007). Cognitive reserve and neuropsychological functioning in patients infected with hepatitis C. *Journal of the International Neuropsychological Society*, 13(4), 687–692.
- Brickman, A. M., Siedlecki, K. L., Muraskin, J., Manly, J. J., Luchsinger, J. A., Yeung, L. K., ... Stern, Y. (2009). White matter hyperintensities and cognition: Testing the reserve hypothesis. *Neurobiology of Aging*, 32(9), 1588–1598.
- Foley, J. M., Ettenhofer, M. L., Kim, M. S., Behdin, N., Castellon, S. A., & Hinkin, C. H. (2012). Cognitive reserve as a protective factor in older HIV-positive patients at risk for cognitive decline. *Applied Neuropsychology. Adult*, 19(1), 16–25.
- Gollan, T. H., Salmon, D. P., Montoya, R. I., & Galasko, D. R. (2011). Degree of bilingualism predicts age of diagnosis of Alzheimer's disease in low-education but not in highly educated Hispanics. *Neuropsychologia*, 49(14), 3826–3830.
- Habeck, C., Hilton, H. J., Zarahn, E., Flynn, J., Moeller, J., & Stern, Y. (2003). Relation of cognitive reserve and task performance to expression of regional covariance networks in an event-related fMRI study of nonverbal memory. *NeuroImage*, 20, 1723–1733.

- Hall, C. B., Lipton, R. B., Sliwinski, M., Katz, M. J., Derby, C. A., & Verghese, J. (2009). Cognitive activities delay onset of memory decline in persons who develop dementia. *Neurology*, 73(5), 356–361.
- Hindle, J. V., Martyr, A., & Clare, L. (2014). Cognitive reserve in Parkinson's disease: A systematic review and meta-analysis. *Parkinsonism & Related Disorders*, 20(1), 1–7.
- Jenkins, R., Fox, N. C., Rossor, A. M., Harvey, R. J., & Rossor, M. N. (2000). Intracranial volume and Alzheimer Disease: Evidence against the cerebral reserve hypothesis. *JAMA Neurology*, 57(2), 220–224.
- Katzman, R., Terry, R., DeTeresa, R., Brown, T., Davies, P., Fuld, P., ... Peck, A. (1988). Clinical, pathological, and neurochemical changes in dementia: A subgroup with preserved mental status and neocortical plaques. *Annals of Neurology*, 23(2), 138–144.
- Kesler, S. R., Adams, H. F., Blasey, C. M., & Bigler, E. D. (2003). Premorbid intellectual functioning, education, and brain size in traumatic brain injury: An investigation of the cognitive reserve hypothesis. *Applied Neuropsychology*, 10(3), 153–162.
- Lane, E. M., Paul, R. H., Moser, D. J., Fletcher, T. D., & Cohen, R. A. (2011). Influence of education on subcortical hyperintensities and global cognitive status in vascular dementia. *Journal of the International Neuropsychological Society*, 17(3), 531–536.
- Manly, J. J., Schupf, N., Tang, M. X., & Stern, Y. (2005). Cognitive decline and literacy among ethnically diverse elders. *Journal of Geriatric Psychiatry and Neurology*, 18, 213–217.
- Mondini, S., Madella, I., Zangrossi, A., Bigolin, A., Tomasi, C., Michieletto, M., ... Mapelli, D. (2016). Cognitive reserve in dementia: Implications for cognitive training. *Frontiers in Aging Neuroscience*, 8, 84.
- Nucci, M., Mapelli, D., & Mondini, S. (2012). Cognitive Reserve Index questionnaire (CRIq): A new instrument for measuring cognitive reserve. *Aging Clinical and Experimental Research*, 24(3), 218–226.
- Nunnari, D., DeCola, M. C., Costa, A., Rifici, C., Bramanti, P., & Marino, S. (2016). Exploring cognitive reserve in multiple sclerosis: New findings from a cross-sectional study. *Journal of Clinical and Experimental Neuropsychology*, 38(10), 1158–1167.
- Perneczky, R., Drzezga, A., Boecker, H., Ceballos-Baumann, A. O., Granert, O., Förstl, H., ... Häussermann, P. (2008). Activities of daily living, cerebral glucose metabolism, and cognitive reserve in Lewy Body and Parkinson's disease. *Dementia and Geriatric Cognitive Disorders*, 26, 475–481.
- Perneczky, R., Wagenpfeil, S., Lunetta, K. L., Cupples, L. A., Green, R. C., Decarli, C., ... MIRAGE Study Group. (2010). Head circumference, atrophy, and cognition: Implications for brain reserve in Alzheimer disease. *Neurology*, 75(2), 137–142.
- Placek, K., Massimo, L., Olm, C., Ternes, K., Firn, K., Van Deerlin, V., ... McMillan, C. T. (2016). Cognitive reserve in frontotemporal degeneration. *Neurology*, 87(17), 1813–1819.
- Richards, M., & Sacker, A. (2003). Lifetime antecedents of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology*, 25(5), 614–624.
- Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for the threshold theory. *Neuropsychology*, 7(3), 273–295.
- Scarmeas, N., Levy, G., Tang, M. X., & Stern, Y. (2001). Influence of leisure activity on the incidence of Alzheimer's disease. *Neurology*, 57(12), 2236–2242.
- Scarmeas, N., Zarahn, E., Anderson, K. E., Habeck, C. G., Hilton, J., Flynn, J., ... Stern, Y. (2003). Association of life activities with cerebral blood flow in Alzheimer disease: Implications for the cognitive reserve hypothesis. *JAMA Neurology*, 60(3), 359–365.
- Steffener, J., & Stern, Y. (2012). Exploring the neural basis of cognitive reserve in aging. *Biochimica et Biophysica Acta*, 1822(3), 467–473.
- Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society*, 8(3), 448–460.
- Stern, Y. (2017). An approach to studying the neural correlates of reserve. *Brain Imaging and Behavior*, 11(2), 410–416.

- Stern, Y., Gurlan, B., Tatemichi, T. K., Tang, M. X., Wilder, D., & Mayeux, R. (1994). Influence of education and occupation on the incidence of Alzheimer's disease. *Journal of the American Medical Association*, 271(13), 1004–1010. <https://doi.org/10.1001/jama.271.13.1004>
- Stern, Y., Zarahn, E., Habeck, C., Holtzer, R., Rakitin, B. C., Kumar, A., ... Brown, T. (2008). A common neural network for cognitive reserve in verbal and object working memory in young but not old. *Cerebral Cortex*, 18(4), 959–967.
- Sumowski, J. F., Rocca, M. A., Leavitt, V. M., Riccitelli, G., Comi, G., DeLuca, J., & Filippi, M. (2013). Brain reserve and cognitive reserve in multiple sclerosis: What you've got and how you use it. *Neurology*, 80(24), 2186–2193.
- Tate, D. F., Neeley, E. S., Norton, M. C., Tschanz, J. T., Miller, M. J., Wolfson, L., ... Bigler, E. D. (2011). Intracranial volume and dementia: Some evidence in support of the cerebral reserve hypothesis. *Brain Research*, 1385, 151–162.
- Valenzuela, M., & Sachdev, P. (2006). Brain reserve and dementia: A systematic review. *Psychological Medicine*, 36(4), 441–454.
- Wilson, R. S., Nag, S., Boyle, P. A., Hise, L. P., Yu, L., Buchman, A. S., ... Bennett, D. A. (2013). Neural reserve, neuronal density in the locus ceruleus, and cognitive decline. *Neurology*, 80(13), 1202–1208.
- Zarahn, E., Rakitin, B., Abela, D., Flynn, J., & Stern, Y. (2007). Age-related changes in brain activation during a delayed item recognition task. *Neurobiology of Aging*, 28(5), 784–798.

APOE as a Risk Factor for Age-Related Cognitive Impairment: Neuropsychological and Neuroimaging Findings

Laura E. Korthauer and Ira Driscoll

Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA

Alzheimer's disease (AD) is the sixth leading cause of death in the United States, with an estimated 5.3 million living with the disease (Alzheimer's Association, 2015). The $\epsilon 4$ allele of the apolipoprotein E (*APOE*) gene (located on chromosome 19q13.2) is the best-established genetic risk factor for AD. Two single nucleotide polymorphisms (SNPs), rs7412 and rs429358, define the three major alleles: $\epsilon 2$, $\epsilon 3$, and $\epsilon 4$. The $\epsilon 3$ allele is most common, found in 79% of the population. Approximately 14% of individuals carry at least one copy of the $\epsilon 4$ allele, while 7% are $\epsilon 2$ carriers (Bertram, McQueen, Mullin, Blacker, & Tanzi, 2007).

In Caucasian samples, presence of the $\epsilon 4$ allele increases AD risk in a dose-dependent manner. For example, $\epsilon 3/\epsilon 4$ carriers have 3.68 times greater risk than $\epsilon 3$ homozygotes, while $\epsilon 4/\epsilon 4$ carriers have a sevenfold greater risk of developing AD (Bertram et al., 2007). The $\epsilon 2$ allele is considered protective; it is associated with longevity and 38% lower risk of AD (Bertram et al., 2007). Although the same relative pattern is observed in African American and Hispanic samples (risk among $\epsilon 4$ carriers $> \epsilon 3 > \epsilon 2$), the magnitude of the association between the $\epsilon 4$ allele and AD risk is weaker in these groups compared with Caucasians, suggesting that other genetic or environmental factors may be contributory (Farrer et al., 1997).

APOE encodes a lipid transporter that is expressed throughout the brain and the liver. The ApoE protein binds to low-density lipoprotein (LDL) receptors. The E4 isoform of ApoE has lower stability than E3 and E2, making it more likely to form destabilized protein conformations (Hatters, Peters-Libeu, & Weisgraber, 2006). This destabilized conformation contributes to altered lipid binding, protein misfolding, and protein aggregation, suggesting potential mechanisms for neurodegenerative processes (Hatters et al., 2006).

AD is associated with profound disturbance in cognition, with a clinical diagnosis of probable AD requiring deficits in two or more cognitive domains. Cognition progressively declines over the course of the disease, resulting in significant functional impairments. Elucidating how *APOE* modulates cognition and brain integrity can inform our understanding of the pathological progression of AD. Furthermore, identifying neuropsychological and neuroimaging phenotypes in relation to *APOE* status may help identify novel targets for prevention and treatment efforts. It is particularly important to study the effects of *APOE* across the lifespan, as the magnitude and direction of its effects on cognition and brain integrity differ from young adulthood through older age. Thus, we aim to provide a comprehensive overview of the relationship between *APOE*, cognition, and neuroimaging findings across the human lifespan.

APOE Genotype and Cognition

Given the profound impact of AD on episodic memory, studies of *APOE* and cognitive abilities have naturally focused on this aspect of cognitive performance. However, accumulating evidence suggests that *APOE* may also impact executive functions, processing speed, and general intellectual abilities (Wisdom, Callahan, & Hawkins, 2011).

Young Adulthood

Although *APOE* $\epsilon 4$ is associated with higher risk of AD, it may actually be associated with better cognitive performance earlier in the lifespan. Fetuses carrying the $\epsilon 4$ allele are less likely to be stillborn (Becher, Keeling, McIntosh, Wyatt, & Bell, 2006), while *APOE* $\epsilon 2$ has been associated with higher risk of perinatal death. Infant *APOE* $\epsilon 4$ carriers also have higher scores on an index of mental development at 24 months compared with $\epsilon 2$ and $\epsilon 3$ carriers (Wright et al., 2003). These findings suggest that the ApoE E4 isoform may exert a beneficial effect very early in brain development.

During younger adulthood, commonly defined as ages 18–39 years, $\epsilon 4$ carriers outperform noncarriers across a range of cognitive tests. For example, young adult $\epsilon 4$ carriers (aged 22.8 ± 4) have better episodic memory for a word list than $\epsilon 3$ or $\epsilon 2$ carriers (Mondadori et al., 2007). Among younger individuals (aged 24.7 ± 5.4) recovering from traumatic brain injury, *APOE* $\epsilon 4$ carriers score higher on standardized neuropsychological tests of attention, executive functioning, and episodic memory encoding (Han et al., 2007). Young $\epsilon 4$ carriers also have higher IQs (Yu, Lin, Chen, Hong, & Tsai, 2000), better verbal fluency (Alexander et al., 2007), and greater educational achievement (Hubacek et al., 2001) than noncarriers.

Although not all studies of young adults find differences between *APOE* $\epsilon 4$ carriers and noncarriers, collectively, the literature suggests that the $\epsilon 4$ allele may be advantageous during young adulthood. Han and Bondi have proposed that the effects of *APOE* may be an example of antagonistic pleiotropy (Han et al., 2007). Antagonistic pleiotropy is an evolutionary biology term referring to alleles that have differential effects on fitness at different points in the lifespan. Thus, *APOE* may confer a benefit during young adulthood but may be deleterious during older adulthood. It remains unclear when *APOE* $\epsilon 4$ carriers begin to show cognitive impairment relative to noncarriers. Studying middle-aged individuals can shed light on the transition of the effects of *APOE* from young adulthood to older age.

Middle Adulthood

Studies investigating the effects of *APOE* on cognition in middle age have yielded mixed results. This variability may be partially attributed to differing demographic characteristics and definitions of middle age. For instance, a baseline assessment of 452 middle-aged participants in the Wisconsin Registry for Alzheimer's Prevention (WRAP)—all of whom have at least one parent with AD—found no association between *APOE* and neuropsychological performance (Sager, Hermann, & La Rue, 2005). Similarly, the PATH Through Life project cohort of middle-aged adults ($N = 6,560$) reported no effect of *APOE* on cognition despite age-related declines in episodic memory, working memory, and processing speed in the same cohort (Jorm et al., 2007).

In contrast to these null findings, some evidence suggests that middle-aged $\epsilon 4$ carriers may show subtle differences in cognitive performance. No clear pattern of affected cognitive domains has emerged. Middle-aged $\epsilon 4$ carriers score more poorly on learning and memory tasks, verbal delayed recall, sustained attention, and working memory (Caselli et al., 2001; Flory, Manuck, Ferrell, Ryan, & Muldoon, 2000). Whether there is a dose-dependent relationship of the $\epsilon 4$ and cognition during middle age is an area of ongoing research.

The age at which *APOE* $\epsilon 4$ may transition from benefitting cognition to being detrimental remains an area of active research. Large-scale, longitudinal studies are needed to assess the trajectories of cognitive change in *APOE* $\epsilon 4$ carriers and noncarriers. In one such study, $\epsilon 4$ homozygotes began declining on verbal memory measures in their 50s, while noncarriers maintained their memory abilities until their 70s (Caselli et al., 2011). Another longitudinal study found that for *APOE* $\epsilon 4$ carriers younger than 57 years, verbal memory actually increased over the 4-year follow-up interval. In contrast, $\epsilon 4$ carriers older than 57 experienced a significant decline in immediate recall abilities (Jochemsen, Muller, van der Graaf, & Geerlings, 2012). Together, these studies suggest that *APOE* $\epsilon 4$ may begin to have a detectable adverse effect on cognition in the 50s. The changes in underlying neurobiological processes may begin even earlier, making a research into early biomarkers for AD a fertile area for future research.

Older Adulthood

The deleterious effects of *APOE* $\epsilon 4$ on cognition in older adulthood are robust. The *APOE* $\epsilon 4$ allele negatively affects several cognitive domains in a dose-dependent manner. The most recent meta-analysis to answer this question included 77 studies of 40,972 cognitively healthy adults (Wisdom et al., 2011). It assessed the effects of *APOE* on global cognitive ability and seven separate cognitive domains: attention, episodic memory, executive functioning, perceptual speed, primary memory, verbal ability, and visuospatial skill. *APOE* $\epsilon 4$ carriers performed significantly worse on measures of global cognitive ability, episodic memory, executive functioning, and perceptual speed compared with non- $\epsilon 4$ carriers but did not differ on attention or visuospatial functioning. The largest effect sizes were observed for episodic memory. Furthermore, these effects were moderated by age, with older $\epsilon 4$ carriers having larger deficits in episodic memory and global cognitive ability compared with noncarriers. These findings indicate that the adverse relationship between *APOE* $\epsilon 4$ and cognition may be magnified during the aging process.

Mild Cognitive Impairment and Alzheimer's Disease

Presence of the *APOE* $\epsilon 4$ allele is known to increase risk of developing AD, but it may also affect progression of the disease. Mild cognitive impairment (MCI) is a clinical diagnosis given

to individuals who have significant cognitive impairment in one or more domains but are able to maintain independence in their activities of daily living. People with MCI may have impairments in memory (amnesic MCI) or another cognitive domain (non-amnesic MCI). The factors that increase the risk of conversion from MCI to dementia are as yet uncertain, although they may include cardiovascular risk factors, depressed mood, type 2 diabetes, and traumatic brain injury (Cooper, Sommerlad, Lyketsos, & Livingston, 2015).

A 2011 meta-analysis of prospective cohort studies of individuals with MCI found that carriers of a single *APOE* $\epsilon 4$ allele were 2.29 times more likely to convert, while $\epsilon 4$ homozygotes were 3.94 times to convert to AD-type dementia compared with noncarriers (Elias-Sonnenschein, Viechtbauer, Ramakers, Verhey, & Visser, 2011). Compared with noncarriers, $\epsilon 4$ carriers progressed more quickly and at an earlier age than noncarriers.

Summary of Neuropsychological Findings

Taken together, the available evidence supports the idea that the *APOE* $\epsilon 4$ allele is associated with better cognition in young adulthood but poorer cognition later in life. Not only does *APOE* $\epsilon 4$ increase a risk for dementia, but also it is associated with faster progression of age-related cognitive impairment. Longitudinal studies indicate that cognition may begin to decline during middle age in $\epsilon 4$ carriers, maintaining a steeper trajectory of decline into older adulthood. Thus, efforts to prevent or delay the progression of AD pathology likely need to target middle-aged individuals, before gross decline in cognitive ability manifests. Additionally, understanding the interaction of *APOE* with protective factors such as cognitive reserve (e.g., cognitive resilience in the face of neuropathological damage) will be an important avenue for future research.

Neuroimaging: Relationships Between APOE and Brain Integrity

There has been a surge of imaging genetics studies over the past two decades, including studies employing structural magnetic resonance imaging (MRI), functional MRI (fMRI), diffusion tensor imaging (DTI), MRI arterial spin labeling (ASL), and positron emission tomography (PET). These neuroimaging modalities shed light on gross brain morphology, activity of the brain during memory tasks or while at rest, WM microstructural properties, patterns of regional blood flow, and presence of AD neuropathology, respectively. Neuroimaging represents a powerful noninvasive method of detecting subtle differences associated with *APOE* genotype that may precede detectable cognitive impairment.

Magnetic Resonance Imaging

Regional brain volumes

Development of AD is associated with significant regional brain atrophy, with the most pronounced volume loss in the medial temporal lobes (MTL). The majority of studies investigating how *APOE* genotype impacts brain volume have thus focused on the hippocampus and other MTL structures. Homozygous $\epsilon 4$ AD patients have smaller volumes of the right hippocampus, amygdala, and entorhinal cortex compared with patients who are $\epsilon 3/\epsilon 4$ and $\epsilon 3/\epsilon 3$ (Lehtovirta et al., 1995) and may show greater atrophy over time.

In healthy older adults, longitudinal studies have consistently reported that the rate of hippocampal volume loss is greater in $\epsilon 4$ carriers than noncarriers. In at least two of these

studies (Cohen, Small, Lalonde, Friz, & Sunderland, 2001; Crivello et al., 2010), however, the magnitude of the decline in hippocampal volume was not related to any cognitive changes over the same interval. In healthy, middle-aged individuals, findings are equivocal. One sample of 60–64-year-olds showed no volumetric differences associated with *APOE* genotype (Cherbuin et al., 2008). In contrast, another report demonstrated smaller hippocampal volumes in $\epsilon 4$ carriers younger than 65, with no differences in individuals older than 65 (Lind et al., 2006).

Collectively, literature suggests that *APOE* seems to moderate hippocampal volume loss during aging. *APOE* $\epsilon 4$ -associated hippocampal atrophy may be related to neuronal cell death, loss of neuropil, or degeneration of fibers terminating in the hippocampus. Furthermore, it remains unclear whether hippocampal atrophy drives the higher AD risk among $\epsilon 4$ carriers or is a by-product of some other neurodegenerative process.

Task-based functional MRI (fMRI)

fMRI measures blood oxygenation level-dependent (BOLD) response in brain tissue. Higher BOLD response reflects increased neural activity in a given region. Comparing BOLD response while performing a cognitive task and during a control condition allows inference about *APOE*-related differences in regional brain activation during the specific tasks.

fMRI studies using episodic memory paradigms are equivocal. Some report higher (Bondi, Houston, Eyler, & Brown, 2005), while others report lower (Fleisher, Podraza, et al., 2009) BOLD signal in $\epsilon 4$ carriers. Differences in the memory tasks, participant age, and family history of AD, among other factors, may be contributing to the inconsistencies among the reports. Imaging studies focusing on healthy, young adult $\epsilon 4$ carriers typically report higher MTL activation during memory tasks (Filippini et al., 2009). Additionally, a lifespan study found overactivation in young $\epsilon 4$ carriers compared with noncarriers, while healthy older adults showed a pattern of task-based deactivation (Filippini et al., 2011). This evidence seems to suggest that younger *APOE* $\epsilon 4$ carriers depend on compensatory mechanisms to maintain performance, while early pathology in older $\epsilon 4$ carriers leads to lower recruitment of relevant brain areas.

Healthy, adult $\epsilon 4$ carriers also have higher activation compared with noncarriers on tasks requiring working memory (Wishart et al., 2006), verbal fluency (Smith et al., 2002), semantic categorization (Persson et al., 2008), and executive control (Trachtenberg et al., 2012). Thus, differential patterns of activation extend beyond the MTL to include frontal, parietal, and posterior cingulate regions. One interpretation of such findings is that $\epsilon 4$ carriers must recruit a broader network of brain regions to maintain performance. Longitudinal studies are needed to assess *APOE* $\epsilon 4$ -related change in activation patterns over time.

Functional brain networks

In addition to probing task-related differences in BOLD activation, fMRI can be used to characterize patterns of activity when the brain is at rest. During resting-state fMRI, synchronous patterns of coactivity between discrete brain regions reliably emerge. These so-called resting-state networks overlap with functionally relevant regions known to be involved in visual processing, motor functioning, auditory processing, attention, and memory. Resting-state networks are neuronal in origin, and functional connectivity within these networks reflects underlying structural connectivity (Greicius, Supekar, Menon, & Dougherty, 2009).

MCI and AD involve loss of functional brain connectivity in several resting-state networks. The best-documented differences are in the default mode network (DMN), which includes the posterior cingulate, medial prefrontal, and inferior parietal cortices, and the hippocampus. DMN activity is anticorrelated with activity of brain regions recruited during cognitively demanding tasks. Integrity and activity of this network is thought to support episodic memory

and self-referential thought. A limited number of studies have examined resting-state functional connectivity in *APOE* $\epsilon 4$ carriers. Two studies of healthy young and middle-aged adults have reported that $\epsilon 4$ carriers have higher DMN connectivity (Filippini et al., 2009; Fleisher, Sherzai, et al., 2009), but studies in healthy elderly and early AD show lower connectivity in $\epsilon 4$ carriers. This includes functional disconnection between the hippocampus and posterior cingulate cortex, precuneus, dorsal anterior cingulate cortex, and middle temporal cortex (Sheline et al., 2010). Disrupted functional connectivity is more pronounced in older individuals and is observed even in the absence of amyloid plaques (Sheline et al., 2010).

More research is needed to delineate the effects of *APOE* genotype on functional connectivity of the DMN and other resting-state networks. Characterizing longitudinal changes in network connectivity and dynamic interactions between networks will inform our understanding of connectivity loss with AD progression.

White matter integrity

Although early brain imaging studies focused on gray matter pathology in AD patients, the disease is also associated with profound changes in WM (Gunning-Dixon & Raz, 2000). DTI is a method of imaging the diffusion of water in brain tissue that provides information about the direction of WM tracts as well as their microstructural properties. Thus, DTI metrics may be more sensitive indices of WM disruption than WM hyperintensities. Compared with healthy older adults, AD patients show a more posterior profile of WM abnormalities, including alterations in the parahippocampal gyrus, temporal WM, splenium of the corpus callosum, and posterior cingulum (see Chua, Wen, Slavin, & Sachdev, 2008, for review).

WM microstructural alterations in *APOE* $\epsilon 4$ carriers appear to parallel the differences seen in AD patients. $\epsilon 4$ carriers have lower fractional anisotropy (FA) and higher mean diffusivity (MD), measures of microstructural disorganization, in the MTL and other regions (Honea, Vidoni, Harsha, & Burns, 2009). Preliminary evidence suggests that these microstructural differences are connected to cognitive abilities. For example, one study of 92 healthy older adults found that higher FA in the entorhinal cortex was associated with better memory performance in $\epsilon 4$ but not $\epsilon 3$ carriers (Westlye et al., 2012). Integrating DTI with other neuroimaging modalities will provide a more comprehensive description of brain network integrity and is an important area for future research.

Cerebral perfusion

ASL is a noninvasive method of quantifying cerebral blood flow with MRI. Deficits in cerebral perfusion increase with AD progression and serve as an index of cortical dysfunction (Alsop, Dai, Grossman, & Detre, 2010). Findings related to cerebral perfusion in *APOE* $\epsilon 4$ carriers have been mixed. For instance, a small study ($N = 24$) found that $\epsilon 4$ carriers have higher cerebral perfusion at rest but attenuated changes in MTL during memory encoding (Fleisher, Podraza, et al., 2009). Another study found a similar pattern of $\epsilon 4$ -associated resting hyperperfusion in MCI patients, while $\epsilon 4$ carriers with AD had lower perfusion than $\epsilon 3$ carriers (Kim et al., 2013). This is consistent with an interpretation that $\epsilon 4$ carriers have greater metabolic demands at rest to compensate for their functional impairments early in the disease process, while this compensatory mechanism collapses in the face of more significant pathological burden in AD.

Positron Emission Tomography (PET)

AD neuropathology comprises two main processes: extracellular deposition of beta-amyloid ($A\beta$) plaques and intracellular accumulation of hyperphosphorylated tau protein in neurofibrillary tangles. Traditionally, these hallmarks of AD pathology were only detectable upon

postmortem examination. The development of novel PET ligands has aided understanding of the progression of AD neuropathology *in vivo*. For example, Pittsburgh compound B (^{11}C -PiB) binds to A β in the cortex, permitting visualization of deposition of A β plaques. In healthy older adults and patients with MCI or AD, there is a gene-dose relationship between number of *APOE* $\epsilon 4$ alleles and higher ^{11}C -PiB retention in the frontal, temporal, and parietal cortices (Drzezga et al., 2009). Furthermore, longitudinal imaging studies report that older $\epsilon 4$ carriers have a greater rate of PiB retention compared with $\epsilon 4$ noncarriers over a 2–3 year follow-up interval (Villemagne et al., 2011). More recently, development of a novel tau ligand (^{18}F -THK523) has permitted *in vivo* imaging of neurofibrillary tangles. The effects of *APOE* genotype on ^{18}F -THK523 retention have not yet been explored, but this remains an exciting area for future research.

In addition to *in vivo* characterization of hallmarks of AD neuropathology, PET can be used to assess glucose metabolism using a fludeoxyglucose (^{18}F -FDG) tracer. Patients with AD have significantly lower regional cerebral glucose metabolism, and lower ^{18}F -FDG uptake may predict which patients with MCI will convert to AD (Ch  telat et al., 2003). Among AD patients, *APOE* $\epsilon 4$ carriers consistently show lower cerebral glucose metabolism compared with non- $\epsilon 4$ carriers, particularly within temporal, frontal, parietal, and posterior cingulate cortical regions (Lehmann et al., 2014). Differences in cerebral glucose metabolism are observed before the onset of cognitive symptoms, with healthy older $\epsilon 4$ carriers showing hypometabolism in the posterior cingulate, temporal, parietal, and prefrontal cortices (Langbaum et al., 2010).

In summary, *APOE* $\epsilon 4$ carriers show neuropathological signs characteristic of AD even before the onset of clinical symptoms, including higher deposition of A β plaques visualized via PET PiB imaging and cerebral hypometabolism in frontal, temporal, parietal, and posterior cingulate cortices.

Summary of Neuroimaging Findings

Collectively, neuroimaging literature suggests a role for *APOE* $\epsilon 4$ in brain integrity throughout the lifespan. Consistent with evidence that *APOE* $\epsilon 4$ is associated with better cognition early in life before becoming deleterious in older adulthood, younger $\epsilon 4$ carriers have higher task-based activation and resting-state functional connectivity in studied brain networks. Older $\epsilon 4$ carriers show the opposite patterns, including lower functional connectivity, smaller MTL volumes, disorganization of WM microstructure, cerebral hypometabolism, and greater deposition of A β plaques in the regions of interest. Ongoing longitudinal imaging studies will soon be able to answer important questions about the trajectory and magnitude of age-related changes that may serve as neuroimaging biomarkers of risk for AD.

Summary and Conclusions

Characterizing the effects of *APOE*, the best-established genetic risk factor for AD on neuropsychological functioning and brain health remains an area of active research. *APOE* appears to have a differential role on cognition and underlying brain integrity across the lifespan. Younger $\epsilon 4$ carriers, albeit exhibiting better cognitive performance, seem to require greater recruitment of functional brain resources. In older age, *APOE* $\epsilon 4$ becomes deleterious and is associated with cognitive decline as well as poorer brain integrity. It is possible that the $\epsilon 4$ allele was evolutionarily conserved because of its beneficial effects early in the lifespan; with humans today living decades longer than our ancestors, the adverse effects of *APOE* are

now observed during aging. Alternatively, interactions between *APOE* and other AD risk factors (both genetic and environmental) may explain the conflicting patterns of findings across the lifespan. Given that older age is the biggest risk factor for AD, general age effects and inclusion of individuals in the early stages of age-related cognitive decline may confound the studies of older adults. Studying middle age as a possible transitional period will provide a rich source of information about the effects of *APOE* on cognition. By investigating the additive or synergistic effects of *APOE* with other genetic risk factors for AD, we may advance our understanding of the complex interplay between genes, brain integrity, and cognitive health. Furthermore, using multimodal imaging to create integrated descriptions of brain integrity will increase our ability to predict who is at greatest risk for MCI and AD. This is essential to identify those who may benefit most from early prevention and treatment efforts, reducing the global burden of AD.

Author Biographies

Laura E. Korthauer, MA, is a PhD candidate in the Clinical Psychology Program at the University of Wisconsin-Milwaukee, with an emphasis in clinical neuropsychology. Her research focuses on genetic contributions to structural and functional brain networks that may serve as preclinical biomarkers of age-related cognitive decline.

Ira Driscoll, PhD, is an associate professor of psychology and neuroscience at the University of Wisconsin-Milwaukee. Dr. Driscoll's primary research focus revolves around brain changes as early predictors of cognitive deficits and dementia and the role for hormones and genetic background as modulators of age-related cognitive decline.

References

- Alexander, D. M., Williams, L. M., Gatt, J. M., Dobson-Stone, C., Kuan, S. A., Todd, E. G., ... Gordon, E. (2007). The contribution of apolipoprotein E alleles on cognitive performance and dynamic neural activity over six decades. *Biological Psychology*, 75(3), 229–238. doi:10.1016/j.biopsycho.2007.03.001
- Alsop, D. C., Dai, W., Grossman, M., & Detre, J. A. (2010). Arterial spin labeling blood flow MRI: Its role in the early characterization of Alzheimer's disease. *Journal of Alzheimer's Disease*, 20(3), 871–880. doi:10.3233/JAD-2010-091699
- Alzheimer's Association. (2015). 2015 Alzheimer's disease facts and figures. *Alzheimer's & Dementia*, 11(3), 332–384.
- Becher, J. C., Keeling, J. W., McIntosh, N., Wyatt, B., & Bell, J. (2006). The distribution of apolipoprotein E alleles in Scottish perinatal deaths. *Journal of Medical Genetics*, 43(5), 414–418. doi:10.1136/jmg.2005.033936
- Bertram, L., McQueen, M. B., Mullin, K., Blacker, D., & Tanzi, R. E. (2007). Systematic meta-analyses of Alzheimer disease genetic association studies: The AlzGene database. *Nature Genetics*, 39(1), 17–23. doi:10.1038/ng1934
- Bondi, M. W., Houston, W. S., Eyler, L. T., & Brown, G. G. (2005). fMRI evidence of compensatory mechanisms in older adults at genetic risk for Alzheimer disease. *Neurology*, 64(3), 501–508. doi:10.1212/01.WNL.0000150885.00929.7E
- Caselli, R. J., Dueck, A. C., Locke, D. E., Hoffman-Snyder, C. R., Woodruff, B. K., Rapcsak, S. Z., & Reiman, E. M. (2011). Longitudinal modeling of frontal cognition in *APOE* ϵ 4 homozygotes, heterozygotes, and noncarriers. *Neurology*, 76(16), 1383–1388. doi:10.1212/WNL.0b013e3182167147

- Caselli, R. J., Osborne, D., Reiman, E. M., Hentz, J. G., Barbieri, C. J., Saunders, A. M., ... Alexander, G. E. (2001). Preclinical cognitive decline in late middle-aged asymptomatic apolipoprotein E- ϵ 4/4 homozygotes: A replication study. *Journal of the Neurological Sciences*, 189(1-2), 93–98.
- Cherbuin, N., Anstey, K. J., Sachdev, P. S., Maller, J. J., Meslin, C., Mack, H. A., ... Easta, S. (2008). Total and regional gray matter volume is not related to APOE*E4 status in a community sample of middle-aged individuals. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 63(5), 501–504.
- Chételat, G., Desgranges, B., de la Sayette, V., Viader, F., Eustache, F., & Baron, J. C. (2003). Mild cognitive impairment: Can FDG-PET predict who is to rapidly convert to Alzheimer's disease? *Neurology*, 60(8), 1374–1377.
- Chua, T. C., Wen, W., Slavin, M. J., & Sachdev, P. S. (2008). Diffusion tensor imaging in mild cognitive impairment and Alzheimer's disease: A review. *Current Opinion in Neurology*, 21(1), 83–92. doi:10.1097/WCO.0b013e3282f4594b
- Cohen, R. M., Small, C., Lalonde, F., Friz, J., & Sunderland, T. (2001). Effect of apolipoprotein E genotype on hippocampal volume loss in aging healthy women. *Neurology*, 57(12), 2223–2228.
- Cooper, C., Sommerlad, A., Lyketsos, C. G., & Livingston, G. (2015). Modifiable predictors of dementia in mild cognitive impairment: A systematic review and meta-analysis. *American Journal of Psychiatry*, 172(4), 323–324.
- Crivello, F., Lemaître, H., Dufouil, C., Grasset, B., Delcroix, N., Tzourio-Mazoyer, N., ... Mazoyer, B. (2010). Effects of ApoE-epsilon4 allele load and age on the rates of grey matter and hippocampal volumes loss in a longitudinal cohort of 1186 healthy elderly persons. *NeuroImage*, 53(3), 1064–1069. doi:10.1016/j.neuroimage.2009.12.116
- Drzezga, A., Grimmer, T., Henriksen, G., Mühlau, M., Perneczky, R., Miederer, I., ... Kurz, A. (2009). Effect of APOE genotype on amyloid plaque load and gray matter volume in Alzheimer disease. *Neurology*, 72(17), 1487–1494. doi:10.1212/WNL.0b013e3181a2e8d0
- Elias-Sonnenschein, L. S., Viechtbauer, W., Ramakers, I. H., Verhey, F. R., & Visser, P. J. (2011). Predictive value of APOE- ϵ 4 allele for progression from MCI to AD-type dementia: A meta-analysis. *Journal of Neurology, Neurosurgery, and Psychiatry*, 82(10), 1149–1156. doi:10.1136/jnnp.2010.231555
- Farrer, L. A., Cupples, L. A., Haines, J. L., Hyman, B., Kukull, W. A., Mayeux, R., ... van Duijn, C. M. (1997). Effects of age, sex, and ethnicity on the association between apolipoprotein E genotype and Alzheimer disease: A meta-analysis. *Journal of the American Medical Association*, 278(16), 1349–1356.
- Filippini, N., Ebmeier, K. P., MacIntosh, B. J., Trachtenberg, A. J., Frisoni, G. B., Wilcock, G. K., ... Mackay, C. E. (2011). Differential effects of the APOE genotype on brain function across the lifespan. *NeuroImage*, 54(1), 602–610. doi:10.1016/j.neuroimage.2010.08.009
- Filippini, N., MacIntosh, B. J., Hough, M. G., Goodwin, G. M., Frisoni, G. B., Smith, S. M., ... Mackay, C. E. (2009). Distinct patterns of brain activity in young carriers of the APOE-epsilon4 allele. *Proceedings of the National Academy of Sciences of the United States of America*, 106(17), 7209–7214. doi:10.1073/pnas.0811879106
- Fleisher, A. S., Podraza, K. M., Bangen, K. J., Taylor, C., Sherzai, A., Sidhar, K., ... Buxton, R. B. (2009). Cerebral perfusion and oxygenation differences in Alzheimer's disease risk. *Neurobiology of Aging*, 30(11), 1737–1748. doi:10.1016/j.neurobiolaging.2008.01.012
- Fleisher, A. S., Sherzai, A., Taylor, C., Langbaum, J. B., Chen, K., & Buxton, R. B. (2009). Resting-state BOLD networks versus task-associated functional MRI for distinguishing Alzheimer's disease risk groups. *NeuroImage*, 47(4), 1678–1690. doi:10.1016/j.neuroimage.2009.06.021
- Flory, J. D., Manuck, S. B., Ferrell, R. E., Ryan, C. M., & Muldoon, M. F. (2000). Memory performance and the apolipoprotein E polymorphism in a community sample of middle-aged adults. *American Journal of Medical Genetics*, 96(6), 707–711.
- Greicius, M. D., Supekar, K., Menon, V., & Dougherty, R. F. (2009). Resting-state functional connectivity reflects structural connectivity in the default mode network. *Cerebral Cortex*, 19(1), 72–78. doi:10.1093/cercor/bhn059

- Gunning-Dixon, F. M., & Raz, N. (2000). The cognitive correlates of white matter abnormalities in normal aging: A quantitative review. *Neuropsychology*, 14(2), 224–232.
- Han, S. D., Drake, A. I., Cessante, L. M., Jak, A. J., Houston, W. S., Delis, D. C., ... Bondi, M. W. (2007). Apolipoprotein E and traumatic brain injury in a military population: Evidence of a neuropsychological compensatory mechanism? *Journal of Neurology, Neurosurgery & Psychiatry*, 78(10), 1103–1108. doi:10.1136/jnnp.2006.108183
- Hatters, D. M., Peters-Libeu, C. A., & Weisgraber, K. H. (2006). Apolipoprotein E structure: Insights into function. *Trends in Biochemical Sciences*, 31(8), 445–454. doi:10.1016/j.tibs.2006.06.008
- Honea, R. A., Vidoni, E., Harsha, A., & Burns, J. M. (2009). Impact of APOE on the healthy aging brain: A voxel-based MRI and DTI study. *Journal of Alzheimer's Disease*, 18(3), 553–564. doi:10.3233/JAD-2009-1163
- Hubacek, J. A., Pitha, J., Skodová, Z., Adámková, V., Lánská, V., & Poledne, R. (2001). A possible role of apolipoprotein E polymorphism in predisposition to higher education. *Neuropsychobiology*, 43(3), 200–203.
- Jochimsen, H. M., Muller, M., van der Graaf, Y., & Geerlings, M. I. (2012). APOE $\epsilon 4$ differentially influences change in memory performance depending on age. The SMART-MR study. *Neurobiology of Aging*, 33(4), 832. e815–822. doi:10.1016/j.neurobiolaging.2011.07.016
- Jorm, A. F., Mather, K. A., Butterworth, P., Anstey, K. J., Christensen, H., & Easteal, S. (2007). APOE genotype and cognitive functioning in a large age-stratified population sample. *Neuropsychology*, 21(1), 1–8. https://doi.org/10.1037/0894-4105.21.1.1
- Kim, S. M., Kim, M. J., Rhee, H. Y., Ryu, C. W., Kim, E. J., Petersen, E. T., & Jahng, G. H. (2013). Regional cerebral perfusion in patients with Alzheimer's disease and mild cognitive impairment: Effect of APOE epsilon4 allele. *Neuroradiology*, 55(1), 25–34. doi:10.1007/s00234-012-1077-x
- Langbaum, J. B., Chen, K., Caselli, R. J., Lee, W., Reschke, C., Bandy, D., ... Reiman, E. M. (2010). Hypometabolism in Alzheimer-affected brain regions in cognitively healthy Latino individuals carrying the apolipoprotein E epsilon4 allele. *Archives of Neurology*, 67(4), 462–468. doi:10.1001/archneurol.2010.30
- Lehmann, M., Ghosh, P. M., Madison, C., Karydas, A., Coppola, G., O'Neil, J. P., ... Rabinovici, G. D. (2014). Greater medial temporal hypometabolism and lower cortical amyloid burden in ApoE4-positive AD patients. *Journal of Neurology, Neurosurgery & Psychiatry*, 85(3), 266–273. doi:10.1136/jnnp-2013-305858
- Lehtovirta, M., Laakso, M. P., Soininen, H., Helisalmi, S., Mannermaa, A., Helkala, E. L., ... Hartikainen, P. (1995). Volumes of hippocampus, amygdala and frontal lobe in Alzheimer patients with different apolipoprotein E genotypes. *Neuroscience*, 67(1), 65–72.
- Lind, J., Larsson, A., Persson, J., Lars-Göran Nilsson, M. I., LarsBäckman, R. A., Cruts, M., ... Nyberg, L. (2006). Reduced hippocampal volume in non-demented carriers of the apolipoprotein E $\epsilon 4$: Relation to chronological age and recognition memory. *Neuroscience Letters*, 396(1), 23–27. https://doi.org/10.1016/j.neulet.2005.11.070
- Christian R. A. Mondadori, Dominique J. -F. deQuervain, Andreas Buchmann, Henrietta Mustovic, M. Axel Wollmer, Conny F. Schmidt, Peter Boesiger, Christoph Hock, Roger M. Nitsch, Andreas Papassotiropoulos, Katharina Henke (2007). Better memory and neural efficiency in young apolipoprotein E $\epsilon 4$ carriers. *Cerebral Cortex*, 17(8): 1934–1947, https://doi.org/10.1093/cercor/bhl103
- Persson, J., Lind, J., Larsson, A., Ingvar, M., Slegers, K., Van Broeckhoven, C., ... Nyberg, L. (2008). Altered deactivation in individuals with genetic risk for Alzheimer's disease. *Neuropsychologia*, 46(6), 1679–1687. doi:10.1016/j.neuropsychologia.2008.01.026
- Sager, M. A., Hermann, B., & La Rue, A. (2005). Middle-aged children of persons with Alzheimer's disease: APOE genotypes and cognitive function in the Wisconsin Registry for Alzheimer's Prevention. *Journal of Geriatric Psychiatry and Neurology*, 18(4), 245–249. doi:10.1177/0891988705281882
- Sheline, Y. I., Morris, J. C., Snyder, A. Z., Price, J. L., Yan, Z., D'Angelo, G., ... Mintun, M. A. (2010). APOE4 allele disrupts resting state fMRI connectivity in the absence of amyloid plaques or decreased CSF A β 42. *Journal of Neuroscience*, 30(50), 17035–17040. doi:10.1523/JNEUROSCI.3987-10.2010

- Smith, C. D., Andersen, A. H., Kryscio, R. J., Schmitt, F. A., Kindy, M. S., Blonder, L. X., & Avison, M. J. (2002). Women at risk for AD show increased parietal activation during a fluency task. *Neurology*, 58(8), 1197–1202.
- Trachtenberg, A. J., Filippini, N., Cheeseman, J., Duff, E. P., Neville, M. J., Ebmeier, K. P., ... Mackay, C. E. (2012). The effects of APOE on brain activity do not simply reflect the risk of Alzheimer's disease. *Neurobiology of Aging*, 33(3), 618. e611-618.e613. doi:10.1016/j.neurobiolaging.2010.11.011
- Villemagne, V. L., Pike, K. E., Chételat, G., Ellis, K. A., Mulligan, R. S., Bourgeat, P., ... Rowe, C. C. (2011). Longitudinal assessment of Aβ and cognition in aging and Alzheimer disease. *Annals of Neurology*, 69(1), 181–192. doi:10.1002/ana.22248
- Westlye, E. T., Hodneland, E., Haász, J., Espeseth, T., Lundervold, A., & Lundervold, A. J. (2012). Episodic memory of APOE ε4 carriers is correlated with fractional anisotropy, but not cortical thickness, in the medial temporal lobe. *NeuroImage*, 63(1), 507–516. doi:10.1016/j.neuroimage.2012.06.072
- Wisdom, N. M., Callahan, J. L., & Hawkins, K. A. (2011). The effects of apolipoprotein E on non-impaired cognitive functioning: A meta-analysis. *Neurobiology of Aging*, 32(1), 63–74. doi:10.1016/j.neurobiolaging.2009.02.003
- Wishart, H. A., Saykin, A. J., Rabin, L. A., Santulli, R. B., Flashman, L. A., Guerin, S. J., ... McAllister, T. W. (2006). Increased brain activation during working memory in cognitively intact adults with the APOE epsilon4 allele. *American Journal of Psychiatry*, 163(9), 1603–1610. doi:10.1176/ajp.2006.163.9.1603
- Wright, R. O., Hu, H., Silverman, E. K., Tsaih, S. W., Schwartz, J., Bellinger, D., ... Hernandez-Avila, M. (2003). Apolipoprotein E genotype predicts 24-month bayley scales infant development score. *Pediatric Research*, 54(6), 819–825. doi:10.1203/01.PDR.0000090927.53818.DE
- Yu, Y. W., Lin, C. H., Chen, S. P., Hong, C. J., & Tsai, S. J. (2000). Intelligence and event-related potentials for young female human volunteer apolipoprotein E epsilon4 and non-epsilon4 carriers. *Neuroscience Letters*, 294(3), 179–181.

The Serotonin Transporter-Linked Polymorphic Region (5-HTTLPR) Polymorphism, Stress, and Depression

Christina Di Iorio, Kimberly Johnson, Daniel Sheinbein, Samantha Kahn, Patrick England, and Ryan Bogdan

BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA

Introduction

Diathesis–stress theories postulate that genetic vulnerability promotes the development of depression in the context of stress. The intuitive appeal of this theory was supported by twin research by Kendler and colleagues (Kendler et al., 1995) showing that risk for depression was substantially elevated among individuals whose co-twin had depression, but only if they themselves were recently exposed to a severe stressful life event. However, it was not until 2003 that molecular genetic variation was first associated with vulnerability to the depressogenic effects of stress (Caspi et al., 2003). Specifically, Caspi and colleagues reported evidence that the short allele of the serotonin transporter-linked polymorphic region (5-HTTLPR) polymorphism, which had been previously linked to reduced serotonin transporter expression as well as putative intermediate depressive phenotypes (Hariri et al., 2002; Heils et al., 1996; Lesch et al., 1996), confers vulnerability to stress-related depression. This initial report of a gene $\times$ environment ($G \times E$) interaction spurred multiple subsequent replication and extension attempts that have collectively produced conflicting findings with even meta-analyses reaching opposing conclusions (Duncan & Keller, 2011; Karg, Burmeister, Shedden, & Sen, 2011; Munafo, Durrant, Lewis, & Flint, 2009; Risch et al., 2009; Sharpley, Palanisamy, Glyde, Dillingham, & Agnew, 2014; Taylor & Munafo, 2016; Table 1). These opposing results have led to great controversy within the literature, with divergent interpretations (Caspi, Hariri, Holmes, Uher, &

Table 1 Meta-analyses of the 5-HTTLPR×stress interaction and depression.

<i>Meta-analysis</i>	<i>Data coding</i>	<i>Meta-analytic method</i>	<i>Conclusion</i>
Munafo, Durrant, et al. (2009)	Depression: Depression diagnoses and self-reported depression symptoms and severity	Evaluated main effects of 5-HTTLPR and stress as well as their interaction using fixed effect model	No evidence of G×E, OR: 1.16, 95% CI: 0.89–1.49 Results did not differ with random effects models or with different SLE coding
15 studies 11,158 participants	Stress: Presence (1) or absence (0) of stressful life event. Also tested 2 or more stressful life events relative to 0–1. Stress included lifetime trauma exposure, maltreatment during childhood Genotype: Short allele dominance, i.e., S carriers (S/S, or S/L) vs. L homozygotes (L/L)	pooling odds ratios (ORs). Random effect models implemented when significant association observed in the context of between study heterogeneity	
Risch et al. (2009)	Depression: Depression diagnosis (interview-based diagnosis or >85% on a measure of depression symptoms) Stress: Stressful life events (i.e., 0, 1, 2, ≥3). Genotype: Number of S alleles (additive): 0 (L/L), 1 (S/L), and 2 (S/S). Also compared models of S dominance (i.e., S carriers vs. L homozygotes) or recessive (i.e., S homozygotes vs. L Carriers)	Evaluated main effects of 5-HTTLPR and stress as well as their interaction using random effects model pooling ORs. Also evaluated presence of gene- environment correlation (rGE). Analyses conducted with sexes combined (14 studies) and separated (10 studies)	No evidence of GxE, OR: 1.01, 95% CI: 0.94–1.10 Results did not differ when sexes were considered separately. Findings comparable in dominant and recessive models
14 studies 14,250 participants			

Table 1 (Continued)

<i>Meta-analysis</i>	<i>Data coding</i>	<i>Meta-analytic method</i>	<i>Conclusion</i>
Karg et al. (2011) 54 studies 40,749 participants	Depression: Depression diagnosis and self-reported depression symptoms and severity Stress: Childhood maltreatment, specific medical conditions, and SLE Genotype: As coded in original study	<i>P</i> values extracted from effects reported in each article (using a variety of genotype coding, stress measures, and depression measures) Analyses conducted across all forms of stress reported and stratified by stressor type (i.e., childhood maltreatment, specific medical conditions, and SLE), as well as method of stress assessment (i.e., objective, interview, and self-report).	Significant $G \times E$: $p < .00002$. Short allele associated with greater depression in context of stress exposure. Robust to the exclusion of any single study Stratified analyses by stressor type: childhood maltreatment $p = .00007$, specific medical group, $p = .0004$, SLE $p = .03$. Robust to the exclusion of any single study in the childhood maltreatment and medical group analyses but not the SLE analyses Stratified analyses by stress assessment: Objective measure: $p = .000003$, interview: $p = .0002$, self-report questionnaire: $p = .042$. Robust to the exclusion of any single study in the objective measure and interview analyses but not the self-report questionnaire analyses
Mak, Kong, Mak, Sharma, and Ho (2013) 4 studies 642 participants	Depression: Assessed clinically by structured psychiatric diagnostic interview; either post-stress depression or no depression after stroke Stress: Stroke diagnosis confirmed by imaging studies or medical record Genotype: Short allele dominance, i.e., S carriers (S/S or S/L) vs. L homozygotes	Evaluated the relationship between 5-HTTLPR genotype variations and risk of developing post- stroke depression (PSD) Random effects pooled OR	SS genotype associated with greater post-stroke depression, OR: 2.05, 95% CI: 1.41–2.98, $z = 3.79$, $p < .001$.

(Continued)

Table 1 (Continued)

<i>Meta-analysis</i>	<i>Data coding</i>	<i>Meta-analytic method</i>	<i>Conclusion</i>
Sharpley et al. (2014)	Depression: Assessed by either self-report depression scale or clinical interview	<i>P</i> values extracted from each article. Articles were classified according to whether the studies supported the relationship between the <i>S</i> allele, stress, and depression, or whether the studies did not support the relationship.	Significant $G \times E$, $p = .0000009$. Short allele associated with increased depression in the context of stress exposure.
81 studies 55,269 participants	Stress: Stress included illness, childhood adversity, and recent life stress	Stratified by research design, stressor type, and type of stressor assessment	Robust to the exclusion of any single study
	Genotype: Short allele dominance, i.e., <i>S</i> carriers (<i>S/S</i> or <i>S/L</i>) vs. <i>L</i> homozygotes		Stratified stress assessment: Objective method: $p = .00000100$, Interview: $p = .00002486$, Self-report questionnaire: $p = .00152065$. Robust to exclusion of any single study.
			Stratified by type of stressor: Medical condition: $p = .00029$, childhood adversity: $p = .00026011$, stressful life events: $p = .00061359$. Robust to exclusion of any single study
			Stratified by research design: Exposed only: $p = .002$, longitudinal: $p = .0026$, case control: $p = .0061$, Cross-sectional: $p = .003$. Exposed-only association is nonsignificant when one study removed
			21 of 81 studies (nearly 26%) did not support association between <i>S</i> allele, stress, and depression.

Table 1 (Continued)

<i>Meta-analysis</i>	<i>Data coding</i>	<i>Meta-analytic method</i>	<i>Conclusion</i>
Suppli et al. (2015)	Biallelic genotype: Short allele dominance: SS, SL, and LL as comparison	Evaluated the association between 5-HTTLPR and depression in cancer patients	No evidence that 5-HTTLPR is associated with depression among cancer patients (SL genotype: OR: 1.44, 95% CI: 0.78–2.65; SS genotype (OR: 1.05, 95% CI: 0.71–1.57).
5 studies 1,484 participants (33 head and neck cancer patients and 642 breast cancer patients, 806 colorectal cancer patients)	Depression: users and nonusers of antidepressants	Calculated odds ratios using numbers of antidepressant users and nonusers in biallelic genotype groups. ORs unadjusted and inverse variance weighting for pooling. Computed random effects models	

Moffitt, 2010; Duncan & Keller, 2011) highlighting the need for further study and collaborative meta-analyses (Moffitt & Caspi, 2014; Taylor & Munafo, 2016). Here, we briefly introduce the 5-HTTLPR polymorphism before summarizing the current literature on the 5-HTTLPR polymorphism and depression and exploring potential neural mechanisms through which these associations may emerge.

The 5-HTTLPR Polymorphism

The 5-HTTLPR polymorphism resides within the promoter region of the serotonin transporter gene (*SLC6A4*) (Lesch et al., 1996). The serotonin transporter (5-HTT) is a reuptake protein that regulates the duration and magnitude of synaptic serotonin. It is the target of common antidepressants and anxiolytics, such as serotonin-specific reuptake inhibitors (SSRIs), which block the transporter resulting in increased synaptic serotonin and signaling. The 5-HTTLPR polymorphism is a 43 (initially reported as 44)-base pair imperfect repeat sequence in a GC-rich repetitive sequence that is located approximately 1 kb upstream of the 5-HTT gene transcription initiation site (Wendland, Martin, Kruse, Lesch, & Murphy, 2006). There are two commonly occurring alleles within this polymorphism, the short (s; 14 repeat) and long (l; 16 repeat) repeat, resulting in three genotype groups (s/s, s/l, l/l), although uncommon extra-long repeats have been reported in East Asian and some African samples (Goldman, Gleib, Lin, & Weinstein, 2010; Nakamura, Ueno, Sano, & Tanabe, 2000). 5-HTTLPR allelic frequencies differ according to ancestral origin; relative to European samples, the frequency of the short allele is higher in Asian and lower in African populations with evidence that some phenotypic associations may be dependent upon ancestral origin (Murphy, Maile, & Vogt, 2013). Notably, a common orthologue of the 5-HTTLPR in rhesus macaques enables cross-species investigation (Caspi et al., 2010).

The 5-HTTLPR short allele has been associated with relatively less efficient transcription of the 5-HTT, resulting in less 5-HT uptake and elevated levels of synaptic 5-HT relative to the long allele (Heils et al., 1996; Lesch et al., 1996). In close proximity to the 5-HTTLPR polymorphism are two single nucleotide polymorphisms, rs25531 (G/A) and rs25532 (C/T), that moderate the influence of the 5-HTTLPR polymorphism on *SLC6A4* expression (Murphy et al., 2013). When the rs25531 G allele occurs alongside the 5-HTTLPR long allele, there is relatively less *SLC6A4* expression compared with when the rs25531 A allele is present (Hu et al., 2006). As a consequence, the presence of the G allele with the 5-HTTLPR long allele may render the long allele functionally similar to the short allele, leading many studies to recode alleles according to high and low expression with the 5-HTTLPR long and rs25531 G allele combination being grouped with the short allele as low expressing alleles. For rs25532, the T allele is associated with reduced *SLC6A4* expression, with a greater relative reduction when alongside the long relative to short allele (Wendland et al., 2008). While studies are more frequently including rs25531 in 5-HTTLPR investigations, the lack of consideration of rs25531 and rs25532 as well as other functional polymorphisms within *SLC6A4* may, at least in part, contribute to the mixed 5-HTTLPR psychiatric and neuroscience literature (Murphy et al., 2013).

5-HTTLPR Associations with Depression

The 5-HTTLPR polymorphism is among the most frequently studied genetic polymorphisms within psychiatry and neuroscience (Caspi et al., 2010). Notably, the vast majority of this research has been conducted in samples with European ancestral origins, leaving the generalizability of these findings across populations uncertain. Lesch and colleagues were the first to link this polymorphism to a psychiatric phenotype when they observed that the short allele was associated with elevated neuroticism (Lesch et al., 1996). This association has been replicated in some studies, with meta-analyses supporting evidence of a small but significant positive association between the short 5-HTTLPR allele and neuroticism, but not other anxiety-related measures (Munafo et al., 2009; Schinka, Busch, & Robichaux-Keene, 2004; Sen, Burmeister, & Ghosh, 2004). Further, similar associations with anxiety-like behavior have been observed with an orthologous variant in rhesus macaques (Caspi et al., 2010).

In a 2003 study that captured the attention of researchers and the general public alike, Caspi and colleagues reported evidence that the 5-HTTLPR polymorphism moderates the depressogenic effects of stress (Caspi et al., 2003). Specifically, these authors found that the short allele, which is associated with less 5-HTT expression and consequently less 5-HT reuptake and elevated synaptic 5-HT, was associated with greater rates of depression, but only among individuals who were exposed to adversity early in life or who experienced stressful life events as adults. The observation of a molecular G×E interaction predicting depression built upon existing twin studies (Kendler et al., 1995) and generated excitement that molecular G×E interaction studies may provide unique etiologic insight into psychopathology and resulted in a host of subsequent replication and extension studies. When these studies did not universally replicate the original report and subsequent meta-analyses reached opposing conclusions (Table 1), excitement was replaced by sober realizations that the effect was likely smaller than originally anticipated or potentially a false positive association that has been perpetuated by publication and citation biases (de Vries, Roest, Franzen, Munafo, & Bastiaansen, 2016; Duncan & Keller, 2011).

Differences in meta-analytic approaches such as study inclusion and analytic procedures may have resulted in divergent outcomes (Table 1; Taylor & Munafo, 2016). Meta-analyses that only included studies that closely approximated the methodology of the original report (Caspi et al., 2003) found null effects (Munafo, Durrant, et al., 2009; Risch et al., 2009), while later meta-analyses that adopted broader criteria allowing for more studies to be included have reported evidence of a 5-HTTLPR $\times$ stress interaction predicting depression or related phenotypes (Karg et al., 2011; Sharpley et al., 2014 but see also Taylor & Munafo, 2016). Meta-analyses focusing on specific medical populations have produced mixed results (Mak et al., 2013; Suppli et al., 2015).

Inconsistent results across meta-analyses may also result from how variability in study design among included studies is modeled. The two meta-analyses reporting evidence that the 5-HTTLPR $\times$ stress interaction does predict depression conducted further analyses stratified by stress assessment and type of stressor (Karg et al., 2011; Sharpley et al., 2014). These found that all methods of assessment (e.g., objective measure, interview, self-report) produced significant effects but that objective and interview-based assessments of stress produced the most robust results (Karg et al., 2011; Sharpley et al., 2014) and that self-report measures, which are often used in larger studies with less deep phenotyping, were not robust to individual study exclusion within the meta-analysis (Karg et al., 2011). The explanation of this difference could be attributable to more refined phenotyping leading to more accurate assessments of stress exposure that may enhance real associations (Moffitt & Caspi, 2014) but could also be attributable to the fact that such deep phenotyping is often present in studies of smaller samples, which are inherently more prone to produce false positive and negative results.

The type of and timing of stress exposure appears to moderate the magnitude of the 5-HTTLPR $\times$ stress interaction predicting depression. For example, Karg et al. (2011) found that the 5-HTTLPR $\times$ stress interaction was much larger when considering childhood maltreatment and that this effect was robust to the exclusion of any single study, which was not the case for stressful life events predominantly assessed during adulthood. The larger effect of childhood stress in the 5-HTTLPR $\times$ stress interaction may be attributable to the robust effects of early life stress on depression, potential neurodevelopmental vulnerability (Ansorge, Zhou, Lira, Hen, & Gingrich, 2004; Bogdan, Agrawal, Gaffrey, Tillman, & Luby, 2014), temporal plausibility (Moffitt & Caspi, 2014), and the reduced association between subsequent depressive episodes and stressful life event exposure (Kendler & Gardner, 2016). With regard to the last point, the empirically supported kindling theory of depression (Post, 1992) (Kendler & Gardner, 2016) suggests that initial episodes of depression are strongly predicted by the occurrence of stressful life events, while later episodes are predicted by prior episodes of depression but not major stressful life events. Implicit in this hypothesis is that depressive episodes result in biological and/or psychological scars that set the stage for future episodes (i.e., these individuals are kindled). Importantly, however, stress may not be entirely unpaired from subsequent episodes, but the extent of stress needed to evoke depression may be diminished. More specifically, evidence suggests that minor stressors, such as daily hassles, that would not be detected in many assessments of stressful life events may promote a depressed episode in a kindled individual (Monroe & Harkness, 2005). As such, it is plausible that kindling may also moderate the 5-HTTLPR $\times$ stress interaction predicting depression. This is supported by evidence that the 5-HTTLPR $\times$ stress interaction is most robust when considering early life stress (Bogdan et al., 2014; Karg et al., 2011) as well as evidence that low level stressors during adulthood are most predictive of depression in the context of the 5-HTTLPR genotype $\times$ stress interaction (Kendler, Kuhn, Vittum, Prescott, & Riley, 2005). However, the possibility that

kindling may influence the 5-HTTLPR $\times$ stress interaction predicting depression remains speculative as kindling has not been formally tested in the context of a longitudinal design and 5-HTTLPR $\times$ stress interaction.

One aspect of the 5-HTTLPR, stress, and depression literature that is puzzling is the directionality of association in studies reporting positive results. The short allele, which is associated with stress-related depression in some studies, is associated with less *SLC6A4* expression. Because less 5-HTT availability is semi-analogous to the effects of common treatments for depression (e.g., SSRIs), the directionality of results may appear counterintuitive. However, converging work from nonhuman animal and human studies suggests that increases in 5-HT conferred by the short allele may result in later depression due to elevated levels of serotonin experienced during early life and neurodevelopment. In a nonhuman animal model, rodents treated with SSRIs shortly after birth (equivalent to the third trimester of pregnancy in humans) behave normally as pups but display depressive- and anxiety-like behavior later as adults (Ansorge et al., 2004). In a human cohort study using data from the Finnish national registry found that prenatal exposure to SSRIs was associated with a three- to fourfold risk of depression during adolescence relative to offspring born of mothers with psychiatric disorders who discontinued SSRI use during pregnancy and mothers with psychiatric disorders who were not taking medication (Malm et al., 2016). Collectively, these data suggest that unlike the antidepressant effects of increasing synaptic serotonin by blocking the 5-HTT in adults, elevations of serotonin during prenatal development, and potentially into childhood, may increase risk for depression during adolescence and/or adulthood. Taken together, these findings suggest that the 5-HTTLPR short allele, which is associated with reduced 5-HTT efficiency and greater synaptic 5-HT levels, may increase risk for depression through exposure to elevated serotonin throughout development.

5-HTTLPR: Potential Neural Mechanisms Underlying Associations with Stress-Related Depression

Amygdala and Neuroticism

Imaging genetics is a research strategy that examines associations between both genetic and epigenetic variation and variability in brain structure, function, and connectivity, as well as risk for psychopathology (Bogdan et al., 2017). Imaging genetics provide a unique approach to mechanistically relate differences in 5-HTTLPR genotype to neural systems that mediate cognition, emotion, and behavior in health and disease (Hariri & Holmes, 2006). Most commonly, 5-HTTLPR imaging genetic studies have investigated amygdala responsivity to threatening information. Broadly, the amygdala and its connections are necessary for recognizing possible threat in the environment and then generating and regulating physiologic and behavioral reactions. Elevated amygdala response to threat is a hallmark of various forms of psychopathology and in particular stress-related disorder (Bogdan, Pagliaccio, Baranger, & Hariri, 2016). Amygdala function is regulated by serotonin (Holmes & Hariri, 2003), and Hariri and colleagues (Hariri et al., 2002) linked the short allele to elevated amygdala reactivity in one of the first imaging genetic studies. This finding has not been consistently replicated across studies, with meta-analyses concluding that a small effect may be present (Munafo, Brown, & Hariri, 2008; Murphy et al., 2013 but see also Bastiaansen et al., 2014). As such, heightened amygdala reactivity associated with the short allele remains a plausible mechanism, among many (e.g., hypothalamic–pituitary–adrenal axis function; Gotlib, Joormann, Minor, &

Hallmayer, 2008) through which the 5-HTTLPR polymorphism may confer vulnerability to the depressogenic effects of stress. Indeed, recent evidence that elevated amygdala reactivity prospectively predicts elevated depression symptoms following stressful life event exposure provides support for this speculation (Swartz, Knodt, Radtke, & Hariri, 2015). A related interpretation is that given the links between amygdala reactivity and neuroticism (Cunningham, Arbuckle, Jahn, Mowrer, & Abduljalil, 2010), it is plausible that elevated amygdala reactivity among short allele carriers contributes to neuroticism, which itself moderates the impact of stressful life events on the development of depression (Kendler, Kuhn, & Prescott, 2004). Future adequately powered prospective longitudinal studies employing structural equation models alongside convergent nonhuman animal models are needed to evaluate these putative mechanisms (Bogdan et al., 2016).

Neural Development

As alluded to earlier, 5-HTTLPR genotype may confer vulnerability to stress-related depression due to the neurodevelopmental consequences of reduced 5-HTT function. First, as mentioned above, pharmacologic blockade of the 5-HTT during early development in rodents (perinatal) and humans (prenatal) is associated with elevated anxiety and depression in adolescence and adulthood (Ansorge et al., 2004; Malm et al., 2016). Second, human neuroimaging studies suggest that the associations between 5-HTTLPR genotype and amygdala function is not related to current 5-HTT availability as measured with positron emission tomography, but by brain structure; this raises the possibility that differences conferred by 5-HTT genotype arise from indirect neurodevelopmental consequences on brain structure as opposed to its direct acute effects on serotonin transporter function (Kobiella et al., 2011). Lastly, there is the possibility that both 5-HTTLPR and early life stress may both result in downstream consequences associated with elevated 5-HT activity. For instance, both 5-HTTLPR genotype and early life stress are associated with diminished 5-HT_{1A} receptor binding, suggesting a potential shared molecular pathway with additive influences (Spinelli et al., 2010). Further, this reduced 5-HT_{1A} receptor binding is associated with differences in neurodevelopment and may confer vulnerability to depression and anxiety through disrupted prefrontal regulation of the amygdala and other subcortical structures (Pezawas et al., 2005).

Conclusions

The initial excitement generated by the Caspi et al. (2003) report that variation in the common 5-HTTLPR polymorphism moderates the depressogenic effects of stress have been tempered by replication studies and meta-analyses that have reached opposing conclusions. What is presently clear is that whether 5-HTTLPR genotype increases the risk for stress-related depression remains an empirical question that will be best informed by large longitudinal studies with deep phenotyping. In addition to conducting meta-analyses of existing studies that model between study variability, it is important for further research to evaluate possible reasons that may contribute to this mixed literature, such as the timing of stress exposure and whether kindling may reduce the 5-HTTLPR x stress interaction. Lastly, given accumulating evidence of epigenetic variation within *SLC6A4* associated with depression-related phenotypes (Nikolova et al., 2014; Swartz, Hariri, & Williamson, 2016), it will be important to not only consider genetic variation across the serotonergic system and related systems but also epigenetic signatures.

Author Biographies

Christina Di Iorio is a first-year clinical psychology graduate student and a research assistant in Dr. Bogdan's Behavioral Research and Imaging Neurogenetics (BRAIN) Lab at Washington University in St. Louis. She investigates the interaction of serotonin (5-HT) and NMDA with stress and reward processing and how these interactions impact brain structure and function as well as behavior.

Dr. Kimberly Johnson is an associate professor at the Brown School of Social Work at Washington University in St. Louis. Her research focuses on identifying genetic and environmental factors for cancer, with a particular emphasis on pediatric cancer in both the general and high-risk (neurofibromatosis type 1) populations. She also collaborates with investigators at the Washington University McDonnell Genome Institute on discovery of genomic risk factors for cancer in both adults and children.

Daniel Sheinbein is a medical student at the University of Missouri School of Medicine. His research focuses on behavioral correlates of treatments targeting obesity in children and adults.

Samantha Kahn is a research assistant at Dr. Bogdan's Behavioral Research and Imaging Neurogenetics (BRAIN) Lab. She joined the BRAIN Lab in December of 2014 and obtained her Bachelors in Psychological and Brain Sciences from Washington University in St. Louis in 2016.

Patrick England is a medical student at the Washington University School of Medicine and was previously a member of Dr. Ryan Bogdan's Behavioral Research and Imaging Neurogenetics (BRAIN) Lab in 2015 examining relationships between stress and other mental health outcomes.

Dr. Ryan Bogdan is an associate professor in psychological and brain sciences at Washington University in St. Louis. Dr. Bogdan's research is focused on mechanistic factors that drive variability in mood and behavior to gain insight into the etiology of psychopathology. He is particularly interested in how genetic variation and stress influence mood and behavior through neural mechanisms.

References

- Ansorge, M. S., Zhou, M., Lira, A., Hen, R., & Gingrich, J. A. (2004). Early-life blockade of the 5-HT transporter alters emotional behavior in adult mice. *Science*, 306(5697), 879–881. doi:10.1126/science.1101678
- Bastiaansen, J. A., Servaas, M. N., Marsman, J. B. C., Ormel, J., Nolte, I. M., Riese, H., & Aleman, A. (2014). Filling the gap: Relationship between the serotonin-transporter-linked polymorphic region and amygdala activation. *Psychological Science*, 25(11), 2058–2066. doi:10.1177/0956797614548877
- Bogdan, R., Agrawal, A., Gaffrey, M. S., Tillman, R., & Luby, J. L. (2014). Serotonin transporter-linked polymorphic region (5-HTTLPR) genotype and stressful life events interact to predict preschool-onset depression: A replication and developmental extension. *Journal of Child Psychology and Psychiatry*, 55(5), 448–457. doi:10.1111/jcpp.12142
- Bogdan, R., Pagliaccio, D., Baranger, D. A., & Hariri, A. R. (2016). Genetic moderation of stress effects on corticolimbic circuitry. *Neuropsychopharmacology*, 41(1), 275–296. doi:10.1038/npp.2015.216
- Bogdan, R., Salmeron, B. J., Carey, C. E., Agrawal, A., Calhoun, V. D., Garavan, H., ... Goldman, D. (2017). Imaging genetics and genomics in psychiatry: A critical review of progress and potential. *Biological Psychiatry*, 82(3), 165–175. <https://doi.org/10.1016/j.biopsych.2016.12.030>
- Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex

- diseases and traits. *The American Journal of Psychiatry*, 167(5), 509–527. doi:10.1176/appi.ajp.2010.09101452
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., ... Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301(5631), 386–389. doi:10.1126/science.1083968
- Cunningham, W. A., Arbuckle, N. L., Jahn, A., Mowrer, S. M., & Abduljalil, A. M. (2010). Aspects of neuroticism and the amygdala: Chronic tuning from motivational styles. *Neuropsychologia*, 48(12), 3399–3404. doi:10.1016/j.neuropsychologia.2010.06.026
- de Vries, Y. A., Roest, A. M., Franzen, M., Munafo, M. R., & Bastiaansen, J. A. (2016). Citation bias and selective focus on positive findings in the literature on the serotonin transporter gene (5-HTTLPR), life stress and depression. *Psychological Medicine*, 1–9. doi:10.1017/S0033291716000805
- Duncan, L. E., & Keller, M. C. (2011). A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. *The American Journal of Psychiatry*, 168(10), 1041–1049. doi:10.1176/appi.ajp.2011.11020191
- Goldman, N., Gleit, D. A., Lin, Y. H., & Weinstein, M. (2010). The serotonin transporter polymorphism (5-HTTLPR): Allelic variation and links with depressive symptoms. *Depression and Anxiety*, 27(3), 260–269. doi:10.1002/da.20660
- Gotlib, I. H., Joormann, J., Minor, K. L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry*, 63(9), 847–851. doi:10.1016/j.biopsych.2007.10.008
- Hariri, A. R., & Holmes, A. (2006). Genetics of emotional regulation: the role of the serotonin transporter in neural function. *Trends in Cognitive Sciences*, 10(4), 182–191. doi:10.1016/j.tics.2006.02.011
- Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., ... Weinberger, D. R. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science*, 297(5580), 400–403. doi:10.1126/science.1071829
- Heils, A., Teufel, A., Petri, S., Stober, G., Riederer, P., Bengel, D., & Lesch, K. P. (1996). Allelic variation of human serotonin transporter gene expression. *Journal of Neurochemistry*, 66(6), 2621–2624.
- Holmes, A., & Hariri, A. R. (2003). The serotonin transporter gene-linked polymorphism and negative emotionality: Placing single gene effects in the context of genetic background and environment. *Genes, Brain, and Behavior*, 2(6), 332–335.
- Hu, X. Z., Lipsky, R. H., Zhu, G., Akhtar, L. A., Taubman, J., Greenberg, B. D., ... Goldman, D. (2006). Serotonin transporter promoter gain-of-function genotypes are linked to obsessive-compulsive disorder. *American Journal of Human Genetics*, 78(5), 815–826. doi:10.1086/503850
- Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, 68(5), 444–454. doi:10.1001/archgenpsychiatry.2010.189
- Kendler, K. S., & Gardner, C. O. (2016). Depressive vulnerability, stressful life events and episode onset of major depression: A longitudinal model. *Psychological Medicine*, 46(9), 1865–1874. doi:10.1017/S0033291716000349
- Kendler, K. S., Kessler, R. C., Walters, E. E., MacLean, C., Neale, M. C., Heath, A. C., & Eaves, L. J. (1995). Stressful life events, genetic liability, and onset of an episode of major depression in women. *The American Journal of Psychiatry*, 152(6), 833–842.
- Kendler, K. S., Kuhn, J., & Prescott, C. A. (2004). The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. *The American Journal of Psychiatry*, 161(4), 631–636. doi:10.1176/appi.ajp.161.4.631
- Kendler, K. S., Kuhn, J. W., Vittum, J., Prescott, C. A., & Riley, B. (2005). The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression: A replication. *Archives of General Psychiatry*, 62(5), 529–535. doi:10.1001/archpsyc.62.5.529
- Kobiella, A., Reimold, M., Ulshofer, D. E., Ikonomidou, V. N., Vollmert, C., Vollstadt-Klein, S., ... Smolka, M. N. (2011). How the serotonin transporter 5-HTTLPR polymorphism influences amygdala function: The roles of in vivo serotonin transporter expression and amygdala structure. *Translational Psychiatry*, 1, e37. doi:10.1038/tp.2011.29

- Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., Petri, S., ... Murphy, D. L. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science*, 274(5292), 1527–1531. doi:10.1126/science.274.5292.1527
- Mak, K. K., Kong, W. Y., Mak, A., Sharma, V. K., & Ho, R. C. (2013). Polymorphisms of the serotonin transporter gene and post-stroke depression: A meta-analysis. *Journal of Neurology, Neurosurgery, and Psychiatry*, 84(3), 322–328. doi:10.1136/jnnp-2012-303791
- Malm, H., Brown, A. S., Gissler, M., Gyllenberg, D., Hinkka-Yli-Salomaki, S., McKeague, I. W., ... Sourander, A. (2016). Gestational exposure to selective serotonin reuptake inhibitors and offspring psychiatric disorders: A national register-based study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 55(5), 359–366. doi:10.1016/j.jaac.2016.02.013
- Moffitt, T. E., & Caspi, A. (2014). Bias in a protocol for a meta-analysis of 5-HTTLPR, stress, and depression. *BMC Psychiatry*, 14, 179. doi:10.1186/1471-244X-14-179
- Monroe, S. M., & Harkness, K. L. (2005). Life stress, the “kindling” hypothesis, and the recurrence of depression: Considerations from a life stress perspective. *Psychological Review*, 112(2), 417–445. doi:10.1037/0033-295X.112.2.417
- Munafo, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry*, 63(9), 852–857. doi:10.1016/j.biopsych.2007.08.016
- Munafo, M. R., Durrant, C., Lewis, G., & Flint, J. (2009). Gene X environment interactions at the serotonin transporter locus. *Biological Psychiatry*, 65(3), 211–219. doi:10.1016/j.biopsych.2008.06.009
- Murphy, D. L., Maile, M. S., & Vogt, N. M. (2013). 5HTTLPR: White knight or dark blight? *ACS Chemical Neuroscience*, 4(1), 13–15. doi:10.1021/cn3002224
- Nakamura, M., Ueno, S., Sano, A., & Tanabe, H. (2000). The human serotonin transporter gene linked polymorphism (5-HTTLPR) shows ten novel allelic variants. *Molecular Psychiatry*, 5(1), 32–38.
- Nikolova, Y. S., Koenen, K. C., Galea, S., Wang, C. M., Seney, M. L., Sibille, E., ... Hariri, A. R. (2014). Beyond genotype: Serotonin transporter epigenetic modification predicts human brain function. *Nature Neuroscience*, 17(9), 1153–1155. doi:10.1038/nn.3778
- Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., Verchinski, B. A., Munoz, K. E., Kolachana, B. S., ... Weinberger, D. R. (2005). 5-HTTLPR polymorphism impacts human cingulate-amygdala interactions: A genetic susceptibility mechanism for depression. *Nature Neuroscience*, 8(6), 828–834. doi:10.1038/nn1463
- Post, R. M. (1992). Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. *The American Journal of Psychiatry*, 149(8), 999–1010. doi:10.1176/ajp.149.8.999
- Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J., ... Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *JAMA*, 301(23), 2462–2471. doi:10.1001/jama.2009.878
- Schinka, J. A., Busch, R. M., & Robichaux-Keene, N. (2004). A meta-analysis of the association between the serotonin transporter gene polymorphism (5-HTTLPR) and trait anxiety. *Molecular Psychiatry*, 9(2), 197–202. doi:10.1038/sj.mp.4001405
- Sen, S., Burmeister, M., & Ghosh, D. (2004). Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality traits. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 127B(1), 85–89. doi:10.1002/ajmg.b.20158
- Sharpley, C. F., Palanisamy, S. K., Glyde, N. S., Dillingham, P. W., & Agnew, L. L. (2014). An update on the interaction between the serotonin transporter promoter variant (5-HTTLPR), stress and depression, plus an exploration of non-confirming findings. *Behavioural Brain Research*, 273, 89–105. doi:10.1016/j.bbr.2014.07.030
- Spinelli, S., Chefer, S., Carson, R. E., Jagoda, E., Lang, L., Heilig, M., ... Stein, E. A. (2010). Effects of early-life stress on serotonin(1A) receptors in juvenile Rhesus monkeys measured by positron emission tomography. *Biological Psychiatry*, 67(12), 1146–1153. doi:10.1016/j.biopsych.2009.12.030
- Suppli, N. P., Bukh, J. D., Moffitt, T. E., Caspi, A., Johansen, C., Albieri, V., ... Dalton, S. O. (2015). 5-HTTLPR and use of antidepressants after colorectal cancer including a meta-analysis of 5-HTTLPR and depression after cancer. *Translational Psychiatry*, 5, e631. doi:10.1038/tp.2015.121

- Swartz, J. R., Hariri, A. R., & Williamson, D. E. (2016). An epigenetic mechanism links socioeconomic status to changes in depression-related brain function in high-risk adolescents. *Molecular Psychiatry*. doi:10.1038/mp.2016.82
- Swartz, J. R., Knodt, A. R., Radtke, S. R., & Hariri, A. R. (2015). A neural biomarker of psychological vulnerability to future life stress. *Neuron*, 85(3), 505–511. doi:10.1016/j.neuron.2014.12.055
- Taylor, A. E., & Munafo, M. R. (2016). Triangulating meta-analyses: The example of the serotonin transporter gene, stressful life events and major depression. *BMC Psychology*, 4(1), 23. doi:10.1186/s40359-016-0129-0
- Wendland, J. R., Martin, B. J., Kruse, M. R., Lesch, K. P., & Murphy, D. L. (2006). Simultaneous genotyping of four functional loci of human SLC6A4, with a reappraisal of 5-HTTLPR and rs25531. *Molecular Psychiatry*, 11(3), 224–226. doi:10.1038/sj.mp.4001789
- Wendland, J. R., Moya, P. R., Kruse, M. R., Ren-Patterson, R. F., Jensen, C. L., Timpano, K. R., & Murphy, D. L. (2008). A novel, putative gain-of-function haplotype at SLC6A4 associates with obsessive-compulsive disorder. *Human Molecular Genetics*, 17(5), 717–723. doi:10.1093/hmg/ddm343

The Neuroscience of Well-Being: Part 1 (Conceptual Definitions)

Justine Megan Gatt

*Neuroscience Research Australia, Sydney, NSW, Australia
School of Psychology, University of New South Wales, Sydney, NSW, Australia*

Introduction

There is a growing recognition in mental health research for the need to understand the mechanisms of mental well-being over and above mental illness. The need for this research is driven by evidence suggesting that the absence of mental illness is not necessarily equivalent to optimal mental health or well-being. In fact, mental illness and mental well-being share only 25% in common variance (Keyes, 2005). This variance implies that the factors that contribute to mental illness are not necessarily the same factors that also contribute to mental well-being. Another need for the research is that optimal well-being transverses superior physical functioning and predicts increased longevity and healthy aging (Diener & Chan, 2011), resistance to infection (Steptoe, Dockray, & Wardle, 2009), and reduced risk for illness and mortality (Chida & Steptoe, 2008). Therefore, developing an understanding of the factors that mediate and promote mental well-being is vital for the future development of health promotion interventions. In particular, it would be important to understand the neurobiological mechanisms that mediate well-being and determine whether or not an individual is more or less likely to flourish. The purpose of this chapter is to provide a review of the conceptual definitions of well-being and the ways that it is currently being measured. A potential neural framework to help understand well-being is then presented, drawing from previous and current research. Suggestions for future research and clinical developments in this field are then discussed.

Conceptual Definitions of Well-Being and Their Measurement

Mental well-being is conceptualized and defined by one of three paradigms: (a) the hedonic perspective (subjective well-being), (b) the eudaimonic perspective (psychological well-being), and (c) the composite perspective (combination of both subjective and psychological well-being).

Hedonic Perspective, Subjective Well-Being (SWB)

The construct of hedonia or “subjective well-being” (SWB) is defined by measures of happiness and life satisfaction. Bradburn (Bradburn & Caplovitz, 1965) was the first to differentiate positive and negative affect in the context of social change, a factor structure later confirmed by Diener and colleagues (Diener, Smith, & Fujita, 1995). Andrews and Withey (Andrews & Withey, 1976) enhanced this model and added life satisfaction as a separate dimension to the two major types of affect. These three factors were shown to correlate substantially yet were clearly separable using multitrait-multimethod techniques (Lucas, Diener, & Suh, 1996). Wilson (Wilson, 1967) was the first to review the SWB literature of happiness, followed by a 30-year review by Diener (Diener, Suh, Lucas, & Smith, 1999). Diener summarized this work and defined SWB as three correlated yet separable components: *pleasant affect* such as joy, contentment, pride, affection, happiness, and elation; *unpleasant affect* including sadness, anxiety, stress, depression, guilt, and envy; and *life satisfaction* such as satisfaction with current, past, and future life and a desire to change. Other theorists have since expanded on specific elements of this model, specifically in terms of understanding pleasant affect and positivity. For example, Peterson and Seligman (Peterson & Seligman, 2004) attempted to identify and classify positive psychological traits in terms of what they refer to as the “six virtues”: wisdom and knowledge, courage, humanity, justice, temperance, and transcendence. Each virtue taps into 24 specific character strengths, which are thought to be measurable traits that transcend culture and history.

Commonly used self-report questionnaires of SWB include the 5-item Satisfaction with Life Scale (SWLS) (Diener, Emmons, Larsen, & Griffin, 1985) as a measure of overall life satisfaction and the 26-item WHO Quality of Life Scale (WHOQOL) (Murphy, Herrman, Hawthorne, Pinzone, & Evert, 2000) as a measure of quality of life for the domains of physical and psychological health, social relationships, and environment. Other more specific measures of SWB include measures of *happiness* using the 4-item Subjective Happiness Scale (SHS) (Lyubomirsky & Lepper, 1999), *flourishing* using the 8-item Flourishing Scale (Diener et al., 2010), and *positive/negative feelings* using the 12-item Scale of Positive and Negative Experience (SPANE) (Diener et al., 2010).

Eudaimonic Perspective, Psychological Well-Being (PWB)

Eudaimonia or “psychological well-being” (PWB) in contrast defines well-being in terms of the development of the human potential. This perspective was originally derived from Aristotle’s philosophy of happiness and what it means to live a good life as articulated in his *Nicomachean Ethics* (Broadie & Rowe, 2002). Ryff (Ryff, 1989) was a strong proponent for this perspective as she believed the previous literature on well-being was not strongly theoretically guided and that it neglected important aspects of positive psychological functioning. Some missing perspectives included Jung’s (Jung, 1933) formulation of individuation, Roger’s (Rogers, 1961) view of the fully functioning person, Allport’s (Allport, 1961) conception of

maturity, and Maslow's (Maslow, 1968) view of self-actualization. Ryff (Ryff, 1989) also suggested that we need to consider how PWB may change and evolve over the lifespan to deal with ongoing life challenges as purported by Buhler's (Buhler, 1935) work on life fulfillment, Erikson's (Erikson, 1959) psychosocial stage model, and Jahoda's (Jahoda, 1958) definition of positive mental health over and above the absence of illness. To this end, Ryff (Ryff, 1989) proposed six essential dimensions of PWB: *self-acceptance*, the acceptance of self and one's past life; *positive relations with others*, the ability to feel empathy and affection for others and to be able to develop warm and intimate relationships; *autonomy*, the ability to be self-determined and independent and to possess an internal locus of control; *environmental mastery*, the ability to choose or create environments suitable to one's needs, to control complex environments, and to capitalize on environmental opportunities; *purpose in life*, to possess goals, intentions, and a sense of direction that provide a meaning to life; and *personal growth*, the ability to actualize and realize one's potential and to grow and expand as a person. Empirical evidence supports the six-factor structure of these dimensions with a higher-order common factor of PWB (Ryff, 1989).

Scientists have since studied specific elements of eudaimonic well-being to better understand its role in mental health. Goal theorists such as Scheier and Carver (Carver, Scheier, & Segerstrom, 2010) discuss how behavioral self-regulation and well-being can be maintained and emphasize the role of goal orientation, confidence, and optimism. In this sense, working toward particular goals is considered to make an impact on outcomes in future health and one's ability to deal with health scares. Confidence to achieve set goals is thought to lead to a more optimistic assessment of the expected outcome and therefore engages the necessary behaviors to see the goal through to fruition. This concept is akin to Ryff's (Ryff, 1989) dimension of *purpose in life* and how goals are central to achieve a meaningful life. Other theorists focus on a self-deterministic view of well-being whereby eudaimonia is characterized in terms of motivation, like pursuing intrinsic goals such as personal growth, intimacy, being mindful, and acting with a state of awareness (Ryan, Huta, & Deci, 2008). Mindfulness is an awareness of the present moment and facilitates meaningful decision making and enactment of intrinsic values. Intrinsic aspirations and mindfulness are contrasted to extrinsic aspirations such as wealth, fame, and image, which do not increase well-being because they do not satiate the basic psychological needs of autonomy, competence, and relatedness (Ryan et al., 2008).

The Ryff Scales of Psychological Well-Being (Ryff, 1989) are commonly used to measure specific components of PWB including autonomy, environmental mastery, personal growth, positive relations with others, purpose in life, and self-acceptance. Other more specific measures of PWB include measures of *gratitude* using the 6-item Gratitude Questionnaire (GC-6) (McCullough, Emmons, & Tsang, 2002) and *mindfulness* using the 15-item Mindful Attention Awareness Scale (MAAS) (Brown & Ryan, 2003).

Composite Perspective, SWB and PWB Combined

SWB and PWB are conceptually related but empirically distinct streams of positive psychological functioning. A number of studies have demonstrated this distinctiveness with factor analysis across measures of both SWB and PWB (Compton, Smith, Cornish, & Qualls, 1996). Findings from Keyes and Huta suggest that both SWB and PWB are important aspects of well-being and that they may have features that overlap such as mastery, self-acceptance or life satisfaction, and vitality (Huta & Ryan, 2010). Indeed, Keyes went on to develop one of the first composite indices of well-being called the Mental Health Continuum (long and short forms, MHC-LF and MHC-SF, respectively) (Keyes, 2002, 2007). The MHC index measures aspects

of emotional, psychological, and social well-being with strong internal consistency (Keyes, 2005) and test–retest reliability over 4 weeks (Robitschek & Keyes, 2009) and 9 months (Lamers, Glas, Westerhof, & Bohlmeijer, 2012). Ryan further suggested that SWB and PWB are not distinct types of well-being but different endpoints for the same outcome. PWB specifies the conditions of optimal living and the effects of these conditions, which include not only hedonic aspects of pleasure seeking but also other elements of good health such as vitality, intimacy, and a sense of meaning. In contrast, SWB focuses on the experience of pleasure as a state that can be obtained through intrinsic goal-driven eudaimonic living or through extrinsic (albeit, less altruistic) means such as living a life of greed, materialism, or exploitation (Ryan et al., 2008).

More recently, Seligman integrated aspects of PWB and SWB into the PERMA theory of well-being, which includes the constructs of *engagement* (the loss of self-consciousness during an absorbing activity, also referred to as *flow* [Csikszentmihalyi, 1992]), *relationships* (the presence of positive and supportive relationships), *accomplishment* (the intrinsic goal of achievement and goal orientation), *positive emotion* (happiness and life satisfaction), and *meaning* (purpose in life) (Seligman, 2011). Consistent with this theory, Gatt and colleagues have developed a short 26-item composite scale of well-being called COMPAS-W (Gatt, Burton, Schofield, Bryant, & Williams, 2014), which includes some of the constructs proposed by Seligman. The COMPAS-W scale provides both an overall measure of total well-being and specific measures of subjective and PWB, including sub-measures of *composure* (competency and adaptability in stressful situations), *own-worth* (autonomy and independent self-worth), *mastery* (self-confidence and perceived control over one's environment), *positivity* (optimism and positive outlook), *achievement* (goal orientation and striving), and *satisfaction* (satisfaction with life, health, work, personal relationships, and emotions). This scale was tested in 1,669 healthy twins and showed strong internal reliability, test–retest reliability over 12 months, and construct validity with other physical and psychological health indicators. Individuals are scored along a continuous dimension of well-being or classified into three groups: flourishing, moderate, or languishing well-being. Univariate twin modeling of monozygotic and dizygotic twin pairs demonstrated a moderate impact of genetics toward overall well-being (heritability of 48%), plus an important role of unique environment at 52% (Gatt et al., 2014). Evidence from other twin studies has similarly suggested that common genetic factors contribute to overall well-being and its subjective and psychological subcomponents (Keyes, Myers, & Kendler, 2010). Moreover, in a later study, the COMPAS-W well-being scale was shown to share approximately 30% of variance in common with depression and anxiety symptoms in a large sample of healthy adult twins, of which 56% was due to genetic effects (Routledge et al., 2016). This finding is consistent with previous reports in healthy and clinical samples whereby mental health and mental illness have been shown to share about 25% in common variance (Keyes, 2005).

Together, this evidence suggests that both SWB and PWB contribute to overall mental well-being, thereby supporting the use of composite models and composite scales. Twin studies also suggest that both genetics and environment contribute to variance in well-being. The specific role of genetic factors may pertain to a protective effect toward positive well-being attributes, and/or they may modulate sensitivity to environmental influences, heightening responses, or biases toward positive life events and lifestyle factors (e.g., exercise, healthy diet) and against negative life events and factors (e.g., poor diet, alcohol and drug use). The moderate impact of environment also suggests that well-being is malleable to some degree and could be promoted further with enriched positive environments and/or intervention. If we can

understand the neural mechanisms that mediate well-being, we can pinpoint which specific characteristics or patterns of behavior to target in interventions.

In the next chapter (Part 2), the core neural networks that are typically implicated in mental illness are summarized and discussed in relation to mental well-being. Current evidence will be reviewed and suggested future research developments will be discussed.

Acknowledgments

Dr. Justine Gatt is currently supported by an NHMRC CDF Fellowship APP1062495.

Author Biography

Dr. Justine Megan Gatt is group leader and senior research scientist at Neuroscience Research Australia and at the School of Psychology at the University of New South Wales, Australia. Dr. Gatt leads a research program focusing on the neuroscience and gene–environment mechanisms of resilience and mental well-being across adult and youth groups. This work incorporates various genetic, neuroimaging and neurocognitive techniques, and various methodological designs (twin/singleton studies, longitudinal and intervention work), with the ultimate aim being to understand and promote optimal mental health in the general population.

References

- Allport, G. W. (1961). *Pattern and growth in personality*. New York, NY: Holt, Rinehart & Winston.
- Andrews, F. M., & Withey, S. B. (1976). *Social indicators of well-being*. New York, NY: Plenum Press.
- Bradburn, N. M., & Caplovitz, D. (1965). *Reports of happiness*. Chicago, IL: Aldine.
- Broadie, S., & Rowe, C. (2002). 'Preface' in *Aristotle: 2002, Nichomachean ethics*. New York, NY: Oxford.
- Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness and its role in psychological well-being. *Journal of Personality and Social Psychology*, 84, 822–848.
- Buhler, C. (1935). The curve of life as studied in biographies. *Journal of Applied Psychology*, 19, 405–409.
- Carver, C. S., Scheier, M. F., & Segerstrom, S. C. (2010). Optimism. *Clinical Psychology Review*, 30, 879–889. doi:10.1016/j.cpr.2010.01.006
- Chida, Y., & Steptoe, A. (2008). Positive psychological well-being and mortality: A quantitative review of prospective observational studies. *Psychosomatic Medicine*, 70, 741–756.
- Compton, W. C., Smith, M. L., Cornish, K. A., & Qualls, D. L. (1996). Factor structure of mental health measures. *Journal of Personality and Social Psychology*, 76, 406–413.
- Csikszentmihalyi, M. (1992). *Flow: The classic work on how to achieve happiness*. USA: Harper & Row.
- Diener, E., & Chan, M. Y. (2011). Happy people live longer: Subjective well-being contributes to health and longevity. *Applied Psychology: Health and Well-Being*, 3(1), 1–43.
- Diener, E., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment*, 49, 71–75.
- Diener, E., Smith, H., & Fujita, F. (1995). The personality structure of affect. *Journal of Personality and Social Psychology*, 69, 130–141.
- Diener, E., Suh, E. M., Lucas, R. E., & Smith, H. L. (1999). Subjective well-being: Three decades of progress. *Psychological Bulletin*, 125(2), 276–302.

- Diener, E., Wirtz, D., Tov, W., Kim-Prieto, C., Choi, D.-W., Oishi, S., & Biswas-Diener, R. (2010). New well-being measures: Short scales to assess flourishing and positive and negative feelings. *Social Indicators Research*, 97, 143–156.
- Erikson, E. (1959). Identity and the life cycle. *Psychological Issues*, 1, 18–164.
- Gatt, J. M., Burton, K. L. O., Schofield, P. R., Bryant, R. A., & Williams, L. M. (2014). The heritability of mental health and wellbeing defined using COMPAS-W, a new composite measure of wellbeing. *Psychiatry Research*, 219, 204–213.
- Huta, V., & Ryan, R. M. (2010). Pursuing pleasure or virtue: The differential and overlapping well-being benefits of hedonic and eudaimonic motives. *Journal of Happiness Studies*, 11, 735–762.
- Jahoda, M. (1958). *Current concepts of positive mental health*. New York, NY: Basic Books.
- Jung, C. G. (1933). In W. S. Dell & C. F. Baynes (Eds.), *Modern man in search of a soul*. New York, NY: Harcourt, Brace & World.
- Keyes, C. L. M. (2002). The mental health continuum: From languishing to flourishing in life. *Journal of Health and Social Behavior*, 43, 207–222.
- Keyes, C. L. M. (2005). Mental illness and/or mental health? Investigating axioms of the complete state model of health. *Journal of Consulting and Clinical Psychology*, 73(3), 539–548.
- Keyes, C. L. M. (2007). Promoting and protecting mental health as flourishing: A complementary strategy for improving national mental health. *American Psychologist*, 62, 95–108.
- Keyes, C. L. M., Myers, J. M., & Kendler, K. S. (2010). The structure of the genetic and environmental influences on mental well-being. *American Journal of Public Health*, 100, 2379–2384.
- Lamers, S. M. A., Glas, C. A. W., Westerhof, G. J., & Bohlmeijer, E. T. (2012). Longitudinal evaluation of the Mental Health Continuum-Short Form (MHC-SF): Measurement invariance across demographics, physical illness and mental illness. *European Journal of Psychological Assessment*, 28, 290–296. doi:10.1027/1015-5759/a000109
- Lucas, R. E., Diener, E., & Suh, E. (1996). Discriminant validity of well-being measures. *Journal of Personality and Social Psychology*, 71, 616–628.
- Lyubomirsky, S., & Lepper, H. S. (1999). A measure of subjective happiness: Preliminary reliability and construct validation. *Social Indicators Research*, 46, 137–155.
- Maslow, A. H. (1968). *Toward a psychology of being* (2nd ed.). New York, NY: Van Nostrand.
- McCullough, M. E., Emmons, R. A., & Tsang, J. (2002). The grateful disposition: A conceptual and empirical topography. *Journal of Personality and Social Psychology*, 82, 112–127.
- Murphy, B., Herrman, H., Hawthorne, G., Pinzone, T., & Evert, H. (2000). *Australian WHOQoL instruments: User's manual and interpretation guide*. Melbourne, Australia: Australian WHOQoL Field Study Centre.
- Peterson, C., & Seligman, M. E. P. (2004). *Character strengths and virtues: A handbook and classification*. Oxford, UK: Oxford University Press.
- Robitschek, C., & Keyes, C. L. M. (2009). The structure of Keyes' model of mental health and the role of personal growth initiative as a parsimonious predictor. *Journal of Counseling Psychology*, 56, 321–329.
- Rogers, C. R. (1961). *On becoming a person*. Boston, MA: Houghton Mifflin.
- Routledge, K. M., Burton, K. L. O., Williams, L. M., Harris, A., Schofield, P. R., Clark, C. R., & Gatt, J. M. (2016). Shared versus distinct genetic contributions of mental wellbeing with depression and anxiety symptoms in healthy twins. *Psychiatry Research*, 244, 65–70.
- Ryan, R. M., Huta, V., & Deci, E. L. (2008). Living well: A self-deterministic theory perspective on eudaimonia. *Journal of Happiness Studies*, 9, 139–170.
- Ryff, C. D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology*, 57(6), 1069–1081.
- Seligman, M. E. P. (2011). *Flourish: A new understanding of happiness and well-being—And how to achieve them*. London, UK: Free Press.
- Steptoe, A., Dockray, S., & Wardle, J. (2009). Positive affect and psychobiological processes relevant to health. *Journal of Personality*, 77(6), 1747–1776.
- Wilson, W. (1967). Correlates of avowed happiness. *Psychological Bulletin*, 67, 297–306.

Gene × Environment Interactions: BDNF

Lauren E. Salminen¹ and Robert H. Paul²

¹*Imaging Genetics Center, University of Southern California, Marina del Rey, CA, USA*

²*Missouri Institute of Mental Health, St. Louis, MO, USA*

Introduction

Neurotrophins are structurally similar polypeptide growth factors that facilitate the growth and differentiation of the nervous system. Four types of neurotrophins have been identified in humans and animals, all with specialized functions. Nerve growth factor (NGF) was the first neurotrophin to be discovered in the early 1950s for its role in sensory and sympathetic neuron survival. Brain-derived neurotrophic factor (BDNF) was later identified in 1982 for its involvement in the development and maintenance of neurons through axonal and dendritic branching. Neurotrophin-3 (NT-3) and neurotrophin-4/5 (NT-4/5) are the most recent members of the mammalian neurotrophin family to be identified. Although less is known about their independent functions using *in vivo* methods, studies show that NT-3 and NT-4/5 demonstrate unique expression patterns that may be mechanistically important for certain growth pathways (Adachi, Numakawa, Richards, Nakajima, & Kunugi, 2014).

BDNF is the most widely studied neurotrophin in clinical research due to its known impact on human brain structure and behavior. BDNF signaling promotes long-term potentiation (LTP) and plasticity of the hippocampus, facilitates adaptive responses to brain injury, and is an important mediator of protein regulation in various biosynthetic pathways (Adachi et al., 2014; Egan et al., 2003). In this chapter we will review the diverse biological roles of BDNF and its interactions with sociodemographic variables and environmental stimuli to impact human health.

Genetic Structure and Biology of BDNF

Human BDNF is encoded by the *BDNF* gene on chromosome 11. The *BDNF* gene is structurally complex, with eight 5' exons (exons I–VIII, plus the human-specific Vh and VIIIh exons) and functional promoters that are spliced onto one 3' protein-coding exon (exon IX) (Adachi et al., 2014). Alternative splicing at each of the 5' exons can produce different mRNA precursor transcripts (pre-pro BDNF isoforms), but only the 3' region of exon IX contains the common coding sequence to synthesize the BDNF signaling peptide, pro-BDNF (~32 kDa). In humans, exons I, VII, and VIII of the *BDNF* gene contain the necessary ATG sequence to initiate translation, yielding pre-pro BDNF proteins with longer peptide sequences than untranslated exons with start codons in exon IX. Further, *BDNF* gene transcription terminates at two polyadenylation sites in exon IX, resulting in subpopulations of mRNA with short and long 3' untranslated regions (UTR). Although all BDNF isoforms are converted to pro-BDNF with identical primary sequences, differences in signaling peptides and the length of the 3' UTR differentially influence physiological processes and tissue-specific expression, respectively (Adachi et al., 2014). Notably, the promoter elements in exon IV regulate activity-dependent BDNF expression.

Pro-BDNF consists of an N-terminal pro-domain of 129 amino acids and a C-terminal mature domain of 118 amino acids (Hempstead, 2015). Initially synthesized in the endoplasmic reticulum, the Golgi apparatus transports pro-BDNF to the trans-Golgi network (TGN) for packaging and secretion via constitutive or regulated pathways (Adachi et al., 2014). Secretory vesicles in the constitutive pathway transport pro-BDNF to the plasma membrane for continuous extracellular cleavage by plasminogen/plasmin and release of mature BDNF (mBDNF). In the regulated pathway, pro-BDNF vesicles accumulate at the plasma membrane via Ca^{2+} -dependent exocytosis and are eventually released by activity-dependent intracellular triggering signals. Here, pro-BDNF may be converted to mBDNF in the TGN by furin and other endoproteases, or in the immature secretory granules by proprotein convertases (Hempstead, 2015). Unlike other neurotrophins, BDNF is preferentially secreted by the regulated pathway to modulate synaptic activity and neural connectivity.

Mature BDNF mediates neuronal survival and differentiation by binding to the tropomyosin receptor kinase B (TrkB) on pre- and postsynaptic membranes. Conversely, pro-BDNF activates the p75 receptor, a member of the tumor necrosis factor family, which has shown to be proapoptotic when bound to a high-affinity ligand (e.g., sortilin). Indeed, animal studies show that conversion of pro-BDNF to mBDNF promotes late-phase LTP in the hippocampus, but that perfusion of pro-BDNF facilitates long-term depression (LTD). Similar studies show that secretions of pro-BDNF, rather than mBDNF, predominate following experimental conditions that induce LTD. These dichotomous functions of BDNF domains have been described as the “yin and yang of neurotrophic action” (Lu, Pang, & Woo, 2005).

Clues to the bioactive role of pro-BDNF came from investigations of a common single nucleotide polymorphism (SNP) at position 66 of the pro-domain in the *BDNF* gene/rs6265 (Egan et al., 2003). This SNP involves a valine (Val) to methionine (Met) substitution that influences the sorting of BDNF into the constitutive and regulated pathways. Specifically, the Met allele shows diminished capacity for sortilin binding (intracellular trafficking chaperone) and altered targeting of BDNF to secretory granules in the regulated pathway (Adachi et al., 2014). The net result is reduced activity-dependent release of BDNF. Interestingly, there is currently no evidence that the Val66Met marker influences the activity of mBDNF (Forde et al., 2014).

Of the 84 SNPs that have been identified in the *BDNF* gene, most research examining relationships between BDNF expression, brain, and behavior has focused on the Val66Met SNP. The Met variant of the Val66Met SNP is associated with abnormal sorting and secretion of BDNF (Hempstead, 2015), and this mutation is common among European and Asian ancestries. Other mutations within the *BDNF* gene have gained increased attention for their role in antidepressant efficacy and behavior (Colle et al., 2015), though much of this work remains preliminary. Given the large body of work dedicated to *BDNF* SNPs and brain–behavior phenotypes, this chapter will review the most salient gene $\times$ brain $\times$ behavior associations in the literature, with a primary focus on the Val66Met mutation.

BDNF Expression Patterns and Influential Variables of Bioactivity

Animal and human studies show that BDNF is widely expressed across the central nervous system (CNS), with high concentrations in the cerebral cortex, hippocampus, amygdala, hypothalamus, and cerebellum (Adachi et al., 2014). The ratio of pro-BDNF to mBDNF varies across the lifespan, with moderate concentrations of both peptide variants in adolescence, and a predominance of mBDNF in adulthood. However, plasma levels of BDNF decrease with age, as does signaling capacity in the brain. BDNF can also be synthesized and stored in peripheral tissues, yet the brain is the primary source of protein synthesis during development. Accordingly, early life stress has been associated with lower BDNF levels in the hippocampus, anterior cingulate cortex, and amygdala, and these reductions persist into adulthood (reviewed in van Velzen et al., 2016).

Hippocampal BDNF expression has been the focus of many investigations given the high level of BDNF expression in this brain region and its corresponding influence on neurogenesis in the dentate gyrus. Adult neurogenesis occurs through both environmental enrichment (exposure to various learning experiences, social interactions, and physical exercise) and genetic mechanisms, and the direction of these relationships is dependent on BDNF activity. Although the majority of literature regarding environmental enrichment and hippocampal neurogenesis comes from experimental studies in rodents, there is evidence to support a role for BDNF-dependent hippocampal neurogenesis in humans. Much of this evidence can be extrapolated from the neurobiological and behavioral similarities between rodents and humans, but additional support comes from studies showing exercise-induced increases in cerebral blood volume using magnetic resonance imaging (MRI), which has shown to correlate with hippocampal neurogenesis in mice (for review see Clemenson, Deng, & Gage, 2015). Perhaps the most salient evidence, however, comes from the well-known study of London taxi drivers, who exhibit larger hippocampal volume compared with controls and a positive correlation between hippocampal volume and spatial navigation expertise and visuospatial memory (Woollett & Maguire, 2011). Although BDNF concentrations and genotyping were not completed in these studies, the results reinforce a role for environmental enrichment in hippocampal neurogenesis, that is, likely partially BDNF dependent.

Genetic influence on hippocampal neurogenesis is believed to be a complex process that involves many gene $\times$ gene interactions that stimulate BDNF release. Interestingly, there is conflicting evidence whether the *BDNF* Val66Met SNP is a mediator of this process in humans. Below we review existing relationships between the Val66Met marker and hippocampal brain integrity, in addition to its relationship with other brain structures.

Relationships Between the *BDNF* Val66Met SNP and Brain Structure

Transgenic mouse models show that mice with the *BDNF* Met/Met genotype have smaller hippocampal volumes, reduced dendritic arborizations, and impaired synaptic plasticity compared with Val homozygotes and heterozygotes (Adachi et al., 2014). Smaller hippocampal volumes have been reported in a handful of human studies using structural MRI, though these outcomes have not been universally observed (reviewed in Harrisberger et al., 2015). Further, a recent joint meta-analysis of 31 published studies and one new sample ($n = 653$) did not reveal an association between the Val66Met SNP and hippocampal volumes in healthy adults (Harrisberger et al., 2015). The authors concluded that there is limited evidence for a direct relationship between the Val66Met SNP and hippocampal volume and that previous findings may be due to uniquely homogeneous samples and specific gene $\times$ gene interactions.

Apart from the hippocampus, other studies have identified a negative impact of the Met allele on gray matter of the dorsolateral prefrontal cortex, thalamus, amygdala, and fusiform gyrus and parahippocampal gyrus and total gray matter of the occipital and temporal lobes. Still, other studies employing similar analyses have not seen these effects (Forde et al., 2014). As with *BDNF* expression, it is possible that *BDNF* gene $\times$ brain relationships are dependent on moderating variables and that the direction of these relationships are region specific. For example, Van Velzen et al. (2016) revealed lower amygdala volume in maltreated children with at least one Met allele, whereas maltreated subjects with the Val/Val genotype demonstrated reduced cortical thickness.

Only a handful of studies have investigated the impact of the Val66Met SNP on white matter integrity, and there has been limited consistency in the observed outcomes. Perhaps the most interesting findings are from diffusion tensor imaging (DTI) studies showing increased fractional anisotropy (FA) (a marker of axonal integrity) in white matter tracts of healthy adult Met allele carriers. Higher FA is generally associated with more intact structure, suggesting an advantageous effect of the Met allele on white matter connectivity (Chiang et al., 2011). Although these studies group homozygous and heterozygous Met carriers together, met-carrying persons showed higher FA in the corpus callosum, fornix, inferior longitudinal fasciculus, superior longitudinal fasciculus, cingulum bundle, posterior thalamic radiation, posterior corona radiata, and internal capsule. In the largest of these studies, Chiang et al. (2011) examined white matter microstructure in 455 adult twins to determine the degree to which *BDNF* could account for genetic variance in tract-specific FA. Results revealed an FA deficit of 15% in major fiber bundles of Val allele carriers, and *BDNF* explained approximately 80% of the variance in FA (Forde et al., 2014).

More recent work suggests that the lack of a homozygous Met/Met group in previous studies has confounded earlier findings in both gray and white matter tissue. Forde et al. (2014) revealed associations between the heterozygous Val/Met genotype and cortical intrinsic curvature (a measure of surface morphology and cortical connectivity) that was distinct from either homozygous genotype. In addition, analysis of two common neuroimaging techniques, voxel-based morphometry and tract-based spatial statistics, revealed stronger associations between the Val66Met marker and gray matter (adjusted $R^2 = 25\text{--}48\%$) versus white matter (adjusted $R^2 = 14\%$).

Collectively these results suggest that the Val66Met marker influences *BDNF* brain functions other than neurogenesis. Forde et al. (2014) hypothesized that stronger relationships between Val/Met and gray matter indicate that the main effect of this SNP occurs primarily in gray matter synapses where it can modulate activity-dependent release of *BDNF*.

Relationships Between BDNF and Neuropsychiatric Function

There is substantial evidence that BDNF plays an important role in emotional health. Indeed, the *neurotrophin hypothesis of depression* originated from animal studies revealing inverse correlations between BDNF levels and stress-induced depressive behaviors, as well as increases in BDNF expression following chronic antidepressant administration (Russo-Neustadt, Beard, & Cotman, 1999). In humans, a recent large-scale study ($N = 1,751$) revealed that individuals with chronic and remitted depression demonstrate a sharper decline in serum BDNF over a 2-year period compared with those with incident depression and healthy controls. Further, the degree of BDNF decline was similar between the incident depression and control groups (Bus et al., 2015), and initiation or discontinuation of antidepressants did not significantly alter the trajectory of BDNF levels over the course of 2 years. Bus et al. (2015) concluded that depression contributes to lower levels of BDNF rather than low BDNF being causally linked to depression. The authors further speculated that a 2-year time period was too brief to observe meaningful changes in BDNF due changes in antidepressant treatment regimens.

With regard to the Val66Met SNP, there is inconclusive evidence that the Met allele increases risk for depression. However, a meta-analysis by Hosang, Shiles, Tansey, McGuffin, & Uher (2014) revealed a moderating effect of the Met allele between total life stress and depression. This interaction was stronger when life stress was stratified to stressful life events (broadly defined as negatively impactful events) rather than childhood adversity. Finally, additional work shows that sex interacts with *BDNF* SNPs to impact risk for major depressive disorder (MDD). A meta-analysis by Verhagen et al. (2010) showed that males carrying one or two Met alleles are at an increased risk for MDD—an effect that was not observed in females.

There are also conflicting findings regarding the relationship between anxiety disorders, peripheral BDNF levels, and the Val66Met marker. While meta-analyses of the Val66Met SNP fail to show elevated risk for anxiety disorders, a meta-analysis by Suliman, Hemmings, and Seedat (2013) revealed significantly lower plasma BDNF across a range of anxiety conditions. In this latter study, a stratified analysis examining peripheral BDNF in individuals with post-traumatic stress disorder (PTSD) and obsessive-compulsive disorder (OCD) showed lower BDNF in OCD patients, but not in those with PTSD. These incongruent effects of peripheral BDNF and the Val66Met SNP are common, though not well understood. For example, Hemmings et al. (2013) did not observe a direct link between the Val66Met SNP and OCD, but did report a dose-dependent effect of the Met allele as a moderator between childhood emotional abuse and OCD (Hemmings et al., 2013). Conversely, Moreira et al. (2015) *did* reveal a direct effect of the Met variant on increased risk for general anxiety disorder. This study implicates a generalized role for the Val66Met SNP on anxiety-related psychopathology and may explain the lack of relationship with distinct anxiety phenotypes. Alternatively, discrepancies between peripheral BDNF and the Val66Met marker may be a result of downstream interactions with other *BDNF* gene polymorphisms.

The Met allele has also been identified as a risk factor for substance use disorders, anthropometric characteristics, schizophrenia, and personality traits, though none of these findings have been consistently replicated. For many of these investigations, the Met allele served as a moderator of dysfunction between biobehavioral phenotypes and was not in itself a marker of disease risk. However, evidence for direct genetic effects can be observed from GWAS. To date, GWAS have identified the Met variant as a marker of antidepressant efficacy (Colle et al., 2015), introversion (Schlessinger et al., 2016), childhood obesity, and BMI (Mei et al., 2012). Interestingly, GWAS have identified several SNPs in the *BDNF* gene that are associated with BMI (rs4074134, rs4923461, rs925946, rs10501087, rs6265, rs10767664, and rs2030323),

which may be a result of activity-dependent release in the hypothalamus or brain stem (for review see Waterhouse & Xu, 2013).

Relationships Between BDNF and Cognition

Several studies show that the Met allele is detrimental to a wide range of cognitive functions, including episodic memory, delayed recall, working memory, perceptual speed, visuospatial skills, and general cognitive ability (reviewed in Mandelman & Grigorenko, 2012). Interestingly, however, a recent study of 500 older adults reported associations between high BDNF expression and a slower rate of cognitive decline over a 6-year period, yet BDNF expression did not differ according to Met and Val gene variants, and the Val/Met marker was not associated with cognitive status (Buchman et al., 2016). Similarly, a meta-analysis of 23 studies ($N = 7,095$) did not reveal significant associations between the Val66Met SNP and cognitive phenotypes (Mandelman and Grigorenko, 2012). Mandelman and Grigorenko proposed a number of explanations for this finding. Most notably, the authors postulated that a lack of effect could be due to the functional diversity of BDNF isoforms across different brain regions, which may influence cognitions that share bioactive active pathways rather than behavioral similarities. This hypothesis is consistent with evidence that truncated BDNF (a pro-BDNF isoform, ~28kDa) is a better predictor of cognitive performance and affective symptoms rather than total BDNF (exon IX) in serum of schizophrenic patients (Carlino et al., 2011). This is further supported with evidence that abnormal ratios of pro- and mature domains of pro-BDNF result in divergent pathological outcomes (Hempstead, 2015).

Interactions between exercise and BDNF have been shown to enhance various cognitive functions. Activity-dependent release of BDNF is the most common triggering mechanism in the regulated pathway, in which the majority of BDNF is transported to postsynaptic dendrites and spines. A 2015 meta-analysis shows that a single session of exercise can moderately increase BDNF levels, independent of assay type (serum vs. plasma) (Szuhany, Bugatti, & Otto, 2015). Further, interactions between exercise and BDNF on brain function have been consistently demonstrated in the literature, particularly with regard to their impact on learning and memory. It is well established that exercise enhances encoding and consolidation of new memories, and BDNF is a crucial mediator of this process. Recent work now shows that interactions between exercise and the Val66Met SNP significantly influence emotional memory consolidation. Keyan and Bryant (2016) demonstrated that brief aerobic exercise predicted an interaction between increased cortisol response and the Val homozygous genotype to enhance recall of positive, but not negative, memories. No interaction was observed among Met allele carriers, suggesting that Val homozygotes are more likely to exhibit memory benefits from exercise-induced cortisol secretion. The authors hypothesized that the lack of interaction in Met carriers may be due to reduced stress reactivity and blunted cortisol release compared with Val/Val genotypes, which has been demonstrated in earlier work (Keyan and Bryant, 2016).

Finally, the Met allele may play an important role in neurodegenerative disease. In a series of studies by Lim et al. (reviewed in Lim et al., 2016), Met allele carriers demonstrated a more severe preclinical presentation of Alzheimer's disease (AD) endophenotypes compared with highly penetrant mutations of the *APP*, *PSEN1*, and *PSEN2* genes. Lim et al. (2016) further revealed increased levels of tau and phosphorylated tau in cerebrospinal fluid of Met allele carriers, in addition to more pronounced deficits in hippocampal glucose metabolism and episodic memory. In patients with Parkinson's disease (PD), Met allele carriers exhibit greater cognitive impairments (Guerini et al., 2009) and early symptoms of dyskinesia (Foltynie et al.,

2009) compared with noncarriers. However, an association between the Met allele and risk for both AD and PD is not universally reported.

Conclusion

In sum, BDNF is an important neurohormone that supports brain health. Results from genetic studies collectively point to a role for the Val66Met polymorphism in phenotypic variation, yet the nature of this relationship remains highly elusive. The observed discrepancies across *BDNF* gene studies are likely due to a multitude of intervening variables, suggesting that genetic influence of BDNF expression is mechanistically specific. More conclusive evidence for gene $\times$ brain $\times$ behavior relationships may be observed via meta-analyses across more homogeneous population samples in order to capture the specificity of these interactions.

Author Biographies

Lauren E. Salminen, PhD, is a postdoctoral scholar at the Imaging Genetics Center of the University of Southern California in Los Angeles. Her work focuses on the use of advanced methodologies to understand phenotypic brain and behavioral signatures of aging and stress-related conditions, particularly those with early life origins. Currently her research leverages “big data” approaches to conduct high-powered studies of the human brain using structural neuroimaging, genetics, and machine learning.

Robert H. Paul, PhD ABPP-CN, is a board-certified clinical neuropsychologist. He serves as director of the Missouri Institute of Mental Health and professor of psychological sciences at the University of Missouri, St. Louis. His research program is focused on mechanisms of brain dysfunction in health conditions that primarily impact brain structures located deep beneath the surface of the cerebral cortex. He has special interest in clinical neuroscience capacity building in resource-limited environments.

References

- Adachi, N., Numakawa, T., Richards, M., Nakajima, S., & Kunugi, H. (2014). New insight in expression, transport, and secretion of brain-derived neurotrophic factor: Implications in brain-related diseases. *World Journal of Biological Chemistry*, 5(4), 409–428. doi:10.4331/wjbc.v5.i4.409
- Buchman, A. S., Yu, L., Boyle, P. A., Schneider, J. A., De Jager, P. L., & Bennett, D. A. (2016). Higher brain BDNF gene expression is associated with slower cognitive decline in older adults. *Neurology*, 86, 735–741. doi:10.1212/WNL.0000000000002387
- Bus, B. A. A., Molendijk, M. L., Tendolkar, I., Penninx, B. W. J. H., Prickaerts, J., Elzinga, B. M., & Voshaar, R. C. O. (2015). Chronic depression is associated with a pronounced decrease in serum brain-derived neurotrophic factor over time. *Molecular Psychiatry*, 20(5), 602–608. doi:10.1038/mp.2014.83
- Carlino, D., Leone, E., Di Cola, F., Baj, G., Marin, R., Dinelli, G., ... De Vanna, M. (2011). Low serum truncated-BDNF isoform correlates with higher cognitive impairment in schizophrenia. *Journal of Psychiatric Research*, 45(2), 273–279. doi:10.1016/j.jpsychires.2010.06.012
- Chiang, M. C., Barysheva, M., Toga, A. W., Medland, S. E., Hansell, N. K., James, M. R., ... Thompson, P. M. (2011). BDNF gene effects on brain circuitry replicated in 455 twins. *NeuroImage*, 55(2), 448–454. doi:10.1016/j.neuroimage.2010.12.053

- Clemenson, G. D., Deng, W., & Gage, F. H. (2015). Environmental enrichment and neurogenesis: From mice to humans. *Current Opinion in Behavioral Sciences*, 4, 56–62. doi:10.1016/j.cobeha.2015.02.005
- Colle, R., Deflesselle, E., Martin, S., David, D. J., Hardy, P., Taranu, A., ... Corruble, E. (2015). BDNF/TRKB/P75NTR polymorphisms and their consequences on antidepressant efficacy in depressed patients. *Pharmacogenomics*, 16(9), 997–1013. doi:10.2217/pgs.15.56
- Egan, M. F., Kojima, M., Callicott, J. H., Goldberg, T. E., Kolachana, B. S., Bertolino, A., ... Lu, B. (2003). The BDNF val66met polymorphism affects activity-dependent secretion of BDNF and human memory and hippocampal function. *Cell*, 112(2), 257–269.
- Foltynie, T., Cheeran, B., Williams-Gray, C. H., Edwards, M. J., Schneider, S. A., Weinberger, D., ... Bhatia, K. P. (2009). BDNF val66met influences time to onset of levodopa induced dyskinesia in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, 80(2), 141–144. doi:10.1136/jnnp.2008.154294
- Forde, N. J., Ronan, L., Suckling, J., Scanlon, C., Neary, S., Holleran, L., ... Cannon, D. M. (2014). Structural neuroimaging correlates of allelic variation of the BDNF val66met polymorphism. *NeuroImage*, 90, 280–289. doi:10.1016/j.neuroimage.2013.12.050
- Guerini, F. R., Beghi, E., Riboldazzi, G., Zangaglia, R., Pianezzola, C., Bono, G., ... Clerici, M. (2009). BDNF Val66Met polymorphism is associated with cognitive impairment in Italian patients with Parkinson's disease. *European Journal of Neurology*, 16(11), 1240–1245. doi:10.1111/j.1468-1331.2009.02706
- Harrisberger, F., Smieskova, R., Schmidt, A., Lenz, C., Walter, A., Wittfeld, K., ... Borgwardt, S. (2015). BDNF Val66Met polymorphism and hippocampal volume in neuropsychiatric disorders: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 55, 107–118. doi:10.1016/j.neubiorev.2015.04.017
- Hemmings, S. M. J., Lochner, C., van der Merwe, L., Cath, D. C., Seedat, S., & Stein, D. J. (2013). BDNF Val66Met modifies the risk of childhood trauma on obsessive-compulsive disorder. *Journal of Psychiatric Research*, 47(12), 1857–1863. doi:10.1016/j.jpsychires.2013.08.012
- Hempstead, B. L. (2015). Brain-derived neurotrophic factor: Three ligands, many actions. *Transactions of the American Clinical and Climatological Association*, 126, 9–19.
- Hosang, G. M., Shiles, C., Tansey, K. E., McGuffin, P., & Uher, R. (2014). Interaction between stress and the BDNF Val66Met polymorphism in depression: A systematic review and meta-analysis. *BMC Medicine*, 12(1), 7. doi:10.1186/1741-7015-12-7
- Keyan, D., & Bryant, R. A. (2016). Role of BDNF val66met polymorphism in modulating exercised-induced emotional memories. *Psychoneuroendocrinology*, 77, 150–157. doi:10.1016/j.psyneuen.2016.12.013
- Lim, Y. Y., Hassenstab, J., Cruchaga, C., Goate, A., Fagan, A. M., Benzinger, T. L., ... Chhatwal, J. (2016). BDNF Val66Met moderates memory impairment, hippocampal function and tau in preclinical autosomal dominant Alzheimer's disease. *Brain*, 139(Pt 10), 2766–2777. doi:10.1093/brain/aww200
- Lu, B., Pang, P. T., & Woo, N. H. (2005). The yin and yang of neurotrophin action. *Nature Reviews Neuroscience*, 6(8), 603–614. doi:10.1038/nrn1726
- Mandelman, S. D., & Grigorenko, E. L. (2012). BDNF Val66Met and cognition: All, none, or some? A meta-analysis of the genetic association. *Genes, Brain and Behavior*, 11(2), 127–136. doi:10.1111/j.1601-183X.2011.00738
- Mei, H., Chen, W., Jiang, F., He, J., Srinivasan, S., Smith, E. N., ... Berenson, G. S. (2012). Longitudinal replication studies of GWAS risk SNPs influencing body mass index over the course of childhood and adulthood. *PLoS One*, 7(2), e31470. doi:10.1371/journal.pone.0031470
- Moreira, F. P., Fabião, J. D., Bittencourt, G., Wiener, C. D., Jansen, K., Oses, J. P., ... Pinheiro, R. T. (2015). The Met allele of BDNF Val66Met polymorphism is associated with increased BDNF levels in generalized anxiety disorder. *Psychiatric Genetics*, 25(5), 201–207. doi:10.1097/YPG.0000000000000097

- Russo-Neustadt, A., Beard, R. C., & Cotman, C. W. (1999). Exercise, antidepressant medications, and enhanced brain derived neurotrophic factor expression. *Neuropsychopharmacology*, 21(5), 679–682. doi:10.1016/S0893-133X(99)00059-7
- Schlessinger, D., Costa, P., Ferrucci, L., Tanaka, T., Terracciano, A., Deiana, B., ... Sanna, S. (2016). BDNF Val66Met is associated with introversion and interacts with 5-HTTLPR to influence Neuroticism. *Neuropsychopharmacology*, 35(5). doi:10.1038/npp.2009.213
- Suliman, S., Hemmings, S. M., & Seedat, S. (2013). Brain-Derived Neurotrophic Factor (BDNF) protein levels in anxiety disorders: Systematic review and meta-regression analysis. *Frontiers in Integrative Neuroscience*, 7, 55. doi:10.3389/fnint.2013.00055
- Szuhany, K. L., Bugatti, M., & Otto, M. W. (2015). A meta-analytic review of the effects of exercise on brain-derived neurotrophic factor. *Journal of Psychiatric Research*, 60, 56–64. doi:10.1016/j.jpsychires.2014.10.003
- van Velzen, L. S., Schmaal, L., Jansen, R., Milaneschi, Y., Opmeyer, E. M., Elzinga, B. M., ... Penninx, B. W. (2016). Effect of childhood maltreatment and brain-derived neurotrophic factor on brain morphology. *Social Cognitive and Affective Neuroscience*, 11(11), 1841–1852. doi:10.1093/scan/nsw086
- Verhagen, M., Van Der Meij, A., Van Deurzen, P. A. M., Janzing, J. G. E., Arias-Vasquez, A., Buitelaar, J. K., & Franke, B. (2010). Meta-analysis of the BDNF Val66Met polymorphism in major depressive disorder: Effects of gender and ethnicity. *Molecular Psychiatry*, 15(3), 260–271. doi:10.1038/mp.2008.109
- Waterhouse, E. G., & Xu, B. (2013). The skinny on brain-derived neurotrophic factor: Evidence from animal models to GWAS. *Journal of Molecular Medicine*, 91(11), 1241–1247. doi:10.1007/s00109-013-1071-8
- Woollett, K., & Maguire, E. A. (2011). Acquiring “the Knowledge” of London’s layout drives structural brain changes. *Current Biology*, 21(24), 2109–2114. doi:10.1016/j.cub.2011.11.018

Suggested Reading

- Avinun, R., Davidov, M., Mankuta, D., & Knafo-Noam, A. (2018). Predicting the use of corporal punishment: Child aggression, parent religiosity, and the BDNF gene. *Aggressive Behavior*, 44(2), 165–175. doi:10.1002/ab.21740
- Cohen-Gilbert, J. E., Stein, E. R., Gunnar, M. R., & Thomas, K. M. (2018). Association of early stress and BDNF genotype with response inhibition during emotional distraction in adolescence. *The Journal of Early Adolescence*, 38(9), 1265–1285. doi:10.1177/2F0272431616675975
- Young, D. A., Neylan, T. C., O’Donovan, A., Metzler, T., Richards, A., Ross, J. A., & Inslicht, S. S. (2018). The interaction of BDNF Val66Met, PTSD, and child abuse on psychophysiological reactivity and HPA axis function in a sample of Gulf War Veterans. *Journal of Affective Disorders*, 235, 52–60. doi:10.1016/j.jad.2018.04.004
- Youssef, M. M., Underwood, M. D., Huang, Y. Y., Hsiung, S. C., Liu, Y., Simpson, N. R., ... Mann, J. J. (2018). Association of BDNF Val66Met polymorphism and brain BDNF levels with major depression and suicide. *International Journal of Neuropsychopharmacology*, 21(6), 528–538. doi:10.1093/ijnp/pyy008
- Zhao, M., Chen, L., Yang, J., Han, D., Fang, D., Qiu, X., ... Jiang, S. (2017). BDNF Val66Met polymorphism, life stress and depression: A meta-analysis of gene-environment interaction. *Journal of Affective Disorders*. doi:10.1016/j.jad.2017.10.024

The Insula in Women with Posttraumatic Stress Disorder

Steven E. Bruce¹, Katherine R. Buchholz^{2,3}, and
Wilson J. Brown⁴

¹*Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA*

²*VA Boston Healthcare System, Boston, MA, USA*

³*National Center for Post Traumatic Stress Disorder, White River Junction, VT, USA*

⁴*Pennsylvania State University, The Behrend College, Erie, PA, USA*

Introduction

The experience of trauma is common, and the majority of individuals will experience at least one traumatic event within their lifetime (Breslau, 2009; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Approximately 20% of women and 8% of men who experience a traumatic event develop posttraumatic stress disorder (PTSD) (Kessler et al., 1995). PTSD consists of persistent symptoms from four clusters, re-experiencing, effortful avoidance, negative alterations in mood and cognitions, and hyperarousal, after exposure to trauma (DSM-5; American Psychiatric Association [APA], 2013). Given that PTSD requires an etiological traumatic event, it is often thought of as a fear disorder in which an overgeneralized fear response interferes with the cognitive and emotional processing of the traumatic event leading to symptoms related to PTSD (Rauch, Shin, & Phelps, 2006). Others suggest that PTSD should be considered a disorder of affect dysregulation that involves a broader range of emotions beyond fear (Frewen & Lanius, 2006). Both of these models are reflected in research on the neural mechanisms that underlie PTSD and include the insula as a brain region that contributes significantly to the disorder.

This chapter will focus specifically on the insula in women with PTSD. First the importance of investigating the structure and function of brain regions specific to women will be discussed along with why it is thought there may be meaningful sex differences in the insula. Further background information regarding why gender differences in the insula may exist will be discussed. The chapter will then focus on research examining gender differences in the insula,

covering research on structural, functional, and connectivity findings. The information will be summarized, and future directions will be discussed.

Scientific Premise for Sex Differences in PTSD

Traditionally, neuroscience research has utilized male participants, although this has changed in human neuroimaging research over the past 20 years. Despite the increase in female participants, many neuroimaging studies present results that include men and women instead of examining gender-specific findings (Beery & Zucker, 2011). Investigation of gender-specific neural circuitry in PTSD imaging research is necessary given the fact that brain regions associated with PTSD, such as the insula, are sexually dimorphic (Ruigrok et al., 2014). It is possible that these sex differences impact neural function that affect the development, maintenance, and recovery of PTSD. Further, the higher prevalence rates of PTSD in women as compared with men highlight the importance of examining neural circuitry specific to women with PTSD. Although men have a higher occurrence of trauma exposure, the lifetime prevalence of PTSD is more than two times greater in women than men (Breslau, 2009; Kessler et al., 1995). This suggests that understanding mechanisms specifically associated with the development of PTSD in women may have a great impact.

There are social, environmental, and biological factors that likely contribute to differences in neural circuitry between men and women with PTSD. First, the type of traumatic events that men and women experience may impact gender differences in neural mechanisms. Although there are some discrepancies across the research, women are more likely than men to experience rape and sexual molestation (Breslau, 2009; Kessler et al., 1995; Koenen & Widom, 2009), while men are more likely than women to experience witnessing someone being injured or killed, having a life-threatening accident, physical attacks, and combat experience (Kessler et al., 1995). Some studies report a greater level of childhood trauma in women (Kessler et al., 1995), although other research has found that men have comparable with higher levels of childhood physical abuse and neglect than women (Koenen & Widom, 2009). Regardless, it appears that men and women are more likely to experience different types of traumatic events, and the differences in trauma types may differentially impact neurobiology.

Beyond the type of traumatic events experienced, women have a higher conditional risk of developing PTSD after they experience a traumatic event (Breslau, 2009; Kessler et al., 1995). It has been suggested that perhaps women are more likely to experience traumas that are associated with the development of PTSD such as rape. However, research has found that the type of traumatic event does not account for the full difference in conditional risk of PTSD (Breslau, 2009) and that women appear to have a greater sensitivity to assaultive trauma (Breslau & Anthony, 2007; Cromer & Smyth, 2010). Specifically, women as compared with men are more likely to develop PTSD following an assaultive traumatic event. Conversely, men and women have a similar conditional risk of PTSD after non-assaultive traumatic events. The mechanism through which women are more likely to develop PTSD after assaultive trauma is unknown; however, it has been suggested that “assault is a more normative experience for men than women, often occurring in the process of rough-and-tumble play that tends to be more common to boys” (Breslau and Anthony, 2007, p. 611). The higher conditional risk of PTSD for women may indicate that women process assaults differently than men and this may affect underlying neural mechanisms.

Biological sex differences in hormones may also be associated with differential neural circuitry in men and women. Specifically, it has been posited that low estrogen may be a risk

factor for developing posttraumatic stress symptoms (Glover et al., 2012; Lebron-Milad, Graham, & Milad, 2012). The majority of research on estrogen and PTSD utilizes fear conditioning protocols to examine the impact of different estrogen levels on fear and safety learning (i.e., Glover et al., 2012; Merz et al., 2012). It is thought that PTSD is associated with deficits in processes related to fear extinction or inhibition (Jovanovic, Kazama, Bachevalier, & Davis, 2012; Jovanovic & Ressler, 2010). Glover and colleagues (2012) found that women with low estrogen levels as compared with women with high estrogen had significantly higher conditioned fear response during extinction training. Another study (Merz et al., 2012) comparing women on oral contraceptive with men and free-cycling women found unique contributions of cortisol on fear learning in each group. In men and free-cycling women, cortisol decreased fear learning. Conversely, fear learning was heightened when women on oral contraceptive were given cortisol. Although the role of hormones is not fully understood, current research suggests that estrogen may affect neural processes related to PTSD.

The Insula in PTSD

While brain regions such as the amygdala have been considered an integral component of the neurocircuitry model of PTSD since its inception (Rauch et al., 2000), the insula has only recently been implicated in the neural response to trauma. The insula, a small, bilateral cortical structure with anterior connections to the amygdala and posterior connections to the sensory cortex and thalamus, is thought to be responsible for several aspects of emotional processing and functioning, including the experience of core affect (Wager & Barrett, 2004). Recent attention directed at the role of the insula in the neurocircuitry of PTSD is plausible, given the wide range of possible affective responses an individual might experience after a trauma, and the well-known role of the insula in the experience of “disgust” (described more fully below). Inclusion of the insula within the neurocircuitry model of PTSD has coincided with alterations to the diagnostic criteria for PTSD in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) (APA, 2013). Among these changes was the incorporation of a new symptom cluster characterized by “negative alterations in cognitions and mood associated with the traumatic event(s),” which highlights the importance of understanding the multiple functions of the insula in PTSD.

Functions of the Insula

A number of neuroimaging studies have attempted to identify the functional responsibilities related to emotion of various subregions of the insula. In addition to the experience of core affect, the anterior insula is thought to be responsible for the conscious processing of subjective emotions (Craig, 2002, 2009). Lindquist, Wager, Kober, Bliss-Moreau, & Feldman Barrett (2012) specifically identify the left anterior insula as an important region related to the experience of anger and the right anterior insula as an important region related to the perception of disgust. These subregions may be implicated in a maladaptive response to an interpersonal trauma, as Lilly and Valdez (2012) highlight that interpersonal trauma is often associated with a complex emotional response inclusive of guilt, sadness, shame, disgust, and anger.

Another subregion purportedly important for the experience and processing of emotions is the posterior insula. Given that this cluster of neurons projects to the sensory cortex, the posterior insula is thought to be responsible for the transmission of homeostatic information such as pain, temperature, and physical touch (Rolls, Grabenhorst, & Parris, 2008). As such, the posterior insula is a reasonable anatomical substrate for physiological perceptions of and

response to emotion. Activation of this subregion is believed to underlie abnormal hyperarousal symptoms experienced by trauma survivors.

A review conducted by Garfinkel and Liberzon (2009) also identifies relationships between the dorsal anterior insula and motivational states, cognitive control, and autobiographical memory. Within the context of the neurocircuitry of PTSD, overlap between the dorsal anterior insula and the formation, storage, and processing of autobiographical memories is intriguing, as the dysfunction among these processes is likely contributes to the occurrence of re-experiencing symptoms following a trauma. Given the multitude of functions for which various subregions of the insula are responsible, the identification of subregions and specific patterns of insula activation that are clinically relevant to PTSD remains a priority. Studies that have examined the insula in traumatized populations have preliminarily focused on the anterior insula as a key subregion in the neural dysfunction of PTSD (Hopper, Frewen, van der Kolk, & Lanius, 2007; Simmons et al., 2008), as this subregion is hypothesized to play an important role in salience processing (Koch et al., 2014).

PTSD and the Insula

Generally, individuals with PTSD exhibit hyperactivation within the insula as observed on neuroimaging (Etkin & Wager, 2007). Individuals with PTSD also demonstrate differential bilateral activation in the left and right insula in comparison with trauma-exposed and healthy control groups. A meta-analysis of the functional imaging literature by Patel, Spreng, Shin, and Girard (2012) found reliably increased activation in the right insula and left anterior insula in individuals with PTSD in comparison with healthy controls (Fonzo et al., 2010). They also reported that individuals with PTSD demonstrated increased activation in the left insula in comparison with a trauma-exposed control group in two studies: one of which utilized an implicit affective processing paradigm (Sakamoto et al., 2005) and the other that employed an associative learning paradigm (Werner et al., 2009).

However, decreased activation in the left insula in PTSD compared with a trauma-exposed individuals has been reported in Bremner et al. (2005) and Fonzo et al. (2010), often explained as a result of different activation patterns in the subregions of the insula, different paradigms across studies, and task conditions within studies. Some groups argue that decreased activity in the right anterior insula is related to the impaired ability of individuals with PTSD to anticipate and modify interoceptive changes (Simmons, Strigo, Matthews, Paulus, & Stein, 2009; Zhang et al., 2013). However, the relationships described above remain inconclusive, as the potential mediating factors, including gender and trauma type, have yet to be fully explored.

Mixed-trauma and mixed-gender imaging studies have reported relationships between BOLD signals in the insula and PTSD symptom presentations. For example, individuals diagnosed with dissociative PTSD demonstrated enhanced bilateral insula activation in response to the stimulus presentation of nonconscious fearful faces in comparison with individuals with non-dissociative PTSD (Felmingham et al., 2008). This study also reported that dissociation was positively correlated with bilateral insula activation in the conscious fear condition and positively correlated with left insula activation in the nonconscious fear condition. In a sample of victims of motor vehicle accidents (71% female), higher levels of self-reported alexithymia were associated with a greater BOLD signal in the right posterior insula, and lower levels of alexithymia were associated with a greater BOLD signal in the right anterior insula (Frewen et al., 2008). Earlier analyses of this sample yielded a significant positive correlation between right anterior insula activity and re-experiencing symptoms and a significant negative correlation between right anterior insula activity and dissociative symptoms during script-driven

trauma imagery (Hopper et al., 2007). Lanius and colleagues (2007) reported greater activation in the left insula among individuals with PTSD compared with individuals with comorbid PTSD and major depression. This finding promotes further exploration of the left insula as a neural determinant of depressive symptoms following a traumatic event.

Another study found greater activation in the right insula in a sample of male and female victims of “civilian” trauma in comparison with healthy controls (Lindauer et al., 2008). Wei, Wang, Heppner, and Du (2012) reported decreased activation in the right insula in a sample of natural disaster survivors with mild to moderate posttraumatic stress symptoms in comparison with a healthy control group during a monetary donation task, providing further evidence for the role of the insula in the reward/motivational pathway. A sample of fire disaster victims with PTSD (66% female) also exhibited reduced right insular cortex activation during word encoding and retrieval tasks than a healthy control group (Chen, Li, Xu, & Liu, 2009). Interestingly, the clinical sample activated the left insular cortex in a manner similar to the control group. A number of other mixed-trauma imaging studies have also identified significant insula hyperactivation (Whalley, Rugg, Smith, Dolan, & Brewin, 2009) and hypoactivation (Xiong et al., 2013; Zhang et al., 2013) in participants with PTSD.

Functional Imaging of the Insula in Female Interpersonal Trauma Survivors with PTSD

Conclusions regarding differential insula activation in females diagnosed with PTSD as related to interpersonal trauma can only be drawn by directly comparing results from studies on all-female and all-male samples. However, the conflation of trauma type likely remains problematic, as virtually all imaging studies that include only female participants focus on interpersonal trauma survivors. Similarly, those including only male participants often examine combat trauma. Nonetheless, findings from functional imaging studies in female victims of interpersonal trauma have generally been consistent with results from other traumatized samples, including male combat veterans (i.e., Simmons & Matthews, 2012). Aupperle and colleagues (2012) examined functional activation in a sample of 41 women diagnosed with PTSD related to intimate partner violence (IPV) (i.e., “physical and/or sexual abuse committed by a romantic partner ...”). Their results indicated that the anticipation of negative images, in comparison with that of positive images, was associated with greater activation in the bilateral anterior insula. In comparison with the control group, the PTSD group also demonstrated greater activation in the right middle/anterior insula. The authors suggested that the observed insula hyperactivation in the PTSD group reflects a discrepancy between “internally focused affective networks and externally focused cognitive control networks” (Aupperle et al., 2012). In our own research (Bruce et al., 2012), we found similar results in a sample of 32 women with PTSD as a result of interpersonal violence. Compared with 21 matched healthy controls, women with PTSD exhibited significantly higher levels of activation in the insula, particularly when attending to fearful stimuli. These findings suggest that consciously attending to emotional content further increases insular activity (Bruce et al., 2012).

In a related study, women with PTSD related to IPV demonstrated increased activation in the right anterior/middle insula as compared with a healthy control group during the anticipation of negative images (Simmons et al., 2008). A follow-up study on this sample revealed that women with PTSD related to IPV failed to activate the right anterior insula when anticipating an affective stimulus shift in comparison with the healthy control group, suggesting that these women were experiencing difficulty in modulating their interoceptive responses (Simmons et al., 2009). Frewen and colleagues (2008) observed increased activation in the left insula of women with PTSD from IPV during a relaxation condition and hypothesized that

this region may be important for the awareness of autonomic arousal from pleasant affective states. They also noted a significant negative relationship between self-reported positive affect and activity within the right insula in this sample (Frewen et al., 2010).

Fonzo and colleagues (2010) reported similar findings in a group of women diagnosed with PTSD related to IPV. They observed increased activation in the left anterior insula in the PTSD group compared with healthy controls when subjects were tasked with matching fearful faces versus happy target faces. Increased activation of the right middle insula and left anterior insula was also observed in the clinical sample versus the control group when subjects were matching angry faces (Fonzo et al., 2010). A follow-up study on this sample found significant negative correlations between the severity of childhood maltreatment and activation in the left middle insula during the processing of angry faces and activation in the right anterior insula during the processing of fearful faces (Fonzo et al., 2012).

The insula may also play a role in the processing of emotional words in women with PTSD related to interpersonal violence. Bremner and colleagues (2003) observed increased activation in the right insula in response to the retrieval of emotional words from memory in their female sample of abuse survivors. Interestingly, this sample also performed an emotional Stroop task, and the trauma-exposed control group exhibited increased activation in the left insula in comparison with the women with PTSD (Bremner et al., 2004). The right middle and right anterior insula were also identified as subregions of interest in response to pain in women with PTSD related to IPV (Strigo et al., 2010). Participants demonstrated increased activation in the right middle insula during the initial painful stimulation and subsequent decreased activation in the right anterior insula that was significantly associated with avoidance symptoms of PTSD, suggesting deficits in interoceptive awareness in the clinical sample consistent with previous literature (Simmons et al., 2009, Zhang et al., 2013). However, Ludascher and colleagues (2010) reported a positive association between dissociative symptoms and activity in the left and right insula during the presentation of trauma-related script imagery in a small sample of women diagnosed with comorbid borderline personality disorder and PTSD related to interpersonal trauma.

Structural Imaging of the Insula in Female Interpersonal Trauma Survivors with PTSD

In comparison with functional imaging studies of the insula in women with PTSD related to interpersonal trauma, fewer structural studies have examined the insula as a region of interest within this population. Structural imaging studies of heterogeneous trauma samples (Corbo, Clement, Armony, Pruessner & Brunet, 2005; Chen et al., 2006, 2009; Dannlowski et al., 2011) and combat veteran samples (Chao, Weiner, & Neylan, 2013; Herringa, Phillips, Almeida, Insana, & Germain, 2012; Kasai et al., 2008) have generally identified reduced gray matter volumes in the insula in individuals with PTSD. Reduced white matter volume in tracks that innervates the insula has also been reported in a heterogeneous trauma sample of participants diagnosed with PTSD (Sun et al., 2013). Other studies (i.e., Eckart et al., 2012) have failed to find significant differences in insula volumes between heterogeneous trauma samples and controls. Fonzo and colleagues (2012) also reported no differences in insula volume between female interpersonal trauma survivors with PTSD and controls. However, a positive correlation was observed between gray matter volume in the left ventral mid-insula and the severity of childhood maltreatment. Given the positive findings across other traumatized samples, exploration of the relationship between PTSD related to interpersonal trauma and both white and gray matter volumes is warranted.

Functional Connectivity of the Insula in Female Interpersonal Trauma Survivors with PTSD

In a study of functional connectivity networks in a sample of female victims of physical or sexual assault, repeated exposure to a traumatic memory was associated with increased functional connectivity between the right amygdala and right anterior insular cortex (Cisler et al., 2014). The authors also reported increased functional connectivity between the left amygdala and right anterior insular cortex and between the medial prefrontal cortex and the right anterior insular cortex. More severe PTSD symptoms predicted increased functional connectivity between the right anterior insular cortex and related areas, suggesting enhanced network activation relates to PTSD. Cisler et al. (2014) concluded the right insular cortex is a particularly important neural mechanism underlying the fear extinction of traumatic memories.

Fonzo and colleagues (2010) found reduced functional connectivity between the left anterior insula and the dorsal anterior cingulate cortex (ACC), left amygdala, and dorsal left anterior insula seeds during a task requiring participants to match fearful versus target happy faces. Weaker associations were also found between the left and right mid-insula regions and the dorsal left anterior insula seed. Similar to results reported by Cisler and colleagues (2014), right posterior insula connectivity with the dorsal ACC was strengthened during this task (Fonzo et al., 2010). During a similar task of the processing of fearful and angry faces, a negative relationship was observed between childhood maltreatment severity and amygdala–insula connectivity in victims of IPV diagnosed with subsequent PTSD (Fonzo et al., 2012). Using a negative image anticipation paradigm, Simmons and colleagues (2008) reported significantly weaker connectivity between bilateral anterior and right anterior/middle insula and bilateral amygdala in females with PTSD related to IPV compared with controls. The authors suggest that the observed results indicate that amygdala–insula connectivity patterns in individuals with PTSD are task dependent.

More broadly, a recent study by Ross and colleagues (Ross, Lenow, Kilts, & Cisler 2018) suggests that PTSD (via the anterior insula network) is characterized by deficits in reinforcement learning for neutral/positive (i.e., nonfearful stimuli). In a sample of 15 women with PTSD as a result of assaultive violence, the investigators found weakened encoding of positive prediction errors (PE) during a general learning task compared with healthy controls. (Ross et al., 2018). They posited that impairments in PE encoding negatively impact multiple areas of functioning, including social learning and threat appraisal.

Mixed trauma of functional connectivity reveals similar results regarding the insula and related areas. For example, Sripada et al. (2012) found that male combat veterans with PTSD had higher connectivity between the right amygdala and right insula and between the left amygdala and right insula than a healthy combat-exposed control sample. Additionally, functional pathways including the insula have been implicated in relationships with dissociative symptoms (Lanius et al., 2005) and the recall of traumatic memories (Lanius et al., 2004).

Conclusions

The past decade has seen a significant increase in our understanding of the neurobiological mechanisms of PTSD. However, many gaps remain, including further elucidation of the etiology and neurobiology of PTSD within women. This is particularly important as women have a higher conditional risk of developing PTSD than men. Within the context of the neurocircuitry of PTSD, disruption of insula activity (higher or lower activation) is thought to be

associated with several PTSD symptoms, including deficits in autobiographical memories and interoceptive cues. Moreover, recent findings of altered functional connectivity of insular subregions in women with PTSD suggest important neural mechanism impairments as well as possible pathways underlying the fear extinction of traumatic memories. Collectively, the findings provide a framework to guide the development of new targeted psychological or psychiatric interventions for this chronic and debilitating disorder.

Author Biographies

Steven E. Bruce, PhD, is the director of the Center for Trauma Recovery and associate professor in the Department of Psychological Sciences at the University of Missouri–St. Louis. His research interests and clinical specializations include the treatment of anxiety and affective disorders, particularly posttraumatic stress disorder.

Katherine R. Buchholz, PhD, is a research psychologist at the National Center for PTSD, Women's Health Sciences Division, VA, Boston. Her research interests include the neural correlates of rumination in PTSD as well as factors influencing receiving mental health treatment.

Wilson J. Brown, PhD, is an assistant professor of child clinical psychology at Penn State Behrend. His research interests include the interaction between affect, trauma exposure, and PTSD as well as the utilization of translational research methodologies to understand emotion regulation in traumatized populations.

References

- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Washington, DC: Author.
- Aupperle, R. L., Allard, C. B., Grimes, E. M., Simmons, A. N., Flagan, T., Behrooznia, M., ... Stein, M. B. (2012). Dorsolateral prefrontal cortex activation during emotional anticipation and neuropsychological performance in posttraumatic stress disorder. *Archives of General Psychiatry*, 69, 360–371. doi:10.1001/archgenpsychiatry.2011.1539
- Beery, A. K., & Zucker, I. (2011). Sex bias in neuroscience and biomedical research. *Neuroscience and Biobehavioral Reviews*, 35(3), 565–572. doi:10.1016/j.neubiorev.2010.07.002
- Bremner, J. D., Vermetten, E., Schmahl, C., Vaccarino, V., Vythilingam, M., Afzal, N., ... Charney, D. S. (2005). Positron emission tomographic imaging of neural correlates of a fear acquisition and extinction paradigm in women with childhood sexual-abuse-related post-traumatic stress disorder. *Psychological Medicine*, 35, 791–806.
- Bremner, J. D., Vermetten, E., Vythilingam, M., Afzal, N., Schmahl, C., Elzinga, B., & Charney, D. S. (2004). Neural correlates of the classic color and emotional stroop in women with abuse-related posttraumatic stress disorder. *Biological Psychiatry*, 55, 612–620.
- Bremner, J. D., Vythilingam, M., Vermetten, E., Southwick, S. M., McGlashan, T., Nazeer, A., ... Charney, D. S. (2003). MRI and PET study of deficits in hippocampal structure and function in women with childhood sexual abuse and posttraumatic stress disorder. *American Journal of Psychiatry*, 160, 924–932.
- Breslau, N. (2009). The epidemiology of trauma, PTSD, and other posttrauma disorders. *Trauma, Violence & Abuse*, 10(3), 198–210. doi:10.1177/1524838009334448
- Breslau, N., & Anthony, J. C. (2007). Gender differences in the sensitivity to posttraumatic stress disorder: An epidemiological study of urban young adults. *Journal of Abnormal Psychology*, 116(3), 607–611. doi:10.1037/0021-843X.116.3.607

- Bruce, S. E., Buchholz, K., Brown, W., Yan, L., Durbin, A., & Sheline, Y. I. (2012). Altered emotional interference processing in the amygdala and insula in women with post-traumatic stress disorder. *Neuroimage. Clinical*, 2, 43–49. doi:10.1016/j.nicl.2012.11.003
- Chao, L., Weiner, M., & Neylan, T. (2013). Regional cerebral volumes in veterans with current versus remitted posttraumatic stress disorder. *Psychiatry Research*, 30, 193–201. doi:10.1016/j.psychresns.2013.03.002
- Chen, S., Li, L., Xu, B., & Liu, J. (2009). Insular cortex involvement in declarative memory deficits in patients with post-traumatic stress disorder. *BMC Psychiatry*, 9, 39. doi:10.1186/1471-244X-9-39
- Chen, S., Xia, W., Li, L., Liu, J., He, Z., Zhang, Z., et al. (2006). Gray matter density reduction in the insula in fire survivors with posttraumatic stress disorder: A voxel-based morphometric study. *Psychiatry Research*, 146, 65–72.
- Cisler, J. M., Steele, J. S., Lenow, J. K., Smitherman, S., Everett, B., Messias, E., & Kilts, C. D. (2014). Functional reorganization of neural networks during repeated exposure to the traumatic memory in posttraumatic stress disorder: An exploratory fMRI study. *Journal of Psychiatric Research*, 48, 47–55. doi:10.1016/j.jpsychires.2013.09.013
- Corbo, V., Clement, M. H., Armony, J. L., Pruessner, J. C., & Brunet, A. (2005). Size versus shape differences: Contrasting voxel-based and volumetric analyses of the anterior cingulate cortex in individuals with acute posttraumatic stress disorder. *Biological Psychiatry*, 58, 119–124.
- Craig, A. D. (2002). How do you feel? Interoception: The sense of the physiological condition of the body. *Nature Review Neuroscience*, 3, 655–666.
- Craig, A. D. (2009). How do you feel–now? The anterior insula and human awareness. *Nature Review Neuroscience*, 10, 59–70. doi:10.1038/nrn2555
- Cromer, L. D., & Smyth, J. M. (2010). Making meaning of trauma: Trauma exposure doesn't tell the whole story. *Journal of Contemporary Psychotherapy*, 40(2), 65–72. doi:10.1007/s10879-009-9130-8
- Dannlowski, U., Stuhrmann, A., Beutelmann, V., Zwanzger, P., Lenzen, T., Grotegerd, D., et al. (2011). Limbic scars: Long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biological Psychiatry*, 71, 286–293. doi:10.1016/j.biopsych.2011.10.021
- Eckart, C., Kaufmann, J., Kanowski, M., Tempelmann, C., Hinrichs, H., Elbert, T., et al. (2012). Magnetic resonance volumetry and spectroscopy of hippocampus and insula in relation to severe exposure of traumatic stress. *Psychophysiology*, 49, 261–270. doi:10.1111/j.1469-8986.2011.01303.x
- Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry*, 164, 1476–1488.
- Felmingham, K., Kemp, A. H., Williams, L., Falconer, E., Olivieri, G., Peduto, A., & Bryant, R. (2008). Dissociative responses to conscious and non-conscious fear impact underlying brain function in post-traumatic stress disorder. *Psychological Medicine*, 38, 771–780. doi:10.1017/S0033291708002742
- Fonzo, G. A., Flagan, T. M., Sullivan, S., Allard, C. B., Grimes, E. M., Simmons, A. N., ... Stein, M. B. (2012). Neural functional and structural correlates of childhood maltreatment in women with intimate-partner violence-related posttraumatic stress disorder. *Psychiatry Research*, 211, 93–103. doi:10.1016/j.psychresns.2012.08.006
- Fonzo, G. A., Simmons, A. N., Thorp, S. R., Norman, S. B., Paulus, M. P., & Stein, M. B. (2010). Exaggerated and disconnected insular-amygdalar blood oxygenation level-dependent response to threat-related emotional faces in women with intimate-partner violence posttraumatic stress disorder. *Biological Psychiatry*, 68, 433–441. doi:10.1016/j.biopsych.2010.04.028
- Frewen, P., Lane, R. D., Neufeld, R. W., Densmore, M., Stevens, T., & Lanius, R. (2008). Neural correlates of levels of emotional awareness during trauma script-imagery in posttraumatic stress disorder. *Psychosomatic Medicine*, 70, 27–31.
- Frewen, P. A., Dozois, D. J. A., Neufeld, R. W. J., Densmore, M., Stevens, T. K., & Lanius, R. A. (2010). Social emotions and emotional valence during imagery in women with PTSD: Affective and neural correlates. *Psychological Trauma: Theory, Research, Practice, and Policy*, 2(2), 145–157. <https://doi.org/10.1037/a0019154>

- Frewen, P. A., & Lanius, R. A. (2006). Toward a psychobiology of posttraumatic self-dysregulation: Reexperiencing, hyperarousal, dissociation, and emotional numbing. *Annals of the New York Academy of Sciences*, 1071, 110–124. doi:10.1196/annals.1364.010
- Garfinkel, S. N., & Liberzon, I. (2009). Neurobiology of PTSD: A review of neuroimaging findings. *Psychiatric Annals*, 39, 370–381. doi:10.3928/00485713-20090527-01
- Glover, E. M., Jovanovic, T., Mercer, K. B., Kerley, K., Bradley, B., Ressler, K. J., & Norrholm, S. D. (2012). Estrogen levels are associated with extinction deficits in women with posttraumatic stress disorder. *Biological Psychiatry*, 72(1), 19–24. doi:10.1016/j.biopsych.2012.02.031
- Herringa, R., Phillips, M., Almeida, J., Insana, S., & Germain, A. (2012). Post-traumatic stress symptoms correlate with smaller subgenual cingulate, caudate, and insula volumes in unmedicated combat veterans. *Psychiatry Research*, 203, 139–145. doi:10.1016/j.psychres.2012.02.005
- Hopper, J. W., Frewen, P. A., van der Kolk, B. A., & Lanius, R. A. (2007). Neural correlates of reexperiencing, avoidance, and dissociation in PTSD: Symptom dimensions and emotion dysregulation in responses to script-driven trauma imagery. *Journal of Traumatic Stress*, 20, 713–725.
- Jovanovic, T., Kazama, A., Bachevalier, J., & Davis, M. (2012). Impaired safety signal learning may be a biomarker of PTSD. *Neuropharmacology*, 62(2), 695–704. doi:10.1016/j.neuropharm.2011.02.023
- Jovanovic, T., & Ressler, K. J. (2010). How the neurocircuitry and genetics of fear inhibition may inform our understanding of PTSD. *American Journal of Psychiatry*, 167(6), 648–662. doi:10.1176/appi.ajp.2009.09071074
- Kasai, K., Yamasue, H., Gilbertson, M. W., Shenton, M. E., Rauch, S. L., & Pitman, R. K. (2008). Evidence for acquired pregenual anterior cingulate gray matter loss from a twin study of combat-related posttraumatic stress disorder. *Biological Psychiatry*, 63, 550–556.
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the national comorbidity survey. *Archives of General Psychiatry*, 52, 1048–1060.
- Koch, S. B., van Zuiden, M., Nawijn, L., Frijling, J. L., Veltman, D. J., & Olff, M. (2014). Intranasal oxytocin as strategy for medication-enhanced psychotherapy of PTSD: Salience processing and fear inhibition processes. *Psychoneuroendocrinology*, 40, 242–256. doi:10.1016/j.psyneuen.2013.11.018
- Koenen, K. C., & Widom, C. S. (2009). A prospective study of sex differences in the lifetime risk of posttraumatic stress disorder among abused and neglected children grown up. *Journal of Traumatic Stress*, 22(6), 566–574. doi:10.1002/jts.20478
- Lanius, R. A., Frewen, P. A., Girotti, M., Neufeld, R. W., Stevens, T. K., & Densmore, M. (2007). Neural correlates of trauma script-imagery in posttraumatic stress disorder with and without comorbid major depression: A functional MRI investigation. *Psychiatry Research*, 155, 45–56.
- Lanius, R. A., Williamson, P. C., Bluhm, R. L., Densmore, M., Boksman, K., Neufeld, R. W., ... Menon, R. S. (2005). Functional connectivity of dissociative responses in posttraumatic stress disorder: A functional magnetic resonance imaging investigation. *Biological Psychiatry*, 57, 873–884.
- Lanius, R. A., Williamson, P. C., Densmore, M., Boksman, K., Neufeld, R. W., Gati, J. S., & Menon, R. S. (2004). The nature of traumatic memories: A 4-T fMRI functional connectivity analysis. *American Journal of Psychiatry*, 161, 36–44.
- Lebron-Milad, K., Graham, B. M., & Milad, M. R. (2012). Low estradiol levels: A vulnerability factor for the development of posttraumatic stress disorder. *Biological Psychiatry*, 72(1), 6–7. doi:10.1016/j.biopsych.2012.04.029
- Lilly, M. M., & Valdez, C. E. (2012). Interpersonal trauma and PTSD: The roles of gender and a lifespan perspective in predicting risk. *Psychological Trauma: Theory, Research, Practice, and Policy*, 4(1), 140–144. doi:10.1037/a0022947
- Lindauer, R. J., Booij, J., Habraken, J. B., van Meijel, E. P., Uylings, H. B., Olff, M., ... Gersons, B. P. (2008). Effects of psychotherapy on regional cerebral blood flow during trauma imagery in patients with post-traumatic stress disorder: A randomized clinical trial. *Psychological Medicine*, 38, 543–554.
- Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Feldman Barrett, L. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences*, 35, 121–143.

- Ludascher, P., Valerius, G., Stiglmayr, C., Mauchnik, J., Lanius, R. A., Bohus, M., & Schmahl, C. (2010). Pain sensitivity and neural processing during dissociative states in patients with borderline personality disorder with and without comorbid posttraumatic stress disorder: A pilot study. *Journal of Psychiatry & Neuroscience: JPN*, 35, 177–184.
- Merz, C. J., Tabbert, K., Schweckendiek, J., Klucken, T., Vaitl, D., Stark, R., & Wolf, O. T. (2012). Oral contraceptive usage alters the effects of cortisol on implicit fear learning. *Hormones and Behavior*, 62(4), 531–538. doi:10.1016/j.yhbeh.2012.09.001
- Patel, R., Spreng, R. N., Shin, L. M., & Girard, T. A. (2012). Neurocircuitry models of posttraumatic stress disorder and beyond: A meta-analysis of functional neuroimaging studies. *Neuroscience and Biobehavioral Review*, 36, 2130–2142. doi:10.1016/j.neubiorev.2012.06.003
- Rauch, S. L., Shin, L. M., & Phelps, E. A. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: Human neuroimaging research—past, present, and future. *Biological Psychiatry*, 60(4), 376–382. doi:10.1016/j.biopsych.2006.06.004
- Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., ... Pitman, R. K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry*, 47, 769–776.
- Rolls, E. T., Grabenhorst, F., & Parris, B. A. (2008). Warm pleasant feelings in the brain. *NeuroImage*, 41, 504–513. doi:10.1016/j.neuroimage.2008.03.005
- Ross, M. C., Lenow, J. K., Kilts, C. D., & Cisler, J. M. (2018). Altered neural encoding of prediction errors in assault-related posttraumatic stress disorder. *Journal of Psychiatric Research*, 103, 83–90. doi:10.1016/j.jpsychires.2018.05.008
- Ruigrok, A. N. V., Salimi-Khorshidi, G., Lai, M.-C., Baron-Cohen, S., Lombardo, M. V., Tait, R. J., & Suckling, J. (2014). A meta-analysis of sex differences in human brain structure. *Neuroscience and Biobehavioral Reviews*, 39, 34–50. doi:10.1016/j.neubiorev.2013.12.004
- Sakamoto, H., Fukuda, R., Okuaki, T., Rogers, M., Kasai, K., Machida, T., ... Kato, N. (2005). Parahippocampal activation evoked by masked traumatic images in posttraumatic stress disorder: A functional MRI study. *NeuroImage*, 26, 813–821.
- Simmons, A. N., & Matthews, S. C. (2012). Neural circuitry of PTSD with or without mild traumatic brain injury: A meta-analysis. *Neuropharmacology*, 62, 598–606. doi:10.1016/j.neuropharm.2011.03.016
- Simmons, A. N., Paulus, M. P., Thorp, S. R., Matthews, S. C., Norman, S. B., & Stein, M. B. (2008). Functional activation and neural networks in women with posttraumatic stress disorder related to intimate partner violence. *Biological Psychiatry*, 64, 681–690. doi:10.1016/j.biopsych.2008.05.027
- Simmons, A. N., Strigo, I. A., Matthews, S. C., Paulus, M. P., & Stein, M. B. (2009). Initial evidence of a failure to activate right anterior insula during affective set shifting in posttraumatic stress disorder. *Psychosomatic Medicine*, 71, 373–377. doi:10.1097/PSY.0b013e3181a56ed8
- Sripada, R. K., King, A. P., Garfinkel, S. N., Wang, X., Sripada, C. S., Welsh, R. C., & Liberzon, I. (2012). Altered resting-state amygdala functional connectivity in men with posttraumatic stress disorder. *Journal of Psychiatry & Neuroscience: JPN*, 37, 241–249. doi:10.1503/jpn.110069
- Strigo, I. A., Simmons, A. N., Matthews, S. C., Grimes, E. M., Allard, C. B., Reinhardt, L. E., ... Stein, M. B. (2010). Neural correlates of altered pain response in women with posttraumatic stress disorder from intimate partner violence. *Biological Psychiatry*, 68, 442–450. doi:10.1016/j.biopsych.2010.03.034
- Sun, Y., Wang, Z., Ding, W., Wan, J., Zhuang, Z., Zhang, Y., ... Xu, J. (2013). Alterations in white matter microstructure as vulnerability factors and acquired signs of traffic accident-induced PTSD. *PLoS One*, 8. doi:10.1371/journal.pone.0083473
- Wager, T.D. & Barrett, L.F. (2004). *From affect to control: Functional specialization of the insula in motivation and regulation*. Retrieved from <https://canlabweb.colorado.edu/publications-v1/33-published-in-2004/4790-wager-t-d-and-barrett-l-f-2004-from-affect-to-control-functional-specialization-of-the-insula-in-motivation-and-regulation.html>
- Wei, M., Wang, K. T., Heppner, P. P., & Du, Y. (2012). Ethnic and mainstream social connectedness, perceived racial discrimination, and posttraumatic stress symptoms. *Journal of Counseling Psychology*, 59, 486–493. doi:10.1037/a0028000

- Werner, N. S., Meindl, T., Engel, R. R., Rosner, R., Riedel, M., Reiser, M., & Fast, K. (2009). Hippocampal function during associative learning in patients with posttraumatic stress disorder. *Journal of Psychiatric Research*, 43, 309–318. doi:10.1016/j.jpsychires.2008.03.011
- Whalley, M. G., Rugg, M. D., Smith, A. P., Dolan, R. J., & Brewin, C. R. (2009). Incidental retrieval of emotional contexts in post-traumatic stress disorder and depression: An fMRI study. *Brain and Cognition*, 69, 98–107. doi:10.1016/j.bandc.2008.05.008
- Xiong, K., Zhang, Y., Qiu, M., Zhang, J., Sang, L., Wang, L., ... Li, M. (2013). Negative emotion regulation in patients with posttraumatic stress disorder. *PLoS One*, 8(12), e81957. doi:10.1371/journal.pone.0081957
- Zhang, J. N., Xiong, K. L., Qiu, M. G., Zhang, Y., Xie, B., Wang, J., ... Zhang, J. J. (2013). Negative emotional distraction on neural circuits for working memory in patients with posttraumatic stress disorder. *Brain Research*, 1531, 94–101. doi:10.1016/j.brainres.2013.07.042

Suggested Reading

- Hughes, K. C., & Shin, L. M. (2011). Functional neuroimaging studies of post-traumatic stress disorder. *Expert Review of Neurotherapeutics*, 11, 275–285. PMID: 21306214
- Lei, D., Li, K., Li, L., Chen, F., Huang, X., Lui, S., ... Gong, Q. (2015). Disrupted functional brain connectome in patients with posttraumatic stress disorder. *Radiology*, 276, 818–827. doi:10.1148/radiol.15141700
- Satterthwaite, T. D., Cook, P. A., Bruce, S. E., Conway, C., Mikkelsen, E., Satchell, E., ... Sheline, Y. I. (2016). Dimensional depression severity in women with major depression and post-traumatic stress disorder correlates with fronto-amygdalar hypoconnectivity. *Molecular Psychiatry*, 21, 894–902. doi:10.1038/mp.2015.149

Language and Literacy Development

Laura O'Hara, Rebecca A. Meza, and Amanda C. Eaton

*Department of Communication Disorders and Deaf Education, Fontbonne University,
St. Louis, MO, USA*

*Department of Communication & Media, University of Missouri at Saint Louis,
St. Louis, MO, USA*

Language and Literacy Development

Oral language development provides the foundation for later reading ability in grade school (Catts, 1993). The National Institute of Child Health and Human Development (NICHD) Early Child Care Research Network (2005) established that general language proficiency prior to kindergarten predicted reading skill in first grade. Additionally, this study found that first-grade vocabulary skills were positively related to reading in third grade (NICHD, 2005).

As Paul (2012) discusses, there is no longer a question of whether the quantity and quality of language input (nurture) or the biological capacity to learn and use language (nature) impacts language development. It is clear from the extant literature that both critically impact language development. Contemporary research is now focused on delineating the interdependence of biological and environmental factors that mediate language and literacy development, as well as key therapeutic targets to improve language and literacy skills, and ideally alter the developmental trajectories toward optimal performance.

Biological capacities for language and reading begin in the brain. Verbal and written language skills are supported by the perisylvian region of the language-dominant left cortex. However, brain regions responsible for decoding written form exist in both the left and right cerebral hemispheres, and these regions are located distal to areas that underlie core auditory language comprehension (Hickok, 2009). Decoding written words requires the transformation of visual linguistic input from an orthographic (the way a word looks) to a phonological (the way a word sounds) representation. This process is believed to occur bilaterally in the angular and supramarginal gyri in the posterior temporoparietal region. Hemisphere differences exist for orthographic decoding. The right hemisphere is more sensitive to the orthographic features of printed words and considers each grapheme individually, whereas the left hemisphere is able to process unified

whole words that are greater than the sum of their parts (Chiarello, 1988). Both hemispheres complement each other and work in parallel when decoding familiar and unfamiliar words, described by Coltheart, Curtis, Atkins, and Haller (1993) as a dual-route model. Another example of reading specificity in the brain is the visual word form area (VWFA) within the lateral occipitotemporal sulcus. Cohen, Dehaene, Naccache, and Leherily (2000) used functional magnetic resonance imaging (fMRI) along with event-related potentials (ERPs) to characterize the spatial and temporal organization of reading. The VWFA selectively responded to written words relative to line drawings (Cohen et al., 2000). Early controversy existed as to whether the VWFA was solely and uniquely involved in word comprehension because the emergence of written language was too recent and utilized by too few within the total human population to have affected the evolution of the human genome. However, the contemporary hypothesis suggests that reading acquisition engages brain regions that evolved for face and object recognition through a process of neuronal recycling (Dehaene & Cohen, 2007). Therefore, neuronal recycling requires experience, which some argue must occur at specific time periods.

Additionally, later language and reading abilities are influenced by the early development of the brain. During this dynamic time, there are critical periods in which the child's nervous system is particularly impacted by environmental input (Purves et al., 2001). For instance, neuroimaging studies have reported that early literacy experience has a significant influence on the pruning and strengthening of cells and connections within the brain (Castro-Caldas, Petersson, Reis, Stone-Elander, & Ingvar, 1998).

Alarming, less tangible factors can have detrimental effects on the developing brain. For example, language and reading disorders are associated with alterations in brain anatomy and genetic factors. Using magnetic resonance imaging (MRI), De Fosse et al. (2004) found that boys with autism spectrum disorder (a disorder often accompanied with language deficits) and boys with a specific language impairment (SLI) exhibited atypical patterns of asymmetry of the language cortex. In other words, the language dominant area of the brain was found to be in the right hemisphere of the boys rather than the left hemisphere. Additionally, differences in cortical mass in left hemisphere regions and anomalous gyral and sulcal folding patterns have been observed in individuals with developmental dyslexia (Brown et al., 2001). Functional neuroimaging studies of children and teenagers with dyslexia reveal decreased activity in the left occipitotemporal region and increased activity in right hemisphere homologues (Shaywitz et al., 2002).

Studies dating back almost two decades established a heritable component to reading difficulties (Stevenson, Graham, Fredman, & McLoughlin, 1987). In this 1987 study, correlations among identical and fraternal twins were assessed, and genetics were found to contribute moderately to reading ability, after controlling for intelligence. A more recent study of identical and fraternal twin pairs found supporting evidence that genetics have an influence on reading deficits (Astrom, Wadsworth, Olson, Willcutt, & DeFries, 2012). Though research has discovered regions on chromosomes 1, 3, 6, 15, and 18 that possess multiple candidate genes implicated in individual reading differences, including reading disorders such as dyslexia (Schulte-Körne et al., 2007), the studies are complex with results that are not universally replicated. For instance, one of these candidate genes, KIAA0319 (chromosome 6), has been correlated with dyslexia susceptibility as well as multiple reading-related traits in some studies, but in another study, the association between KIAA0319 and dyslexia was not found (Dennis et al., 2009). While these findings suggest that there are genetic and biological factors associated with language and literacy abilities, more research must be completed before assuming causal relationships.

While these etiological studies highlight genetic and neurobiological factors in language and literacy development, except in the most extreme cases of intellectual disability, anyone can be taught to read. There are important reading components that have either genetic or environmental contributions to variance in reading ability, such as word recognition and oral language.

For instance, many people with reading disabilities have poor phonological processing ability, which is found to be highly heritable, while they may also struggle with the spelling of words, which is not significantly heritable (Olson, Wise, Conners, Rack, & Fulker, 1989). Based on research, interventions can be tailored to meet the needs of individuals with different learning abilities. The importance of social guidance in shaping children's development, as supported by Vygotsky's sociocultural theory (1978), is the focus of many investigations by researchers interested in language and overall cognitive development. According to Vygotsky (1978) adult-child interactions provide social and cultural guidance that supports the development of children's cognitive skills. Through parent-child interactions, children learn the behaviors and norms of their culture, including speech and language patterns, as well as any other symbolic knowledge necessary for reading.

Important Home Literacy Practices

Early reading attitudes, knowledge, and abilities, also known as emergent literacy skills (Teale & Sulzby, 1986), are vital for later language and reading competence (Senechal & LeFevre, 2002; Storch & Whitehurst, 2002). Teale and Sulzby (1986) established that the critical time for the development of language and emergent literacy skills occurs during the preschool years. Because children spend much of their time at home during their preschool years, several studies have found that there is a relationship between the home literacy environment and children's later language and literacy skills (Forget-Dubois et al., 2009; Senechal & LeFevre, 2002).

A number of different home literacy practices are capable of improving language and literacy skills. One of the most important factors in language and literacy outcomes is the quantity and quality of language input (Hart & Risley, 1995; Hoff, 2013). Hart and Risley (1995) studied the language input based on socioeconomic status (SES) and found that children living in poverty heard an estimated thirty million fewer words by age four than their higher-SES peers. Hoff (2013) found that higher-SES mothers provided longer, more complex utterances and asked more open-ended questions. The children who were exposed to more quality language had higher outcomes on language assessments.

Book reading is also strongly correlated with overall literacy (Dickinson & DeTemple, 1998; Frijters, Barron, & Brunello, 2000). The frequency of shared reading time at home corresponds to higher vocabularies and stronger literacy skills in preschool and primary school (Raikes et al., 2006). Importantly, the interactive style and quality of parental practices during shared reading time predicts literacy outcomes. For example, shared reading involving eliciting abstract language, adding information, and asking open-ended questions about material read predicts stronger language development (Whitehurst et al., 1994). Maternal sensitivity and responsiveness is also critical as supportive maternal behavior was the strongest single predictor of language and pre-academic skills when entering kindergarten (Landry, Smith, Swank, & Miller-Loncar, 2000).

Critical Components of Quality Reading Instruction

Not surprisingly, academic curriculum plays a significant role in language and literacy development. During the emergent literacy stage, it is essential that preschool literacy instruction includes phonological awareness, print concepts, alphabet knowledge, and literate language ability (Justice & Kaderavek, 2004). *Phonological awareness* refers to knowledge of syllables and rhyme, as well as the ability to blend and segment sounds within words. Knowledge

of *print concepts* is the understanding of how to hold books, how to turn the pages, left-to-right reading progression, awareness of words and letters on the page, etc. *Alphabet knowledge* is knowing the names and sounds of letters and that the sounds make up words. *Literate language* is the understanding of abstract language and awareness of narrative language.

The critical components of literacy in elementary school include phonemic awareness, phonics, reading fluency, vocabulary, and comprehension (National Reading Panel, 2000). *Phonemic awareness* refers to the ability to manipulate phonemes in spoken syllables and words (i.e., what word would you have if you changed the /t/ in cat to a /p/?). *Phonics* is the ability to understand and apply letter-sound correspondence for the purposes of reading and spelling. These lower-level processes are represented in the dual-route processing model, which states that a reader can pronounce a word aloud by following letter-sound correspondence rules starting from left to right of the letter string (i.e., hit is /h/ /I/ /t/), or the individual recognizes the word and provides the correct pronunciation instantaneously (Coltheart et al., 1993). An individual is able to read more fluently with the latter route and with words that they encounter frequently. *Vocabulary* is word knowledge or the ability to understand the words that have been correctly decoded. *Fluency* is the ability to read orally with speed, accuracy, and proper expression. Lastly, *comprehension* is the capacity to synthesize and understand new information in relation to prior experiences. There are many additional factors that are important in regard to reading comprehension, such as working memory capacity, inferencing ability, comprehension monitoring, and executive function (Cutting, Materek, Cole, Levine, & Mahone, 2009). Quality literacy instruction includes teaching all five main components because each of these five skills are interconnected such that a weakness in any element affects reading ability as a whole.

Scarborough (2003) described this interdependence of individual factors as a “reading rope” in which skilled reading results from the integration of background knowledge, vocabulary, language structures, verbal reasoning, and literacy knowledge. When combined with phonological awareness, decoding, and sight recognition, the individual strands or skills result in skilled reading (Paul, 2012).

Conclusion

Overall, language and literacy development are deeply related in a bidirectional manner (Light & McNaughton, 2009). Due to decades of research, it is well understood that genetics plays a significant role in the variation of reading abilities among people and contributes to the risk of developing a reading disorder. Fortunately, genes are not the only contributors of language and literacy development. Children who receive more opportunities for quality language interactions and who receive comprehensive literacy instruction targeting phonemic awareness, phonics, fluency, vocabulary, and comprehension achieve the greatest language and literacy proficiency (National Reading Panel, 2000). Research will continue to investigate the influence gene expression, neurobiology, and environment has on language and literacy development so that interventionists can monitor and support the language and literacy skills of all children.

Author Biographies

Laura O'Hara, PhD, CCC-SLP, is an assistant professor at Fontbonne University. She teaches graduate and undergraduate courses in the areas of sound system disorders, early childhood

language disorders, school-age language disorders, and language and literacy disorders in at-risk learners. She graduated from Fontbonne University with an undergraduate degree and a graduate degree, both in speech-language pathology, and from St. Louis University with a PhD in education (Curriculum and Instruction). She previously provided speech, language, and literacy prevention and intervention for students 3–21 years old as a full-time public school speech-language pathologist.

Rebecca A. Meza earned her master of arts degree in psychology, with an emphasis in behavioral neuroscience, from the University of Missouri–St. Louis. Rebecca is interested in applying cognitive neuroscience toward adapting and improving the education system for all learners. Her passion comes from her extensive teaching and tutoring experience; she has worked with students of various ages and economic backgrounds, as well as internationally where she was a Peace Corps volunteer in West Africa. Rebecca is currently working in the Early Emotional Development Program at the Washington University Medical Campus in St. Louis under the supervision of Dr. Joan Luby.

Amanda C. Eaton, PhD, CCC-SLP is an assistant professor in communication disorders at Fontbonne University. She received a doctorate in psychology with an emphasis in behavioral neuroscience. Her research predominantly focuses on aphasia pathophysiology and recovery, electrophysiology and right hemisphere language processes, and cognitive rehabilitation. Amanda worked as a speech-language pathologist in adult acute and subacute care and currently teaches courses in dysphagia, motor speech disorders, voice pathology, research methods, and neurogenic communication disorders. She recently found the Group Rehabilitation for Aphasia Communication Effectiveness (G.R.A.C.E.) program at the Eardley Family Clinic for Speech, Language, and Hearing, which provides group therapy for adults with aphasia in the St. Louis region.

References

- Astrom, R. L., Wadsworth, S. J., Olson, R. K., Willcutt, E. G., & DeFries, J. C. (2012). Genetic and environmental etiologies of reading difficulties: DeFries–Fulker analysis of reading performance data from twin pairs and their non-twin siblings. *Learning and Individual Differences*, 22(3), 365–369.
- Brown, W., Eliez, S., Menon, V., Rumsey, J., White, C., & Reiss, A. (2001). Preliminary evidence of widespread morphological variations of the brain in dyslexia. *Neurology*, 56, 781–783.
- Castro-Caldas, A., Petersson, K. M., Reis, A., Stone-Elander, S., & Ingvar, M. (1998). The illiterate brain. Learning to read and write during childhood influences the functional organization of the adult brain. *Brain*, 121(6), 1053–1063.
- Catts, H. H. (1993). The relationship between speech-language impairments and reading disabilities. *Journal of Speech and Hearing Research*, 36, 948–958.
- Chiarello, C. (1988). Lateralization of lexical processes in the normal brain: A review of visual half-field research. In H. A. Whitaker (Ed.), *Contemporary reviews in neuropsychology* (pp. 36–76). New York: Springer-Verlag.
- Cohen, L., Dehaene, S., Naccache, L., & Leherlily, S. (2000). The visual word form area: Spatial and temporal characterization of an initial stage of reading in normal subjects and posterior split-brain patients. *Brain*, 123, 291–307.
- Coltheart, M., Curtis, B., Atkins, P., & Haller, M. (1993). Models of reading aloud: Dual-route and parallel-distributed-processing approaches. *Psychological Review*, 100(4), 589.
- Cutting, L. E., Materek, A., Cole, C. A., Levine, T. M., & Mahone, E. M. (2009). Effects of fluency, oral language, and executive function on reading comprehension performance. *Annals of Dyslexia*, 59(1), 34–54.

- De Fosse, L., Hodge, S. M., Makris, N., Kennedy, D. N., Caviness, V. S., Jr., McGrath, L., ... Harris, G. J. (2004). Language-association cortex asymmetry in autism and specific language impairment. *Annals of Neurology*, 56(6), 757–766.
- Dehaene, S., & Cohen, L. (2007). Cultural recycling of cortical maps. *Neuron*, 56, 384–398.
- Dennis, M. Y., Paracchini, S., Scerri, T. S., Prokunina-Olsson, L., Knight, J. C., Wade-Martins, R., ... Monaco, A. P. (2009). A common variant associated with dyslexia reduces expression of the KIAA0319 gene. *PLoS Genetics*, 5(3), e1000436.
- Dickinson, D. K., & DeTemple, J. (1998). Putting parents in the picture: Maternal reports of preschoolers' literacy as a predictor of early reading. *Early Childhood Research Quarterly*, 13(2), 241–261.
- Forget-Dubois, N., Dionne, G., Lemelin, J.-P., Pérusse, D., Tremblay, R. E., & Boivin, M. (2009). Early child language mediates the relation between home environment and school readiness. *Child Development*, 80(3), 736–749.
- Frijters, J. C., Barron, R. W., & Brunello, M. (2000). Direct and mediated influences of home library and literacy interest on prereaders' oral vocabulary and early written language skill. *Journal of Educational Psychology*, 92, 466–477.
- Hart, B., & Risley, T. (1995). *Meaningful differences in the everyday experience of young American children*. Baltimore: Brookes.
- Hickok, G. (2009). The functional neuroanatomy of language. *Physics of Life Reviews*, 6(3), 121–143.
- Hoff, E. (2013). Interpreting the early language trajectories of children from low-SES and language minority homes: Implications for closing achievement gaps. *Developmental Psychology*, 49(1), 4–14.
- Justice, L., & Kaderavek, J. (2004). Embedded-explicit emergent literacy intervention I: Background and description of approach. *Language, Speech, and Hearing Services in Schools*, 35, 201–211.
- Landry, S. H., Smith, K. E., Swank, P. R., & Miller-Loncar, C. L. (2000). Early maternal and child influences on children's later independent cognitive and social functioning. *Child Development*, 71, 358–375.
- Light, J., & McNaughton, D. (2009). Meeting the demands of the curriculum for conventional and advanced readers and writers who require AAC. In G. Soto & C. Zangari (Eds.), *Practically speaking: Language, literacy, and academic development for students with AAC needs*. Baltimore, MD: Brookes Publishing Co.
- National Institute of Child Health and Human Development, Early Child Care Research Network. (2005). Pathways to reading: The role of oral language in the transition to reading. *Developmental Psychology*, 41(2), 428–442.
- National Reading Panel. (2000). *Teaching children to read: An evidence-based assessment of the scientific research literature on reading and its implications for reading instruction* (NIH Publication No. 00-4769). Washington, DC: National Institute of Child Health and Human Development.
- Olson, R., Wise, B., Conners, F., Rack, J., & Fulker, D. (1989). Specific deficits in component reading and language skills: Genetic and environmental influences. *Journal of Learning Disabilities*, 22(6), 339–348.
- Paul, R. (2012). *Language disorders from infancy through adolescence: Listening, speaking, reading, writing, and communicating* (4th ed.). Saint Louis: Elsevier.
- Purves, D., Augustine, G. J., Fitzpatrick, D., Katz, L. C., LaMantia, A. S., McNamara, J. O., & Williams, S. M. (2001). The development of language: A critical period in humans. In *Neuroscience* (2nd ed.). Sunderland, MA: Sinauer Associates.
- Raikes, H., Luze, G., Brooks-Gunn, J., Raikes, H. A., Pan, B. A., Tamis LeMonda, C. S., et al. (2006). Mother-child book reading in low income families: Correlates and outcomes during the first three years of life. *Child Development*, 77, 924–953.
- Scarborough, H. (2003). Connecting early language and literacy to later reading (dis)abilities: Evidence, theory and practice. In S. Newman & D. Dickinson (Eds.), *Handbook of early literacy research* (pp. 97–110). New York, NY: Guilford Press.
- Schulte-Körne, G., Ludwig, K., el Sharkawy, J., Nöthen, M., Müller-Myhsok, B., & Hoffmann, P. (2007). Genetics and neuroscience in dyslexia: Perspectives for education and remediation. *Mind, Brain, and Education*, 1(4), 162–172.

- Senechal, M., & LeFevre, J. (2002). Parental involvement in the development of children's reading skill: A five-year longitudinal study. *Child Development*, 73, 445–460.
- Shaywitz, B. A., Shaywitz, S. E., Pugh, K. R., Mencl, W. E., Fulbright, R. K., Skudlarski, P., ... Gore, J. C. (2002). Distribution of posterior brain system for reading in children with developmental dyslexia. *Biological Psychiatry*, 52(2), 101–110.
- Stevenson, J., Graham, P., Fredman, G., & McLoughlin, V. (1987). A twin study of genetic influences on reading and spelling ability and disability. *Journal of Child Psychology and Psychiatry*, 28(2), 229–247.
- Storch, S. A., & Whitehurst, G. J. (2002). Oral language and code-related precursors to reading: Evidence from a longitudinal structural model. *Developmental Psychology*, 38, 934–947.
- Teale, W. H., & Sulzby, E. (1986). Emergent literacy as a perspective for examining how young children become writers and readers. In W. H. Teale & E. Sulzby (Eds.), *Emergent literacy: Writing and reading* (pp. 7–27). Norwood, NJ: Ablex.
- Vygotsky, L. S. (1978). *Mind in society*. Cambridge, MA: Harvard University Press.
- Whitehurst, G. J., Arnold, D. S., Epstein, J. N., Angell, A. L., Smith, M., & Fischel, J. E. (1994). A picture book reading intervention in day care and home for children from low-income families. *Developmental Psychology*, 30, 679–689.

The Neuroscience of Well-Being: Part 2 (Potential Neural Networks)

Justine Megan Gatt

*Neuroscience Research Australia, Sydney, NSW, Australia
School of Psychology, University of New South Wales, Sydney, NSW, Australia*

Introduction

Part 1 of this chapter series summarized the various ways mental well-being has been conceptualized and measured to date, spanning the hedonic, eudaimonic, and composite well-being perspectives. As discussed, research to date has mostly focused on defining the best ways to measure well-being at the phenotypic level and its role in psychological health. In contrast, knowledge of the neuroscience mechanisms that underpin well-being is still relatively lacking. In the current chapter, the core neural networks of threat, reward, and executive control and their role in mental well-being will be discussed. Some evidence supporting these neural networks in mental well-being is reviewed, and future developments for this work are highlighted.

Potential Neural Networks of Mental Well-Being

Emotional and/or cognitive function are typically impaired in major mental disorders such as major depression and anxiety disorder (Williams, 2016a), and it is neural networks such as threat, reward, and executive control (both attention and cognitive control) that underscore some of the core processes of emotional and cognitive function (Williams, 2016b). These networks are therefore likely to be central to resilience to adversity (Berridge & Kringelbach, 2011; Feder, Nestler, & Charney, 2009), motivated goal-directed behavior (Ernst & Fudge, 2009), and mental well-being. Patterns of network activation are usually measured not only during the presentation of a specific task known to activate the regions of interest but also at rest in the absence of a task to measure “default mode” function (Greicius, Krasnow, Reiss, &

Menon, 2003). It is also sometimes used as an alternative indicator of neural activity because it minimizes the typical limits in methodology that are sometimes associated with task-activated paradigms (e.g., poor signal-to-noise ratio, practice effects, and habituation) (Greicius, 2008). Changes in these networks can be identified physiologically using tools such as functional magnetic resonance imaging (fMRI) and electroencephalography (EEG), each tapping into different spatial and temporal network properties, or behaviorally using neurocognitive performance tasks (Williams et al., 2008).

A breakdown in the structure and interactivity of one or more of these networks can lead to the onset of different psychiatric syndromes. For instance, patients with disorders like post-traumatic stress disorder and major depression have shown a cognitive bias toward stimuli that are negative and threatening over stimuli that are positive and rewarding (Williams, 2016a). Whereas patients with pleasure-seeking disorders such as dependent drug use and compulsive disorders have demonstrated a bias toward stimuli that are pleasurable, combined with a lack of inhibitory behavioral control (Berridge & Kringelbach, 2011; Feder et al., 2009). Changes in neural activation have also been found at rest in different psychiatric disorders including depression, anxiety disorders, and schizophrenia (Fox & Greicius, 2010; Williams, 2016a). It could therefore be hypothesized that individuals who are flourishing will demonstrate patterns of neural activation contrary to patients with mood or anxiety disorders. It could also be predicted that an optimal state of well-being would be associated with neural activity that is demonstrative of a bias in attention toward seeking reward over avoiding threat, in combination with superior executive control.

Threat

Humans and animals have an innate automatic survival instinct to detect threat in their environment. The experience of a potential threat triggers a cascade of neurophysiological changes commonly known as the “fight-or-flight” response. In the fight-or-flight state, the sensory cortex signals the amygdala (the emotional processing center), which then signals the hypothalamic–pituitary–adrenal (HPA) axis. These signals facilitate the response to stress. Following this feedforward response, reentrant feedback is then sent from the higher cortical areas, like the prefrontal cortex (PFC) and the hippocampus, to appraise the threat and then respond. The physiological response to the threat cue is determined by the interplay of the HPA with the autonomic nervous system (ANS) and its sympathetic and parasympathetic nervous system responses. The sympathetic nervous system triggers the fight-or-flight response, which includes the release of corticotropin-releasing hormone (CRH), adrenocorticotrophic hormone (ACTH), and cortisol. The consequent circulation of epinephrine through the bloodstream then triggers other physiological effects like an increase in heart rate, blood pressure, and blood glucose for the production of energy. Once the perceived threat is removed, the parasympathetic system is engaged via the vagus nerve of the nucleus ambiguus (which modulates heart rate oscillations, as measured by heart rate variability), thereby easing the effects of sympathetic activity and returning the body to a calm homeostatic state (Thayer & Lane, 2000).

Stimuli of emotional expressions are frequently used in laboratories to measure a response to threat, particularly a fearful face relative to a neutral or happy face (Williams, Palmer, Liddell, Song, & Gordon, 2006). In stress-related disorders, fear triggers an accentuated neural circuit for threat that involves key projections of the amygdala to the medial prefrontal cortex (MPFC) (Morris et al., 1996). Patients with lesions or damage to the amygdala show specific impairments in recognizing fearful facial expressions (Broks et al., 1998). Studies in clinical cohorts of anxiety disorder patients also show that fear, more than any other emotion, activates the

amygdala (Morris et al., 1996), particularly to masked (or nonconscious) rather than unmasked (or conscious) fear stimuli (P. J. Whalen et al., 1998) and also to other forms of stimuli like an unpleasant picture versus a neutral or pleasant one (Irwin et al., 1996). In animal lesion studies, the amygdala is only activated when new aversive learning is acquired but not in later stages of fear processing or behavioral expressions of anxiety (Kalin & Shelton, 2000).

Negative mood states also asymmetrically activate the PFC. Negative affect or anxiety, induced by film, increases activation in the right side of the prefrontal and anterior temporal regions (Davidson, Marshall, Tomarken, & Henriques, 2000), particularly in the right inferior PFC and right medial orbital prefrontal cortex (OFC) (Rauch, Savage, Alpert, Fischman, & Jenike, 1997). Positron emission tomography (PET) studies of glucose metabolism during evoked affective paradigms show right-sided increases in metabolic rate in anterior orbital, inferior frontal, middle, and superior frontal gyri during induced negative affect (Sutton et al., 1997). These patterns of asymmetry have been shown in clinical patients to phobic stimuli with increased activation in the right inferior PFC and right medial OFC (Rauch et al., 1997) and in patients with bilateral lesions of the ventromedial PFC (VMPFC) who found it difficult to anticipate future negative (or positive) consequences but not immediate punishment or reward (Bechara, Damasio, Damasio, & Anderson, 1994). Asymmetry may also assist an emotional response because individuals with greater left-sided prefrontal activity show greater recovery after the offset of a stimulus that is negative (Larson, Sutton, & Davidson, 1998).

Together, these findings highlight the role of the fight-or-flight response for basic survival and how it can be accentuated toward increased acute and/or chronic stress in patients with psychiatric disorders. Neuroimaging studies need to examine the same relationships in individuals who have varied levels of mental well-being. To better understand how the threat network functions in the flourishing well-being state, we need to investigate the changes in the structural regions of the brain, how those regions may function differently, and also how they may connect differently in interaction with each other. Future studies could also examine whether these differences are more focal to a specific region (e.g., sensory cortex alone) or whether it is the magnitude of activation to the stressor or threat response recovery time rather than overt activation that differentiates levels of well-being separate from levels of risk (Schuyler et al., 2014).

Reward

Reward, like food and social relationships, is also necessary for survival because it induces learning. Moreover, the prospect of reward also induces goal-directed behaviors to achieve the reward and experience subjective well-being and pleasure. The presence of reward encourages more frequent and intense behaviors, and the absence of reward can lead to behavioral extinction (Schultz, 2000). At the neural level, reward is processed by dopamine neurons of the ventral tegmental area (VTA) and the substantia nigra. The VTA projects to the nucleus accumbens (NAc) (of the basal ganglia) through to the amygdala and hippocampus. The VTA also projects to the dorsal and ventral PFC via the mesolimbic pathway, whereas the substantia nigra projects to the caudate and putamen via the nigrostriatal pathway (McClure, York, & Montague, 2004).

A number of neuroimaging studies have started to examine associations between well-being and the reward system. For instance, sustained engagement toward positive images in the striatum and right dorsolateral prefrontal cortex (DLPFC) is positively associated with mental well-being, whereas aggregate striatal activity over the scan session (how fMRI is normally coded) was not (Heller et al., 2013). Both psychological and subjective well-being independently

predict sustained striatal activity, but when combined into the same model, only psychological well-being predicted sustained activity. Individuals with greater sustained striatal activity also had lower daily cortisol output. These same patterns were also found for the DLPFC. Together, sustained activity in the striatum and DLPFC has been shown to account for 29% of the variance in well-being. The striatum helps to anticipate and process learning associated with reward and reinforcement (Haber & Knutson, 2010).

Many studies have also observed that positive mood states asymmetrically activate the PFC. For instance, left-sided electrical brain activity in the prefrontal and anterior temporal regions is increased by reward or positive effect induced by film across EEG and fMRI methods (Davidson, Jackson, & Kalin, 2000; Tomarken, Davidson, Wheeler, & Kinney, 1992). Reward also activates the left dorsolateral prefrontal (DLPFC) and medial orbitofrontal (OFC) regions when examined in EEG studies using source-localization LORETA techniques—specifically within the alpha 2 sub-band (10.5–12 Hz) (Pizzagalli, Sherwood, Henriques, & Davidson, 2005). Some studies indicate that activity in the OFC reflects the amount of reward, whereas other studies indicate that activity in the DLPFC shows the amount of reward and the consequent response in behavior (Wallis & Miller, 2003). Neural connectivity between these regions may channel behavior because OFC activation has been found to peak at 80 ms prior to DLPFC activation (Pizzagalli et al., 2005). Moreover, during the inducement of positive affect, metabolic increases in left-sided pre- and postcentral gyri have been confirmed by PET studies of glucose metabolism evoked during affective paradigms (Sutton et al., 1997). These compelling patterns of asymmetry have also been confirmed in association with self-report measures of psychological and subjective well-being in healthy older adults (Urry et al., 2004). It is predicted that these patterns of asymmetry reflect how the PFC is integral to the organization of emotional behavior (Levenson, 1994) and affective working memory (Watanabe, 1996). Even nonhuman primates with left-sided prefrontal activation show lower levels of cortisol and CRH levels over time, compared with animals with higher right-sided activation (Kalin, Shelton, & Davidson, 2000).

One possible mechanism for the effects on asymmetry is the inhibitory pathway of the MPFC to the amygdala, demonstrated in both rat extinction studies (Morgan, Romanski, & LeDoux, 1993) and human metabolic studies (Abercrombie et al., 1996). Subjects with increased left prefrontal activity may possess greater inhibitory control over the amygdala when exposed to different emotional stimuli (Davidson, Jackson, et al., 2000). However, some studies failed to produce these effects (Murphy, Nimmo-Smith, & Lawrence, 2003). It is possible however that the motivational context of the stimuli may impact the effects as positive stimuli with stronger emotive tendencies (e.g., pictures of desserts) show greater left frontal activation when compared with affective pictures, which did not always cause significant shifts in cortical activity (Harmon-Jones & Gable, 2009). Moreover, other studies suggest that the affective component is not central to determine activity in asymmetry but that it is how the subject is motivated that distinguishes this activity. “Approach motivation” (typically measured using stimuli that display or induce positive affect or anger) is associated with greater left frontal cortical activity, whereas “withdrawal motivation” (fear stimuli) is associated with greater right frontal activity (Harmon-Jones, Gable, & Peterson, 2010).

Within the limbic region, studies suggest associations between reward processing and activity within the amygdala and posterior cingulate cortex (PCC). In particular, the amygdala has been shown to respond to stimuli that are appetitive as well as stimuli that are threatening (Hamann, Ely, Hoffman, & Kilts, 2002). Asymmetrical patterns in the amygdala may affect responses to stimuli that are positive; increased activity in the right amygdala may be specific to stimuli that are unpleasant or to the removal of a reward (such as losing money), whereas

increased activity in the left amygdala occurs with the attainment of reward (winning money) (Zalla et al., 2000). Studies using event-related potentials (ERPs) also highlight the role of the PCC in positive affect. When individuals were shown pleasant and unpleasant pictures of varied intensity, individuals with high extraversion (associated with optimism and high subjective well-being) reacted more to pleasant pictures regardless of emotional intensity and were more resilient to mildly unpleasant pictures compared with individuals with low extraversion (Yuan et al., 2012). Source modeling suggests that this enhanced brain sensitivity to pleasant events and resistance to the impact of unpleasant events are generated by the bilateral PCC (Yuan et al., 2012), which, together with their collected regions (including fusiform gyrus, amygdala, and basal ganglia), are critical to regulate positive emotion (Amin, Constable, & Canli, 2004).

Physiological responses are also fundamental to the reward network, like the acceleration of the heart through the ANS, which is normally heightened with affect intensity but is not necessarily emotion specific (Watson, Wiese, Vaidya, & Tellegen, 1999). For instance, in some studies, increases in positive affect were associated with reduced ambulatory heart rate over the day (Steptoe, Wardle, & Marmot, 2005). In contrast, other studies reported increased cardiovascular function (higher heart rate and blood pressure) with increased positive affect (Schwartz, Warren, & Pickering, 1994), but this may be due to physical activation caused by interpersonal interactions or a greater engagement with challenge (Maier, Waldstein, & Synowski, 2003). Other studies have suggested an “undoing hypothesis”: that positive emotions may speed the recovery from cardiovascular reactivity caused by negative emotions, thereby returning the body to a normal level of activation (Fredrickson, Mancuso, Branigan, & Tugade, 2000). In these experimental studies, Fredrickson showed that participants who viewed clips of positive emotion following a clip of fear had the fastest cardiovascular recovery compared with those who were showed neutral or sad clips (Fredrickson & Levenson, 1998). Fredrickson and her colleagues interpreted this effect according to their *broaden-and-build theory*, which states that positive emotions broaden one’s “thought–action repertoire.” A thought–action repertoire describes acts that are triggered by specific thoughts or emotions. For example, joy creates the urge to play, whereas fear creates the urge to escape (Fredrickson, 2004). Therefore, positive emotions may help to move one from a thought–action repertoire that is narrow and triggered by emotions that are negative, quick, and direct and encourage escape, attack, or expel toward a thought–action repertoire that is broader and expands the scope of one’s tendencies to act. These broader thought–action repertoires could be used to explore, engage in novel experiences, promote self-growth, and expand mechanisms that can help one to cope with stress. Therefore, positive mindsets are thought to possess long-term benefits because they can develop personal resources that are adaptive and resilient (Fredrickson, 2004).

In summary, these findings highlight the importance of reward to survival because it encourages learning and induces behaviors that are goal directed to attain (or maintain) higher levels of well-being and pleasure. Evidence from neuroimaging and EEG studies has started to support the role of reward in well-being. This evidence is in stark contrast to the lack of studies that examine the relationship between well-being and threat. Evidence suggests several key regions in the reward network may be associated with increased flourishing. These include increases in striatal and DLPFC activity in response to reward, as well as asymmetric activation of the left PFC (in particular the DLPFC and OFC), both of which are associated with increased well-being. The relationship between well-being and patterns in asymmetric left PFC activation may reflect an increased inhibitory control over the amygdala when subjected to different emotions. If this is true, we may predict that individuals with elevated well-being

would also possess similar levels of inhibitory control over the amygdala when exposed to threat. The autonomic evidence similarly suggests that the impact of positive emotions on cardiovascular recovery times is modulated following threat exposure. Future neuroscience studies should explore associations between mental well-being and both reward and threat networks to assess their differences and how they may interact.

Executive Control

Executive control is a broad term used to outline the core functions of working memory, response inhibition, and set shifting (Friedman et al., 2008). Executive control, also called the central executive network (CEN), coordinates mental activity to achieve goals and to focus attention on tasks. In contrast, when the brain is at rest and not focused on a task (e.g., when relaxing on a beach), the mind-wandering or “default mode” network (DMN) is activated (Raichle et al., 2001). The circuits central to executive control include activity in the ventral and lateral prefrontal cortex (VLPFC and DLPFC), the inferior parietal cortex to tasks that are inhibitory or require sustained attention (e.g., the go/no-go or n-back working memory tasks), and reduce activation of the motor cortex to enable motor response control (Kelly et al., 2004).

The executive control network also processes and regulates emotion and often involves the same regions with overlapping functions. For instance, the DLPFC is used for attention (Corbetta, Patel, & Shulman, 2008) and working memory (Owen, McMillan, Laird, & Bullmore, 2005) and for regulating emotion (Kalisch, 2009). Various regions of the ACC are thought to respond differently to cues that involve cognition versus emotion. For example, the dorsal ACC is activated more during cognitive processing and top-down/bottom-up connections to the PFC (measured with the classical Stroop), whereas the ventral ACC and amygdala–hypothalamic circuits are activated more during emotional cognitive tasks (i.e., the emotional Stroop) (P. Whalen et al., 1998). The hippocampus regulates context, in particular memory that is declarative and context dependent (Davidson, 2000). Loss in the hippocampus is frequently reported in patients with anxiety or depressive disorder (Bremner et al., 2000), and the impact of this hippocampal loss is thought to affect contextual learning and memory where otherwise “normal” emotional responses are activated in inappropriate contexts. Another key region involved in higher-order regulation is the insula. The insula has been observed in functions involving higher-level cognitive control and attention, including a regulatory role in emotional and empathic processes (Menon & Uddin, 2010). The insula’s sensitivity to salient events (together with the ACC) suggests that these regions are a “neural switchboard” switching between the default and the executive control modes to access attention and memory and guide behavior (Menon & Uddin, 2010). In wellness studies, positive correlations have been reported between the right insula and purpose in life, positive relations, and personal growth in a cross-sectional cohort of healthy participants (Lewis, Kanai, Rees, & Bates, 2013). Other studies have also associated the right insula with self-awareness (Craig, 2009), regulation of bodily states (Singer, Critchley, & Preuschoff, 2009), and agentic control (Lee & Reeve, 2012). However, many of these studies are correlational. Therefore, to predict outcomes in well-being over time, future studies need to investigate how different regions activate and respond during tasks that involve emotional and executive control.

Studying how cognition interacts with emotion may also help us to understand the links between executive control, threat, and reward. Generally speaking, we may anticipate that people who are in a happy and relaxed mood would demonstrate better cognitive performance than someone who is angry or sad. Fredrickson’s (Fredrickson, 2001) broaden-and-build

theory suggests that positive emotions broaden an individual's thought–action repertoires and expand personal resources like cognitive functioning (e.g., executive control, knowledge) and psychological resources (e.g., resilience, creativity), which may lead to more enduring states of well-being. These newly expanded resources can then be drawn on later in times of coping or other cognitive challenges. The broaden hypothesis has been supported in studies that induce positive affect because positive emotions broaden the scope of attention and the size of the thought–action repertoire relative to a neutral state (Fredrickson & Branigan, 2005). Positive emotions are associated with cognitive processes that are more creative, flexible, and expansive (Murray, Sujan, Hirt, & Sujan, 1990) and increase the preference for varied behavioral responses (Kahn & Isen, 1993). Positive emotions also improve persuasive communication at different stages of memory processing: at encoding, participants in a positive mood simplify their message and are persuaded equally by both weak and strong arguments, and at retrieval, participants in a positive mood use more simplified processing strategies by drawing on global rather than detailed evaluations of the message to assess validity (Bless, Mackie, & Schwarz, 1992). Positive affect facilitates cognitive flexibility toward specific tasks such as word association (Isen, Johnson, Mertz, & Robinson, 1985), word fluency (Green & Noice, 1988), and specific problem solving (Ashby, Isen, & Turken, 1999). Usually, increased cognitive flexibility is only detected when the situation is neutral or positive in emotional content or is minimally engaging (Ashby et al., 1999). Furthermore, the presence of positive affect can help a person to cope with negative events because defensive behaviors are reduced (Aspinwall, 1998). Because these studies have used positive affect that is low in approach motivation, it is also important to consider the role of cues that trigger approach motivation such as reproduction, social attachment, and the ingestion of food and water. In contrast, breadth of attention is reduced in an individual with positive affect and high approach motivation because of their attempt to achieve the desired goal (Gable & Harmon-Jones, 2008).

Future Developments

In summary, this chapter has discussed how the three networks of threat, reward, and executive control may interact to influence well-being. Neuroimaging studies of well-being have started to emerge, most particularly for circuits of reward and for specific measures of subjective well-being. Few studies have considered the direct links between well-being and the circuits of threat or executive control. For threat, we predict that the response to stimuli that are negative would attenuate. Similarly for executive control, we predict that increased well-being would be associated with more superior executive control processes that include working memory, response inhibition, and set shifting. Furthermore, we predict that elevated executive control would also make an integrative impact on the processing of emotion for threat and reward. Future neuroscience studies need to examine both the structural morphology of different brain regions using different imaging techniques (e.g., MRI, DTI) and how well different regions of the brain and their connections function during both task-activated and resting paradigms. For a complete and composite view of both structure and function, a combination of functional imaging and electrophysiological techniques (i.e., fMRI and EEG) may also help to combine high spatial resolution with high temporal resolution.

In addition to more comprehensive neuroimaging assessments, future studies also need to incorporate phenotypic assessments of well-being that encompass both subjective and psychological aspects of well-being. The COMPAS-W well-being scale (Gatt, Burton, Schofield, Bryant, & Williams, 2014) is one such example that could be used in future studies as an

all-inclusive index of well-being. This same measure could also be used in studies of *resilience* whereby the trajectory of well-being scores and mental health over time could be tracked in individuals exposed to a childhood or adult trauma or adversity (e.g., the experience of neglect, abuse, war, natural disasters, personal illness or injury, and other major life events). Resilience defines the process of adaptive recovery from adversity and the ability to maintain optimal levels of well-being in the face of dynamic challenges to psychological resources (APA, 2010). Therefore, while mental well-being defines a state of positive mental health, resilience defines the process of returning to that state following adversity or trauma exposure. In this sense, we could predict that some of the same neural and biological mechanisms that underpin well-being may also contribute to resilience. This does not however necessarily implicate that someone who is flourishing at any given point in time would also be resilient should they be exposed to serious adversity; to demonstrate resilience would necessitate the presence of additional factors that buffer against the trauma (e.g., genetic, environmental, or other socio-ecological factors). The characterization of such predictive factors of resilience is still yet to be determined (see Alexander and Gatt, In Press for further discussion).

Many of the well-being (or resilience) studies to date are cross-sectional and/or correlational, which limits interpretation in terms of cause and effect. Longitudinal and experimental studies are required to better understand these underlying networks. For instance, causality could be more easily established in studies that consider whether interventions that improve executive function or other functions (albeit, via behavioral or cognitive training or neurofeedback strategies) also promote increased well-being and resilience. The first step however is to understand the neural mechanisms of well-being in order to develop strategies that optimize these features. Should evidence support the elementary networks of threat, reward, and executive control in well-being, then it may be arguable that interventions would need to promote adaptive responses to threat and perceived stress, a positive outlook, autonomy, higher-order cognitive capacities to master the regulation of emotion and behavior, and the ability to set goals and strive. When viewed as a whole, these strengths contribute to an overall sense of subjective and psychological well-being (Gatt et al., 2014). This framework also complements Fredrickson's "broaden-and-build" theory that similarly suggests that "...positive emotions broaden individuals' thought-action repertoires, enabling them to draw flexibly on higher-level connections and wide-than-usual ranges of precepts, ideas, and action urges; broadened cognition in turn creates behavioral flexibility that over time builds personal resources, such as mindfulness, resilience, social closeness, and even physical health" (p. 3) (Garland et al., 2010). When mapping the trajectory of mental health outcomes in response to such health promotion strategies, or simply over time, it is important to keep in mind the modulating influence of basic demographic variables such as age and sex. The outcome of such studies can then help shape the types of interventions that will promote well-being and resilience and the specific individuals to target, particularly in more vulnerable cohorts such as children and adolescents.

Conclusion

Understanding mental well-being is paramount to psychological and psychiatric research and mental health promotion in the general population. Much work is needed to understand the neurobiological mechanisms of well-being so that these essential qualities can be promoted in mental health programs in children and adults alike. This chapter has provided an outline of possible neural networks involved in well-being and some evidence to date, as well as possible future developments to aid progress in mental health and mental illness research, policy, and treatment.

Acknowledgments

Dr. Justine Gatt is currently supported by a NHMRC CDF Fellowship APP1062495.

Author Biography

Dr. Justine Megan Gatt is a group leader and senior research scientist at Neuroscience Research Australia and at the School of Psychology at the University of New South Wales, Australia. Dr. Gatt leads a research program focusing on the neuroscience and gene–environment mechanisms of resilience and mental well-being across adult and youth groups. This work incorporates various genetic, neuroimaging, and neurocognitive techniques and various methodological designs (twin/singleton studies, longitudinal, and intervention work), with the ultimate aim being to understand and promote optimal mental health in the general population.

References

- Abercrombie, H. C., Schaefer, S. M., Larson, C. L., Ward, R. T., Holden, J. F., Turski, P. A., ... Davidson, R. J. (1996). Medial prefrontal and amygdalar glucose metabolism in depressed and control subjects: An FDG-PET study. *Psychophysiology*, 33, S17.
- Alexander, R., & Gatt, J. M. (in press). Resilience. In A. C. Miu, J. R. Homberg, & K.-P. Lesch (Eds.), *Genes, brain and emotions: Interdisciplinary and Translational Perspectives* (pp. 286–303). Oxford: Oxford University Press. ISBN: 9780198793014.
- Amin, Z., Constable, R. T., & Canli, T. H. (2004). Attentional bias for valenced stimuli as a function of personality in the dot-probe task. *Journal of Research in Personality*, 38, 15–23.
- APA. (2010). The road to resilience. Retrieved from <http://www.apa.org/helpcenter/road-resilience.aspx>
- Ashby, F. G., Isen, A. M., & Turken, A. U. (1999). A neuropsychological theory of positive affect and its influence on cognition. *Psychological Review*, 106(3), 529–550.
- Aspinwall, L. G. (1998). Rethinking the role of positive affect in self-regulation. *Motivation and Emotion*, 22, 1–32.
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50, 7–15.
- Berridge, K. C., & Kringelbach, M. L. (2011). Building a neuroscience of pleasure and well-being. *Psychology of Well-Being: Theory, Research and Practice*, 1(3), 1–26.
- Bless, H., Mackie, D. M., & Schwarz, N. (1992). Mood effects on attitude judgments: Independent effects of mood before and after message elaboration. *Journal of Personality and Social Psychology*, 63, 585–595.
- Bremner, J. D., Narayan, M., Anderson, E. R., Staib, L. H., Miller, H. L., & Charney, D. S. (2000). Hippocampal volume reduction in major depression. *American Journal of Psychiatry*, 157, 115–118.
- Broks, P., Young, A. W., Maratos, E. J., Coffey, P. J., Calder, A. J., Isaac, C. L., ... Hadley, D. (1998). Face processing impairments after encephalitis: Amygdala damage and recognition of fear. *Neuropsychologia*, 36, 59–70.
- Corbetta, M., Patel, G., & Shulman, G. L. (2008). The reorienting system of the human brain: From environment to theory of mind. *Neuron*, 58, 306–324.
- Craig, A. D. (2009). How do you feel—now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, 10, 59–70.
- Davidson, R. J. (2000). Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *American Psychologist*, 55, 1214–1230.
- Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, 126, 890–909.

- Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: Regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry*, 47, 85–95.
- Ernst, M., & Fudge, J. L. (2009). A developmental neurobiological model of motivated behavior: Anatomy, connectivity and ontogeny of the triadic nodes. *Neuroscience & Biobehavioral Reviews*, 33(3), 367–382. doi:10.1016/j.neubiorev.2008.10.009
- Feder, A., Nestler, E. J., & Charney, D. S. (2009). Psychobiology and molecular genetics of resilience. *Nature Review Neuroscience*, 10(6), 446–457.
- Fox, M. D., & Greicius, M. (2010). Clinical applications of resting state functional connectivity. *Frontiers in Systems Neuroscience*, 4, 19.
- Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist*, 56, 218–226.
- Fredrickson, B. L. (2004). The broaden-and-build theory of positive emotions. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 359(1449), 1367–1378. doi:10.1098/rstb.2004.1512
- Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought-action repertoires. *Cognition and Emotion*, 19(3), 313–332.
- Fredrickson, B. L., & Levenson, R. W. (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition and Emotion*, 12, 191–220.
- Fredrickson, B. L., Mancuso, R. A., Branigan, C., & Tugade, M. M. (2000). The undoing effect of positive emotions. *Motivation and Emotion*, 24(4), 237–258.
- Friedman, N. P., Miyake, A., Young, S. E., Defries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual differences in executive functions are almost entirely genetic in origin. *Journal of Experimental Psychology: General*, 137(2), 201–225.
- Gable, P. A., & Harmon-Jones, E. (2008). Approach-motivated positive affect reduces breadth of attention. *Psychological Science*, 19, 476–482.
- Garland, E. L., Fredrickson, B., Kring, A. M., Johnson, D. P., Meyer, P. S., & Penn, D. L. (2010). Upward spirals of positive emotions counter downward spirals of negativity: Insights from the broaden-and-build theory and affective neuroscience on the treatment of emotion dysfunctions and deficits in psychopathology. *Clinical Psychology Review*, 30(7), 849–864.
- Gatt, J. M., Burton, K. L. O., Schofield, P. R., Bryant, R. A., & Williams, L. M. (2014). The heritability of mental health and wellbeing defined using COMPAS-W, a new composite measure of wellbeing. *Psychiatry Research*, 219, 204–213.
- Green, T. R., & Noice, H. (1988). Influence of positive affect upon creative thinking and problem solving in children. *Psychological Reports*, 63, 895–898.
- Greicius, M. (2008). Resting-state functional connectivity in neuropsychiatric disorders. *Current Opinion in Neurology*, 21, 424–430.
- Greicius, M., Krasnow, B., Reiss, A. L., & Menon, V. (2003). Functional connectivity in the resting brain: A network analysis of the default mode hypothesis. *Proceedings of the National Academy of Sciences of the United States of America*, 100, 253–258.
- Haber, S. N., & Knutson, B. (2010). The reward circuit: Linking primate anatomy and human imaging. *Neuropsychopharmacology*, 35, 4–26.
- Hamann, S. B., Ely, T. D., Hoffman, J. M., & Kilts, C. D. (2002). Ecstasy and agony: Activation of the human amygdala in positive and negative emotion. *Psychological Science*, 13, 135–141.
- Harmon-Jones, E., & Gable, P. A. (2009). Neural activity underlying the effect of approach-motivated positive affect on narrowed attention. *Psychological Science*, 20, 406–409.
- Harmon-Jones, E., Gable, P. A., & Peterson, C. K. (2010). The role of asymmetric frontal cortical activity in emotion-related phenomena: A review and update. *Biological Psychology*, 84, 451–462.
- Heller, A. S., van Reekum, C. M., Schaefer, S. M., Lapate, R. C., Radler, B. T., Ryff, C. D., & Davidson, R. J. (2013). Sustained striatal activity predicts eudaimonic well-being and cortisol output. *Psychological Science*, 24(11), 2191–2200.
- Irwin, W., Davidson, R. J., Lowe, M. J., Mock, B. J., Sorenson, J. A., & Turski, P. A. (1996). Human amygdala activation detected with echo-planar functional magnetic resonance imaging. *Neuroreport*, 7, 1765–1769.

- Isen, A. M., Johnson, M. S., Mertz, E., & Robinson, G. F. (1985). The influence of positive affect on the unusualness of word associations. *Journal of Personality and Social Psychology*, 48, 1413–1426.
- Kahn, B. E., & Isen, A. M. (1993). The influence of positive affect on variety-seeking among safe, enjoyable products. *Journal of Consumer Research*, 20, 257–270.
- Kalin, N. H., & Shelton, S. E. (2000). The regulation of defensive behaviors in Rhesus monkeys: Implications for understanding anxiety disorders. In R. J. Davidson (Ed.), *Anxiety, depression and emotion* (pp. 50–68). New York, NY: Oxford University Press.
- Kalin, N. H., Shelton, S. E., & Davidson, R. J. (2000). Cerebrospinal fluid corticotropin-releasing hormone levels are elevated in monkeys with patterns of brain activity associated with fearful temperament. *Biological Psychiatry*, 47, 579–585.
- Kalisch, R. (2009). The functional neuroanatomy of reappraisal: Time matters. *Neuroscience and Biobehavioral Reviews*, 33, 1215–1226.
- Kelly, A. M. C., Hester, R., Murphy, K., Javitt, D. C., Foxe, J. J., & Garavan, H. (2004). Prefrontal-subcortical dissociations underlying inhibitory control revealed by event-related fMRI. *European Journal of Neuroscience*, 19(11), 3105–3112. doi:10.1111/j.0953-816X.2004.03429.x
- Larson, C. L., Sutton, S. K., & Davidson, R. J. (1998). Affective style, frontal brain asymmetry and the time course of the emotion-modulated startle response. *Psychophysiology*, 35, S52.
- Lee, W., & Reeve, J. (2012). Self-determined, but not non-self-determined, motivation predicts activations in the anterior insular cortex: An fMRI study of personal agency. *Social Cognitive and Affective Neuroscience*, 8(5), 538–545. doi:10.1093/scan/nss029
- Levenson, R. W. (1994). Human emotion: A functional view. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 123–126). New York, NY: Oxford University Press.
- Lewis, G. J., Kanai, R., Rees, G., & Bates, T. C. (2013). Neural correlates of the ‘good life’: Eudaimonic well-being is associated with insular cortex volume. *Social Cognitive and Affective Neuroscience*. doi:10.1093/scan/nst032
- Maier, K. J., Waldstein, S. R., & Synowski, S. J. (2003). Relation of cognitive appraisal to cardiovascular reactivity, affect, and task engagement. *Annals of Behavioral Medicine*, 26, 32–41.
- McClure, S. M., York, M. K., & Montague, P. R. (2004). The neural substrates of reward processing in humans: the modern role of FMRI. *The Neuroscientist*, 10(3), 260–268. doi:10.1177/1073858404263526
- Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: a network model of insula function. *Brain Structure & Function*, 214(5-6), 655–667. doi:10.1007/s00429-010-0262-0
- Morgan, M. A., Romanski, L., & LeDoux, J. E. (1993). Extinction of emotional learning: Contribution of medial prefrontal cortex. *Neuroscience Letters*, 163, 109–113.
- Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., & Dolan, R. J. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, 381, 812–815.
- Murphy, F. C., Nimmo-Smith, I., & Lawrence, A. D. (2003). Functional neuroanatomy of emotion: A meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience*, 3, 207–233.
- Murray, N., Sujan, H., Hirt, E. R., & Sujan, M. (1990). The influence of mood on categorization: A cognitive flexibility interpretation. *Journal of Personality and Social Psychology*, 59(3), 411–425.
- Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping*, 25, 46–59.
- Pizzagalli, D. A., Sherwood, R. J., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness: A source-localization study. *Psychological Science*, 16, 805–813.
- Raichle, M. E., MacLeod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America*, 98(2), 676–682.
- Rauch, S. L., Savage, C. R., Alpert, N. M., Fischman, A. J., & Jenike, M. A. (1997). A study of three disorders using positron emission tomography and symptom provocation. *Biological Psychiatry*, 42, 446–452.
- Schultz, W. (2000). Multiple reward signals in the brain. *Nature Review Neuroscience*, 1, 199–207.

- Schuyler, B. S., Kral, T. R. A., Jacquart, J., Burghy, C. A., Weng, H. Y., Perlman, D. M., ... Davidson, R. J. (2014). Temporal dynamics of emotional responding: amygdala recovery predicts emotional traits. *Social Cognitive and Affective Neuroscience*, 9(2), 176–181. doi:10.1093/scan/nss131
- Schwartz, J. E., Warren, K., & Pickering, T. G. (1994). Mood, location and physical position as predictors of ambulatory blood pressure and heart rate: Application of a multi-level random effects model. *Annals of Behavioral Medicine*, 16, 210–220.
- Singer, T., Critchley, H. D., & Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in Cognitive Sciences*, 13, 334–340.
- Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *Proceedings of the National Academy of Sciences*, 102, 6508–6512.
- Sutton, S. K., Ward, R. T., Larson, C. L., Holden, J. E., Perlman, S. B., & Davidson, R. J. (1997). Asymmetry in prefrontal glucose metabolism during appetitive and aversive emotional states: An FDG-PET study. *Psychophysiology*, 34, S89.
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, 61(3), 201–216.
- Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Kinney, L. (1992). Psychometric properties of resting anterior EEG asymmetry: Temporal stability and internal consistency. *Psychophysiology*, 29, 576–592.
- Urry, H. L., Nitschke, J. B., Dolski, I., Jackson, D. C., Dalton, K. M., Mueller, C. J., ... Davidson, R. J. (2004). Making a life worth living: neural correlates of well-being. *Psychological Science*, 15(6), 367–372. doi:10.1111/j.0956-7976.2004.00686.x
- Wallis, J. D., & Miller, E. K. (2003). Neuronal activity in primate dorsolateral and orbital prefrontal cortex during performance of a reward preference task. *European Journal of Neuroscience*, 18, 2069–2081.
- Watanabe, M. (1996). Reward expectancy in primate prefrontal neurons. *Nature*, 382, 629–632.
- Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology*, 76, 820–838.
- Whalen, P., Bush, G., McNally, R. J., Wilhelm, S., McInerney, S. C., Jenike, M. A., & Rauch, S. L. (1998). The emotional counting stroop paradigm: A functional magnetic resonance imaging probe of the anterior cingulate affective division. *Biological Psychiatry*, 44, 1219–1228.
- Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *Journal of Neuroscience*, 18, 411–418.
- Williams, L. M. (2016a). Defining biotypes for depression and anxiety based on large-scale circuit dysfunction: A theoretical review of the evidence and future directions for clinical translation. *Depression and Anxiety*, 34(1), 9–24. doi:10.1002/da.22556
- Williams, L. M. (2016b). Precision psychiatry: A neural circuit taxonomy for depression and anxiety. *The Lancet Psychiatry*, 3(5), 472–480.
- Williams, L. M., Gatt, J. M., Hatch, A., Palmer, D. M., Nagy, M., Rennie, C., ... Paul, R. H. (2008). The integrate model of emotion, thinking and self regulation: An application to the “paradox of aging”. *Journal of Integrative Neuroscience*, 7(3), 367–404.
- Williams, L. M., Palmer, D., Liddell, B. J., Song, L., & Gordon, E. (2006). The ‘when’ and ‘where’ of perceiving signals of threat versus non-threat. *NeuroImage*, 31(1), 458–467.
- Yuan, J., Zhang, J., Zhou, X., Yang, J., Meng, X., Zhang, Q., & Li, H. (2012). Neural mechanisms underlying the higher levels of subjective well-being in extraverts: Pleasant bias and unpleasant resistance. *Cognitive, Affective, & Behavioral Neuroscience*, 12, 175–192.
- Zalla, T., Koechlin, E., Pietrini, P., Basso, G., Aquino, P., Sirigu, A., & Grafman, J. (2000). Differential amygdala responses to winning and losing: A functional magnetic resonance imaging study in humans. *European Journal of Neuroscience*, 12, 1764–1770.

Early Life Stress

Madeline B. Harms and Seth D. Pollak

Department of Psychology, University of Wisconsin-Madison, Madison, WI, USA

Introduction

Early life, defined here as postnatal life from birth through adolescence, is a developmental period during which individuals are especially sensitive to the biological effects of stress (Lupien, McEwen, Gunnar, & Heim, 2009; though prenatal stress also appears to exert profound influence on health outcomes in offspring, this topic is beyond the scope of this chapter). There is strong evidence that environmental input during early life can program physical, cognitive, and emotional traits to be optimally adaptive for survival and reproduction in that environment (Bock, Rether, Groger, Xie, & Braun, 2014; Nederhof & Schmidt, 2012). Severe early life stress, including famine or war, child abuse and/or neglect, poverty, or exposure to family conflict and violence, has been repeatedly associated with negative cognitive, emotional, and physical health outcomes (Brown, Susser, Lin, Neugebauer, & Gorman, 1995; Eriksson, Räikkönen, & Eriksson, 2014; Kaplan et al., 2001). These adverse experiences appear to lead to changes in the body and brain that can be adaptive in a harsh and traumatic environment. However, these adaptations can increase risk for developing a range of psychiatric disorders and social adjustment problems later in life (Heim & Nemeroff, 2001). The focus of this article is the mechanisms through which early life stress influences neural development and in turn behavioral and psychological outcomes.

Classifying Types of Early Life Stress

The term “stress” has been used to describe different concepts, including both environmental events and individuals’ physical and psychological responses to these events. Here, we define stress as activation of bodily and neural systems that respond to potential threats in the environment. We define a “stressor” as “any unpredictable or uncontrollable stimulus that could—potentially—pose a threat to the organism” (Levine & Ursin, 1991). In early childhood, many of the most salient stressors are those that disrupt the attachment bond between a child and

his/her caregiver(s). The establishment and maintenance of an emotional bond between an infant and its mother or parents is believed to “program” emotional and social development throughout the lifespan (Ainsworth, 1962; Bowlby & King, 2004). Adverse experiences that can disrupt this bond include separation from caregivers, neglect, parental psychopathology, and various forms of abuse.

Early life stress has traditionally been characterized in terms of allostatic load, the cost of repeated biological responses to chronic stress exposure (McEwen, 2012) or cumulative risk models, the combination of risks posed by aggregate exposure to multiple stressors (Evans, Li, & Whipple, 2013). These models consider an individual’s global exposure to multiple stressors in predicting risk for negative physical or psychological health outcomes. For example, a child who was abused and had an incarcerated parent would be at higher risk than a child who was abused but was not exposed to additional stressors. More recently, some researchers have advocated that subtypes of early life stressors should be categorized in terms of the presence of harmful input, such as caregiver abuse or exposure to violence, and inadequate input, such as caregiver neglect, separation from caregivers, or scarcity of resources (Humphreys & Zeanah, 2015). Subtypes of early life stressors have similarly been characterized in terms of threat or deprivation (McLaughlin & Sheridan, 2016; Pollak, Cicchetti, Homung, & Reed, 2000). In these models, a neglected child has been exposed to qualitatively different types of stressors than a child who has been abused, and we would expect divergent outcomes between the two. However, exposure to multiple subtypes of early life stressors is common (Vachon, Krueger, Rogosch, & Cicchetti, 2015). Although there is some preliminary evidence that these subtypes of stressors may be associated with different neural and behavioral outcomes (Dennison et al., 2017), this is a relatively new idea, and consequently current scientific understanding of these differential pathways is limited.

In terms of human development, more is known about effects of deprivation-related stressors than threat-related stressors. Many of the seminal human studies on neurobehavioral effects of early life stress have stemmed from the Bucharest Early Intervention Project (Zeanah et al., 2003), a randomized controlled trial conducted between 2000 and 2005 that compared foster care as an alternative with institutional care for young Romanian children. In this project, half of a group of 136 children were randomly assigned to high-quality foster care and the other half to remain in institutional care. Children in the intervention group were adopted into foster care between 6 and 31 months of age, and all children were seen for follow-up assessments several times throughout childhood. Findings thus far indicate that early institutionalization leads to profound deficits in many domains of functioning, including cognitive and socioemotional development, brain structure and function, and an elevated incidence of psychological impairment. Children randomized to foster care showed reliably better outcomes in each of these areas than those who remained in institutional care (Nelson et al., 2007). In addition, children who remained in institutional care showed blunted physiological responses to psychosocial stressors compared with children randomized to foster care and to community controls (McLaughlin et al., 2015). As a whole, findings from this project have been interpreted to suggest that the deprivation of a consistent attachment figure in infancy and early childhood is a significant stressor that impairs the optimal development of systems that promote physical and psychological health.

Other forms of early life deprivation, such as caregiver neglect, poverty, and food insecurity, also appear to adversely influence development. Caregiver neglect, defined as a failure to meet children’s basic physical needs with respect to clothing, hygiene, food, and/or safety (Leeb, Paulozzi, Melanson, Simon, & Arias, 2008), is one of the most common forms of child maltreatment; it is estimated that 20% of children worldwide experience neglect (World Health

Organization, 2002). Neglect can be similar to institutionalization, in that children who experience severe neglect lack a caregiver that reliably responds to their needs (Bruce, Gunnar, Pears, & Fisher, 2013). Often, these children are placed into foster care families. Also like children who were institutionalized, individuals with a documented history of neglect show dysregulated stress hormone levels (Fisher, Stoolmiller, Gunnar, & Burraston, 2007), as well as deficits in higher-level cognitive function. Poverty is another deprivation-related stressor that has been associated with adverse outcomes in terms of brain development (Brito & Noble, 2014), language development and higher-level cognition (Noble, McCandliss, & Farah, 2007), academic achievement (Hair, Hanson, Wolfe, & Pollak, 2015), and emotion regulation (Kim et al., 2013) by prospective longitudinal studies. While children who are neglected or institutionalized do not reliably have their emotional needs met from their caregiver, children exposed to poverty, especially poverty severe enough to result in food insecurity, may not reliably have their physical needs met. In sum, a child's exposure to uncontrollable, and potentially unpredictable, failure to receive necessary physical and/or emotional stimulation can be characterized as a significant stressor.

In contrast to these deprivation-related stressors, caregiver abuse represents a direct and acute threat to a child's well-being. Children in abusive families may experience physical harm and threat (Bick & Nelson, 2016; Pollak, 2015), which results in chronic stress. Children in these environments may also experience a nonnormative emotional learning environment. While children in abusive families may have their physical needs met, abusive caregivers are often inconsistent in their responses to a child's behavior—sometimes responding in normative ways to their children and other times becoming either extremely reactive or unresponsive to their children (Milner & Robertson, 1989). This type of inconsistency can make it challenging for children to learn environmental contingencies between their own behavior and their caregiver's reactions. In addition, abusive caregivers often provide ambiguous emotional signaling to their children, producing unclear facial and vocal expressions of emotion (Shackman et al., 2010). All these factors likely contribute to significant alterations in emotional, cognitive, and brain development that are well documented in abused individuals (Hanson et al., 2015; Harms, Shannon-Bowen, Hanson, & Pollak, 2017; Hart & Rubia, 2012), as well as their increased risk for developing psychiatric disorders (Heim & Nemeroff, 2001).

In addition to overt maltreatment, less severe and more common forms of early adversity such as disrupted parent-child relationships have also been linked to poorer mental and physical health in adulthood. For example, individuals who grow up in families characterized by high levels of conflict and aggression, relationships that are cold, unsupportive, or neglectful, and chaotic daily lives tend to show more behavioral problems in childhood and poorer health in adulthood (Carroll et al., 2013; Repetti, Robles, & Reynolds, 2011; Repetti, Taylor, & Seeman, 2002). Individuals exposed to these subtler forms of early life stress within the family show problems in emotion processing and social competence, disruptions in stress-responsive biological regulatory systems, and poor health behaviors such as substance abuse (Repetti et al., 2002). In sum, the term "early life stress" encompasses a range of stressors and levels of severity, which poses a challenge for research in this area.

Measurement of Early Life Stress

Another challenge for researchers in early life stress relates to measurement of early adversity. For ethical reasons, the Bucharest Early Intervention Project is the only study using random assignment involving humans that has been conducted to examine the effects of early life

stress. In the majority of studies, children who are exposed to early adversity within the family environment also tend to experience high levels of stress in other domains, such as school-related stress and tension within the neighborhood and community. Thus, it is difficult to isolate how specific sources of early life stress impact children's health and development. However, a number of experimental studies involving rodents provide insight into the specific effects of disrupted caregiver behaviors on offspring.

Most animal studies of stress have either focused on the effects of stress during adulthood or on how early stress influences later neurobehavioral patterns in adults (Malter Cohen et al., 2013). For the most part, rodent models of childhood stress have tended to utilize periods of maternal separation. This approach tends to induce intermittent, rather than chronic stress (i.e., pups are separated from the mother and then reunified). Recently, however, a reliable model of chronic fragmented maternal care, including maltreatment, has been established for rodents. In this model, insufficient bedding and nesting materials in the home cage causes mothers to neglect or even abuse their pups and, in turn, induces early life psychosocial stress in offspring (Baram et al., 2012; Molet, Maras, Avishai-Eliner, & Baram, 2014). In parallel with human studies of maltreatment, this model of early family adversity has been shown to contribute to cognitive and emotional dysfunctions, as well as disrupted brain development (Brunson et al., 2005) and disrupted biological stress responses (Gunn et al., 2013) in offspring.

However, animal models have limitations in terms of generalizing results to humans. First, certain types of early life stress that may be unique to humans—such as emotional abuse—cannot be simulated. Rodent models such as the limited bedding/nesting paradigm tend to induce maternal behavior that most closely resembles neglect, but rarely results in physical abuse and obviously cannot approximate emotional abuse. In addition, the fact that rodents are born in litters makes rearing environments fundamentally different between rodents and humans. Several studies have examined early life stress in nonhuman primates, who exhibit similar mother–infant interactions as humans (Nelson & Winslow, 2009). A subset of nonhuman primate mothers do physically abuse their infants, though abuse differs from human families in that it tends to be short lived, confined to an infant's first month of life, and does not co-occur with neglect (Sanchez, 2006). Thus, the specific types of maltreatment environments that human children experience cannot be fully replicated in animal models. Second, rodent and even nonhuman primate models unable to reproduce the rich repertoire of cognitive and emotional behaviors that occur in human development. Third, brain regions mature at different rates and trajectories, which differ across species (Avishai-Eliner, Brunson, Sandman, & Baram, 2002). These differences pose a challenge for researchers who intend to directly compare the age of a developing animal with a specific human age in terms of overall brain development, especially in rodent studies. Due to these limitations, research examining the sequelae of early life stress in humans remains necessary.

Studies involving human subjects typically take either a retrospective or prospective approach to measuring early adversity. Retrospective reports of childhood experiences can be obtained from adults and are therefore a convenient measure in many situations. Some commonly used retrospective measures are questionnaires such as the Childhood Trauma Questionnaire (CTQ) (Bernstein, Fink, Handelsman, & Foote, 1994) and the Adverse Childhood Events Scale (ACES) (Felitti et al., 1998), which ask participants if they experienced maltreatment or negative life events in childhood. Other studies use interview methods, such as the UCLA Life Stress Interview (Hammen, Marks, Mayol, & de Mayo, 1985), which assesses current chronic stress in addition to a retrospective report of childhood stress.

The validity of retrospective reporting of early life stress is debated. A potential confound with this approach is that adults' current levels of stress and/or mood may affect their subjective

feelings regarding their childhood (Colman et al., 2016): for example, current life difficulties may cause them to have a negative bias of their lives as children. In addition, retrospective reports are subject to omission errors (Widom & Morris, 1997), and studies report weak correlations between retrospective reports and prospective measures. For example, in a large longitudinal birth cohort study (Reuben et al., 2016), a prospective measure of adverse experiences that were documented during childhood was only moderately correlated with retrospective reports by adult participants. The accuracy of retrospective reporting may also depend on the types of events that are queried. For example, in this study, higher agreement between prospective and retrospective measurement was reported for objectively adverse events, such as the loss of a parent, than for events involving subjective interpretation, such as emotional abuse. Furthermore, personality traits (neuroticism and agreeableness) were associated with the extent to which retrospective and prospective measurements were congruent. Another important difference between prospective and retrospective measurement is that prospective measures are often objective (e.g., measured through Child Protective Services Records or other informant reports), while retrospective reports are inherently subjective. Indeed, Reuben et al. (2016) found that prospectively measured early life stress best predicted objectively measured health outcomes, while retrospective reports best predicted subjective health outcomes. These associations suggest the possibility for common method bias—i.e., individuals who feel worse about their health also have more negative views regarding their early life experiences. For all these reasons, it is recommended that researchers use prospective measures whenever possible.

Behavioral Sequelae of Early Life Stress

Despite measurement issues, findings from studies using different methods, or involving humans versus animals, show substantial overlap in terms of the neurobehavioral effects of early adversity, including impaired cognitive and emotional development (Lupien et al., 2009). For example, studies involving both rodents (Ivy et al., 2010) and humans (Hanson et al., 2017; Harms, Shannon-Bowen, et al., 2017) have found deficits in learning and memory, along with abnormal development of associated brain regions, following early life stress (Teicher, Tomoda, & Andersen, 2006). Examining emotional development, Malter Cohen et al. (2013) tested parallel models of early life stress in both rodents (limited nesting material) and humans (early institutionalization), reporting evidence that early adversity inhibited the ability to suppress attention toward potentially threatening information in favor of goal-directed action, indicating deficits in fear regulation in both species. These findings have been replicated by many other studies that examined humans or rodents separately (Heim & Nemeroff, 2001; Tottenham & Sheridan, 2010).

Early stress appears to alter threat perception, which can lead to increases in both anxiety and aggressive behavior. In Shackman, Shackman, and Pollak (2007), abused children showed increased attention to visual and auditory anger cues, as indexed by event-related potentials (ERPs) relative to non-abused controls. In addition, the amount of attention children allocated to these threatening emotional stimuli mediated the relationship between physical abuse and child-reported anxiety. In a later study (Shackman & Pollak, 2014), the same researchers showed that a history of maltreatment was also associated with higher levels of negative affect expression and aggression (measured by negative feedback directed toward a putative peer) during a laboratory task. Attention to threat (angry faces) again mediated the relationships between maltreatment and both negative affect and aggression. Thus, over-attending to threat

appears to be a key mechanism through which early stress becomes associated with emotional and behavioral problems.

Behavioral and neuroimaging studies also suggest abnormal reward processing, which integrates emotional and cognitive systems, in individuals exposed to early stress (Birn, Roeber, & Pollak, 2017; Dillon et al., 2009; Goff et al., 2013; Hanson, Hariri, & Williamson, 2015; Mehta et al., 2010; Pechtel & Pizzagalli, 2011; Weller & Fisher, 2013). For example, in an incentive-based learning task, abused children and adolescents did not respond to differences in reward probability, failing to increase their response speed as the chance of winning increased (Guyer et al., 2006). This finding suggests that the abused children were unable to acquire or effectively use information about differing reward probabilities. In addition, studies of risk-taking found reduced exploration behavior, which could have yielded larger rewards, in neglected (Loman et al., 2014) and abused adolescents (Sujan, Humphreys, Ray, & Lee, 2014) relative to control groups. Finally, Harms, Shannon-Bowen, et al. (2017) found deficits in both acquisition and reversal of stimulus–reward associations among adolescents who had been physically abused. Although this is a relatively new area of research, and requires replication in experimental animal research, this emerging evidence suggests that early life stress has profound impacts on reward processing.

Abnormal development in threat sensitivity, emotion regulation, and reward processing may contribute to the association between early adversity and increased risk for a range of psychiatric disorders and behavioral problems (Jaffee, 2017). Early life stress, particularly maltreatment, has been linked to elevated risk for developing major depression (Scott, Smith, & Ellis, 2010) and anxiety disorders (Cohen et al., 2006), including PTSD (Breslau et al., 2014), as well as increased levels of suicidal thoughts (Thornberry, Henry, Ireland, & Smith, 2010) and self-injurious behavior (Yates, Tracy, & Luthar, 2008). In addition to higher internalizing symptomatology, victims of maltreatment show increased externalizing problems in adulthood, including higher incidence of antisocial behavior (Johnson & Leff, 1999), and more frequent criminal activity and arrests (Maxfield & Widom, 1996; Thornberry, Henry, Ireland, & Smith, 2010).

Neurobiological Sequelae of Early Life Stress

Early life stress influences an individual's development at multiple levels, including behavior, neurobiology, and the epigenome. To appreciate these influences, it is necessary to understand how organisms respond biologically to stressors in the environment.

The Stress Response

In humans and animals, sophisticated biological systems have evolved to respond to potential threats in the environment. Here, we provide a brief overview of this stress response. An acute stressor activates the hypothalamic–pituitary–adrenal (HPA) axis, which induces the release of corticotropin-releasing hormone (CRH) and vasopressin (AVP) from the hypothalamus and adrenocorticotrophic hormone (ACTH) from the pituitary into the blood. This stimulates the release of glucocorticoid (cortisol in humans and corticosterone in rodents) from the adrenal cortex. The HPA axis follows a basal circadian activity rhythm and is regulated by a negative feedback mechanism moderated by corticosteroids that constrain the response of the axis after its activation. This negative feedback mechanism is complex, but briefly, corticosteroids bind to glucocorticoid receptors (GRs) and mineralocorticoid receptors (MRs) that are expressed

in most bodily tissues and act as transcription factors (Larsson et al., 2012; Sanchez, Arnt, Hyttel, & Moltzen, 1993). GR activation causes a negative feedback loop at several levels of the HPA axis that ends the stress response and the release of cortisol once a threat has been removed (for more information, see Provencal & Binder, 2015).

Through this negative feedback mechanism, the HPA system responds very effectively to acute, short-term environmental threats, which were typical throughout most of human evolution. However, the HPA axis is less well equipped to deal with chronic stress. In this context, the negative feedback mechanisms that regulate the stress response can break down, resulting in either hypersensitive or blunted HPA system reactivity. In animal models, early life stress resulting from maternal separation, or from the limited nesting model, tends to lead to hyperreactivity of the HPA axis in adulthood, including increased CRH signaling in response to a stressor and impaired GR-mediated negative feedback (van Bodegom, Homberg, & Henckens, 2017). However, in another model of early life stress, early social deprivation, rodent pups are temporarily separated from their mother and littermates and housed in a novel environment. This more severe stress can decrease stress-induced corticosterone levels in these animals are either blunted or unaffected relative to non-stressed controls (Sandi & Haller, 2015), suggesting reduced HPA sensitivity relative to non-stressed controls. Human findings similarly show variation in the effects of early adversity on HPA axis function. For example, exposure to interparental aggression, corporal punishment, or frequent emotional maternal withdrawal was found to increase baseline (i.e., non-stressed) cortisol levels (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009) and cortisol stress responses (Bugental et al., 2003). In contrast, there is also evidence institutionalization or maltreatment can result in HPA axis hyporeactivity. For example, a recent study showed that childhood maltreatment was associated with decreases in cortisol response during psychosocial stress in adulthood (Grimm et al., 2014). Similarly, studies involving human children exposed to severe neglect, abuse, orphanage/institutional care, or involvement with child protective services report lower basal levels of corticosteroids (Bernard, Zwerling, & Dozier, 2015; Bruce, Fisher, Pears, & Levine, 2009; Gunnar and Donzella, 2002). In general, the literature suggests that more severe early stress is more likely to lead to hypo- versus hypercortisolism.

Inconsistent findings of either increased or decreased stress responsiveness after early adversity might be explained by a trajectory of initial hyperactivation of the HPA axis that progresses to a state of sustained stress hyporeactivity when the stressors are severe and/or chronic (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Pryce, Bettschen, Nanz-Bahr, & Feldon, 2003). HPA hyporeactivity may serve as a compensatory adaptation that protects the organism from deleterious effects of chronic high levels of stress hormones. Initial evidence for this trajectory of HPA axis responsiveness resulting from chronic stress has been demonstrated in animal models (Saltzman et al., 2006; Sterlemann et al., 2008). However, more research is needed to investigate mediating and moderating factors that influence how stress affects development of the HPA system.

Early Life Stress and Epigenetics of the Stress Response

Epigenetic mechanisms—changes in gene expression that do not involve changes in the DNA sequence—appear to be important factors that mediate the extent to which early life stress leads to blunting or hypersensitivity of the HPA axis (Conradt, 2017). Although the specific epigenetic modifications that may contribute to differential HPA system trajectories following early stress are not fully understood, the field has made rapid progress in recent years in elucidating how some of these mechanisms operate. For example, we now know that decreases in

GR expression and activation tend to be associated with an increase in the stress response, and this can be explained by epigenetic mechanisms (Provencal & Binder, 2015). DNA methylation, which can function to switch genes between “on” or “off” positions, is one epigenetic mechanism used by cells to regulate gene expression. Stress that occurs during certain developmental sensitive periods may lead to long-term changes in the methylation of genes that regulate the stress response (Zannas, Wiechmann, Gassen, & Binder, 2016). One such gene that has generated recent interest is the FK506 binding protein 5 (FKBP5) gene, which regulates glucocorticoid sensitivity. Childhood maltreatment has been associated with long-term demethylation of this gene (Klengel et al., 2013), which contributes to increased expression of FKBP5 (Harms et al., 2017). Higher expression of FKBP5 appears disrupt the negative feedback mechanism that regulates the HPA axis, resulting in glucocorticoid resistance, higher cortisol levels, and prolonged recovery following exposure to stress (Tyrka, Ridout, & Parade, 2016; Zannas & Binder, 20143). Another important gene that regulates the HPA axis is the glucocorticoid receptor gene, *NR3C1*. This gene shows greater methylation in physically abused children relative to non-maltreated children (Romens, McDonald, Svaren, & Pollak, 2015). A recent study examined the entire human genome in girls exposed to varying levels of childhood stress, finding that girls with high levels of stress exposure showed differential methylation in 122 genes, as well as changes in expression in 12 genes (Papale, Seltzer, Madrid, Pollak, & Alisch, 2018). Several of these genes are known to be involved in the stress response systems, but others serve different regulatory functions throughout the body, suggesting system-wide changes in biological functioning as a result of stress. Thus, early stress in the form of maltreatment appears to induce multiple epigenetic changes in stress response system and beyond that have implications for individuals’ biological reactivity to subsequent stress.

Influences of Early Life Stress on Neural Circuitry

In addition to alterations in the stress response, epigenetic changes induced by early life stress appear to play a role in altering brain structure and function following early life stress (Labonte et al., 2012). According to the neurotoxicity hypothesis (Sapolsky, Krey, & McEwen, 1986), chronic glucocorticoid release (induced by extended stress exposure and potential epigenetic modifications that contribute to impaired GR negative feedback) facilitates neurodegenerative processes, resulting in structural changes in the volumes of various brain regions. The brain regions most consistently affected by early life stress include the hippocampus, amygdala, and prefrontal cortex. These regions coordinate learning and memory processes, emotional reactivity, and emotion regulation.

The limbic system, a set of brain regions immediately beneath the cerebral cortex, supports a variety of functions including emotion, motivation, and memory formation. Limbic regions, particularly the hippocampus (facilitating memory formation) and amygdala (facilitating emotional processing), are profoundly affected by early life stress. Studies of hippocampal development in stressed animals provided some of the earliest evidence for effects of chronic stress on the brain, showing decreased branching, reduced length, and atrophy of dendrites in hippocampal neurons among stressed relative to non-stressed rats (e.g., Cook & Wellman, 2004; McEwen, 1999). The limited bedding/nesting model has also been associated with a reduction of synapses and dendritic spines, as well as dendritic atrophy in the hippocampus (Brunson et al., 2005). In humans, structural findings regarding the effects of early life stress on limbic system development are more mixed and tend to involve both hippocampus and amygdala (or finding that early adversity is associated with changes in one region but not the

other). For example, some studies show larger amygdala volumes relative to control groups among previously institutionalized children who were adopted (Mehta et al., 2009; Tottenham, 2010), or smaller hippocampal volumes, with later adopted children showing larger abnormalities (Hodel et al., 2015). However, several studies show no differences in hippocampal (Mehta et al., 2009; Sheridan, Fox, & Zeanah, 2012; Tottenham, Henry, Ireland, & Smith, 2010) or amygdala volumes (Hodel et al., 2015) between previously institutionalized children and controls. Another study that employed detailed hand tracing of amygdala and hippocampus volumes showed that various forms of early life stress (physical abuse, early neglect, or low socioeconomic status) were associated with smaller amygdala and hippocampal volumes (Hanson et al., 2015). Divergent findings in humans could be due to a number of factors, including developmental timing of stress exposure, nonlinear brain development, different effects based on type of stressor, and methodological factors. However, the preponderance of evidence suggests that early stress alters trajectories of limbic brain development, which has implications for memory and emotion processing.

In addition to the limbic system, the prefrontal cortex and its connectivity with the limbic system are affected by early life stress. In animals, maternal separation is associated with long-term changes in prefrontal microstructure (Braun, Lange, Metzger, & Poeggel, 2000; Ovtcharoff & Braun, 2001; Poeggel et al., 2003) in rodents and with decreased medial prefrontal cortex volume in nonhuman primates (Lyons, Parker, Katz, & Schatzberg, 2009; Spinelli et al., 2009). Similarly, in humans, early maltreatment is associated with reduced gray matter volumes in medial prefrontal cortex and anterior cingulate gyrus (Dannowski et al., 2012; De Brito et al., 2013; Edmiston et al., 2011; Hanson et al., 2010) as well as decreased functional connectivity between medial prefrontal cortex and amygdala (Burghy et al., 2012). Furthermore, a higher number of self-reported adverse childhood experiences have been linked with a smaller volume in regions such as anterior cingulate cortex and caudate that interface with the limbic system and prefrontal cortex (Cohen et al., 2006).

Studies using diffusion tensor imaging (DTI) studies, a method for evaluating structural connectivity through white matter tracts, show that early life stress disrupts microstructural integrity in the brain. Previously institutionalized or neglected children and adolescents show reduced integrity of the uncinate fasciculus (Eluvathingal et al., 2006; Govindan, Behen, Helder, Makki, & Chugani, 2010; Hanson et al., 2013), a white matter tract connecting limbic and frontal lobe regions (Behen et al., 2009). Moreover, several studies have found reduced global white matter volume, disrupted prefrontal white matter organization, or reduced prefrontal cortical thickness (reflecting the width of the cortical gray matter and potential brain atrophy) in children exposed to early deprivation (Eluvathingal et al., 2006; Mehta et al., 2009; Sheridan et al., 2012; McLaughlin et al., 2014). A study that assessed exposure to adverse childhood experiences through self-report found reduced microstructural integrity of the corpus callosum, which connects the two brain hemispheres (Paul et al., 2008). In this study, all participants were free of psychiatric symptoms, suggesting that white matter structure can be impacted by stress even in the absence of psychopathology. Finally, there is evidence that early life stress disrupts the global organization of brain networks—young adults with a history of childhood maltreatment show decreased network centrality in a number of cortical areas relative to non-maltreated individuals (Harms et al., 2017) (Teicher, Anderson, Ohashi, & Polcari, 2014). Decreased network centrality is likely to impair high-level cognitive functions that rely on the integration of information from multiple regions. In sum, various forms of severe early stress appear to alter both fronto-limbic structure and connectivity. These consequences may impact high-level cognitive processes such as emotion regulation and goal-directed behavior.

Gene–environment interactions and neural circuitry

Effects of early stress on neural structure and function appear to vary based on genetic factors. Genes involved in regulating serotonin levels and brain-derived neurotrophic factor (BDNF), a moderator of synaptic plasticity, have been shown to moderate the effects of stress on neural circuitry. Some individuals in the population carry “risk alleles” of these genes that increase their neurobiological sensitivity to negative environmental events. In one study, early life stress combined with the HTR3A CC genotype group of the serotonin 3A receptor gene was associated with smaller gray matter volume in hippocampus and frontal cortex and higher negative affect, compared with the T carriers (Gatt, Williams, et al., 2010). In another study, carriers of both BDNF methionine and HTR3A CC risk alleles who were exposed to high levels of early life stress showed elevated emotion-elicited heart rate, as well as right frontal hyperactivation and right parietotemporal hypoactivation in EEG asymmetry, compared with non-risk allele carriers (Gatt, Nemeroff, et al., 2010). These findings reflect gene–environment interactions associated with early stress that may increase the likelihood of developing internalizing psychopathology among risk allele carriers.

Additional Considerations

Additional factors that appear to moderate the effects of early stress on physical and mental health outcomes include developmental timing of stress and sex differences. In addition, we outline promising research addressing prevention and intervention mechanisms that may promote resilience in the face of early adversity.

Developmental Timing of Stress

Evidence suggests that stressors exert different neurobehavioral effects depending on when they occur during an organism’s development, and early life stress is generally associated with longer-lasting impacts than stress experienced during adulthood (Lupien et al., 2009). However, in rodents, whose offspring are born relatively immature in terms of neuroendocrine development, there is strong evidence for a stress hyporesponsive period (SHRP) during the first two weeks of postnatal life, in which the HPA axis does not respond, or reacts only minimally, to environmental stressors (Levine, 1994). This period may have evolved to protect the developing brain from the effects of elevated glucocorticoids. There is controversy as to whether such a period exists in humans, though there is evidence that caregivers (when not absent or abusive) provide a strong buffering effect against stress in early childhood (Gunnar & Cheatham, 2003). It should also be noted that, because rodent pups are born less mature but mature at a faster rate than human infants, perinatal stress will impact different stages of development depending on which species is studied (Lupien et al., 2009).

In terms of brain development, stress exposure at different developmental periods is likely to have different impacts because regions mature at different rates (Andersen, 2003). For example, one study found that childhood sexual abuse experienced between 3 and 5 years of age was associated with reduced hippocampal volume, while abuse experienced in adolescence was associated with reduced gray matter volume in the frontal cortex (Andersen & Teicher, 2008). These findings are congruent with the developmental trajectories of these regions, with the hippocampus maturing in early childhood and the prefrontal cortex continuing to develop throughout adolescence (Lupien et al., 2009). Another study found that stress reported in late childhood (ages 8 and up) was associated with smaller anterior cingulate and insula volumes,

while stress reported in early childhood did not impact the volume of these regions (Baker et al., 2013). This finding may be due to changes in the emotional processing of stressful events across development.

Stress experienced during certain developmental periods can show incubation effects, where impacts do not become apparent until later ages: for example, having a mother with maternal depression has been associated with abnormal HPA activity and depressive symptoms in adolescence (Halligan, Hubert, Goodyer, & Murray, 2007). Indeed, a number of mental health problems associated with early life stress tend to surface for the first time in adolescence (Giedd, 2008). Studies show that adolescents are highly vulnerable to psychosocial stress, possibly due to a protracted glucocorticoid response to stress that persists into adulthood (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; McCormick & Matthews, 2007). For example, chronic variable stress in adolescent rats has been shown to induce long-term alterations in hippocampal structure, cognition, and HPA axis function (Isgor, Kabaj, Akil, & Watson, 2004). However, most scientific knowledge regarding the impacts of adolescent-specific stress is derived from animals, and more research is needed to determine the long-term impacts of adolescent stress in humans.

Sex Differences

A number of studies suggest that males and females respond differently to developmental stressors, with most evidence indicating that females are more vulnerable to early life stress. Females who experienced trauma, who experienced physical abuse, or whose mothers were depressed during infancy show higher rates of depression, anxiety, and PTSD compared with males (Baker and Shalhoub-Kevorkian, 1999; Macmillan et al., 2001; Pitzer et al., 2011). Sex differences following early life stress are also found in HPA system reactivity. For example, exposure to early trauma has been shown to be positively associated with cortisol response to CRH challenge in men but not in women (DeSantis et al., 2011). This finding suggests that women are more prone to a blunting of the HPA axis as a result of early stress. A recent study involving youth also found that in girls relative to boys, cortisol responses to stress were more highly correlated with genetic risk factors in genes that regulate the HPA axis, suggesting higher susceptibility to “risky” alleles in girls (Pagliaccio et al., 2014). There is also evidence that white matter structure is affected by early stress to a greater extent in females than in males (Paul et al., 2008). Animal models suggest that sex hormones play a role in the diverging neurobiological and behavioral responses to stress that occurs during adolescence between males and females (McCormick & Matthews, 2007). However, more research that specifically examines mechanisms for sex differences in the neurobiological sequelae of early stress is called for. Such research is especially important given that the risk for depression, for which early stress is often a contributing factor, is much higher in females beginning in adolescence (Kessler et al., 2003).

Prevention and Intervention Research

There are promising areas of research addressing prevention and intervention mechanisms to ameliorate negative effects of early life stress on health. Given that stress first affects the HPA system, the most effective prevention and intervention mechanisms should moderate long-term alterations in this system as a result of early stress (Bruce et al., 2013), and two intervention programs so far have been shown to do so. Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) is a preventive intervention that focuses on the use of consistent,

contingent parenting strategies. MTFC-P has been shown to reduce placement disruptions, increase secure attachment-related behaviors (Fisher, Burraston, & Pears, 2005; Fisher & Kim, 2007), and prevent blunting of diurnal cortisol slopes in foster children (Fisher et al., 2007). These positive effects among children may be related to a reduction in caregiver stress resulting from the intervention (Fisher & Stoolmiller, 2008). Another intervention, the Incredible Years Series, was designed to reduce externalizing behaviors by enhancing parenting practices and child social competence among families with preschool-aged children who have siblings convicted for criminal activity. This preventive intervention has been shown to decrease child aggressive behavior (Brotman et al., 2008) and prevent blunted cortisol responses to social challenge (Brotman et al., 2007). Among families with low parental warmth, there is evidence that this intervention reduces child aggression via increased cortisol response, suggesting that HPA axis function mediates the positive impact of this intervention on child behavior (O'Neal et al., 2010).

Throughout infancy, childhood, and adolescence, the body and brain are undergoing dramatic changes and are profoundly shaped by environmental cues. As discussed earlier, many of the deleterious effects of early life stress are associated with a mismatch between the early life environment and later environments that an individual experiences. For example, the adaptations a child develops as a result of institutional care (blunted HPA reactivity, early puberty, preservation of emotional brain systems at the expense of higher-level cognitive systems) may increase their ability to survive long enough to reproduce in a very harsh, deprived environment. Similarly, increased vigilance to threat in the context of physical abuse increases a child's likelihood of avoiding harm in that particular family environment. However, these adaptations can hinder optimal functioning in an adoptive family and resource-rich environment, in school, or at a job later in life, resulting in a higher risk for physical and mental health problems. Stress-exposed individuals tend to exhibit reduced cognitive flexibility, meaning that once a stress-exposed individual learns a specific behavioral strategy during childhood, she/he may persevere with this strategy even if it becomes maladaptive when the environment changes (Harms, Shannon Bowen, et al., 2017). This dilemma presents another potential area for intervention: for children whose adverse environments cannot be fully replaced, perhaps early intermittent exposure to "mismatching" environments may facilitate cognitive and behavioral flexibility in novel situations (Bock et al., 2014). Programs such as Head Start and Boys and Girls Clubs might show positive impacts in part through exposing children from impoverished backgrounds to novel environments. Given the systemic effects of early life stress on physical and mental health outlined above, more research addressing prevention and intervention mechanisms is a profound societal need.

Conclusion

In sum, early life stress takes many forms, but all types of severe early adversity studied thus far have potentially negative impacts on long-term health and development. Early childhood stress influences lifetime physical and psychological health through alterations in the stress response system, the epigenome, and the development of brain circuitry that facilitates cognitive and emotional process. Priorities for future research on early life stress should include an emphasis on prospective measures of stress, longitudinal designs, attention to developmental timing (especially studies that examine adolescent-specific stress), and a focus on prevention and intervention work to mitigate the negative health effects of severe early stress.

Author Biographies

Madeline B. Harms is a visiting assistant professor at Gustavus Adolphus College in St. Peter, MN. She earned her PhD from the Institute of Child Development at the University of Minnesota and then completed a postdoctoral fellowship through the Emotion Training Grant at the University of Wisconsin–Madison. Dr. Harms has published on topics including emotion recognition, executive function development, and the effects of maltreatment on children's learning ability and brain development.

Seth D. Pollak is the College of Letters and Science Distinguished Professor of Psychology and professor of Pediatrics, Anthropology, Neuroscience, and Public Affairs at the University of Wisconsin–Madison. He is also an investigator at the University's Waisman Center for Human Development. Pollak earned dual PhDs from the University of Rochester in brain and cognitive sciences and in child clinical psychology. Professor Pollak has published widely on topics including children's emotional development, the influences of social risk factors on children's brain and behavioral development, and the effects of early experience on children's learning and health. He is a recipient of the American Psychological Association's Distinguished Early Career Award and the Boyd-McCandless Award for Distinguished Contributions to Child Development. Dr. Pollak is a fellow of the American Association for the Advancement of Science and the Association for Psychological Science, as well as a member of the Society for Research in Child Development's Governing Council.

References

- Ainsworth, M. D. (1962). The effects of maternal deprivation: A review of findings and controversy in the context of research strategy. *Public Health Papers*, 14, 97–165.
- Andersen, S. L. (2003). Trajectories of brain development: Point of vulnerability or window of opportunity? *Neuroscience and Biobehavioral Reviews*, 27, 3–18.
- Anderson, S. L., & Teicher, M. H. (2008). Stress, sensitive periods, and maturational events in adolescent depression. *Trends in Neurosciences*, 31(4), 183–191. doi:10.1016/j.tins.2008.01.004
- Avishai-Eliner, S., Brunson, K. L., Sandman, C. A., & Baram, T. Z. (2002). Stressed-out, or in (utero)? *Trends in Neurosciences*, 25(10), 518–524.
- Baker, A., & Shalhoub-Kevorkian, N. (1999). Effects of political and military traumas on children: The Palestinian case. *Clinical Psychology Review*, 19(8), 935–950.
- Baker LM, Williams LM, Korgaonkar MS, Cohen RA, Heaps JM, & Paul RH. (2013). Impact of early vs. late childhood early life stress on brain morphometrics. *Brain Imaging Behaviour*, 7(2), 196–203.
- Baram, T. Z., Davis, E. P., Obenaus, A., Sandman, C. A., Small, S. L., Solodkin, A., & Stern, H. (2012). Fragmentation and unpredictability of early-life experience in mental disorders. *American Journal of Psychiatry*, 169, 907–915. doi:10.1176/appi.ajp.2012.11091347
- Behen, M. E., Muzik, O., Saporta, A. S. D., Wilson, B. J., Pai, D., Hua, J., & Chugani, H. T. (2009). Abnormal fronto-striatal connectivity in children with histories of early deprivation: A diffusion tensor imaging study. *Brain Imaging and Behavior*, 3, 292–297. <https://doi.org/10.1007/s11682-009-9071-6>
- Bernard, K., Zwerling, J., & Dozier, M. (2015). Effects of early adversity on young children's diurnal cortisol rhythms and externalizing behavior. *Developmental Psychobiology*, 57, 935–947. doi:10.1002/dev.21324
- Bernstein, D. P., Fink, L., Handelsman, L., & Foote, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry*, 151(8), 1132–1136.

- Bick, J., & Nelson, C. A. (2016). Early adverse experiences and the developing brain. *Neuropsychopharmacology*, 41(1), 177–196. doi:10.1038/npp.2015.252
- Birn, R. M., Roeber, B. J., & Pollak, S. D. (2017). Early childhood stress exposure, reward pathways, and adult decision making. *PNAS*, 114(51), 13549–13554. <https://doi.org/10.1073/pnas.1708791114>
- Bock, J., Rether, K., Groger, N., Xie, L., & Braun, K. (2014). Perinatal programming of emotional brain circuits: An integrative view from systems to molecules. *Frontiers in Neuroscience*, 8, 11. doi:10.3389/fnins.2014.00011 eCollection 2014
- Bowlby, J., & King, P. (2004). *Fifty years of attachment theory: Recollections of Donald Winnicott and John Bowlby*. London: Karnac Books.
- Braun, K., Lange, E., Metzger, M., & Poeggel, G. (2000). Maternal separation followed by early social deprivation affects the development of monoaminergic fiber systems in the medial prefrontal cortex of Octodon degus. *Neuroscience*, 95(1), 309–318.
- Breslau, N., Koenen, K. C., Luo, Z., Agnew-Blais, J., Swanson, S., & Houts, R. M. (2014). Childhood maltreatment, juvenile disorders and adult post-traumatic stress disorder: A prospective investigation. *Psychological Medicine*, 44(9), 1937–1945.
- Brito, N. H., & Noble, K. G. (2014). Socioeconomic status and structural brain development. *Frontiers in Neuroscience*, 8, 276. doi:10.3389/fnins.2014.00276
- Brotman, L. M., Gouley, K. K., Huang, K. Y., Kamboukos, D., Fratto, C., & Pine, D. S. (2007). Effects of a psychosocial family-based preventive intervention on cortisol response to a social challenge in preschoolers at high risk for antisocial behavior. *Archives of General Psychiatry*, 64, 1172–1179.
- Brotman, L. M., Gouley, K. K., Huang, K. Y., Rosenfelt, A., O'Neal, C., Klein, R. G., & Shrout, P. (2008). Preventive intervention for preschoolers at high risk for antisocial behavior: Long-term effects on child physical aggression and parenting practices. *Journal of Clinical Child and Adolescent Psychology*, 37, 386–396.
- Brown, A. S., Susser, E. S., Lin, S. P., Neugebauer, R., & Gorman, J. M. (1995). Increased risk of affective disorders in males after second trimester prenatal exposure to the Dutch hunger winter of 1944–45. *The British Journal of Psychiatry*, 166, 601–606.
- Bruce, J., Fisher, P. A., Pears, K. C., & Levine, S. (2009). Morning cortisol Levels in preschool-aged foster children: Differential effects of maltreatment type. *Developmental Psychobiology*, 51, 14–23. doi:10.1002/dev.20333
- Bruce, J., Gunnar, M. R., Pears, K. C., & Fisher, P. A. (2013). Early adverse care, stress neurobiology, and prevention science: Lessons learned. *Prevention Science*, 14(3), 247–256.
- Brunson, K. L., Kramar, E., Lin, B., Chen, Y., Colgin, L. L., Yanagihara, T. K., ... Baram, T. Z. (2005). Mechanisms of late-onset cognitive decline after early-life stress. *The Journal of Neuroscience*, 25, 9328–9338. doi:10.1523/JNEUROSCI.2281-05.2005
- Bugental, D. B., Martorell, G. A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior*, 43(1), 237–244.
- Burghy, C. A., Stodola, D. E., Ruttle, P. L., Molloy, E. K., Armstrong, J. M., Oler, J. A., ... Birn, R. M. (2012). Developmental pathways to amygdala-prefrontal function and internalizing symptoms in adolescence. *Nature Neuroscience*, 15(12), 1736–1741.
- Carroll, J. E., Gruenewald, T. L., Taylor, S. E., Janicki-Deverts, D., Matthews, K. A., & Seeman, T. E. (2013). Childhood abuse, parental warmth, and adult multisystem biological risk in the Coronary Artery Risk Development in Young Adults study. *Proceedings of the National Academy of Sciences of the United States of America*, 110(42), 17149–17153. doi:10.1073/pnas.1315458110
- Cohen, R. A., Grieve, S., Hoth, K. F., Paul, R. H., Sweet, L., Tate, D., ... Williams, L. M. (2006). Early life stress and morphometry of the adult anterior cingulate cortex and caudate nuclei. *Biological Psychiatry*, 59(10), 975–982.
- Colman, I., Kingsbury, M., Garad, Y., Zeng, Y., Naicker, K., ... Thompson, A. H. (2016). Consistency in adult reporting of adverse childhood experiences. *Psychological Medicine*, 46(3), 543–549. <https://doi.org/10.1017/S0033291715002032>
- Conradt, E. (2017). Using principles of behavioral epigenetics to advance research on early-life stress. *Child Development Perspectives*, 11, 107–112. doi:10.1111/cdep.12219

- Cook, S. C., & Wellman, C. L. (2004). Chronic stress alters dendritic morphology in rat medial prefrontal cortex. *Journal of Neurobiology*, 60(2), 236–248.
- Dannlowski, U., Stuhrmann, A., Beutelmann, V., Zwanzger, P., Lenzen, T., Grotegard, D., ... Kugel, H. (2012). Limbic scars: long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biological Psychiatry*, 71(4), 286–293.
- Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., Manning, L. G., & Zale, E. (2009). Children's patterns of emotional reactivity to conflict as explanatory mechanisms in links between interpartner aggression and child physiological functioning. *Journal of Child Psychology and Psychiatry*, 50, 1384–1391. doi:10.1111/j.1469-7610.2009.02154.x
- De Brito, S. A., Viding, E., Sebastian, C. L., Kelly, P. A., Mechelli, A., Maris, H., & McCrory, E. J. (2013). Reduced orbitofrontal and temporal grey matter in a community sample of maltreated children. *Journal of Child Psychology and Psychiatry*, 54(1), 105–112. doi:10.1111/j.1469-7610.2012.02597.x
- Dennison, M. J., Rosen, M. L., Sambrook, K. A., Jenness, J. L., Sheridan, M. A., & McLaughlin, K. A. (2017). Differential associations of distinct forms of childhood adversity with neurobehavioral measures of reward processing: A developmental pathway to depression. *Child Development*.
- DeSantis, S. M., Baker, N. L., Back, S. E., Spratt, E., Ciolino, J. D., Moran-Santa Maria, M., ... Brady, K. T. (2011). Gender differences in the effect of early life trauma on hypothalamic-pituitary-adrenal axis functioning. *Depression and Anxiety*, 28(5), 383–392.
- Dillon, D. G., Holmes, A. J., Birk, J. L., Brooks, N., Lyons-Ruth, K., & Pizzagalli, D. A. (2009). Childhood adversity is associated with left basal ganglia dysfunction during reward anticipation in adulthood. *Biological Psychiatry*, 66, 206–213.
- Edmiston, E. E., Wang, F., Mazure, C. M., Guiney, J., Sinha, R., Mayes, L. C., & Blumberg, H. P. (2011). Corticostriatal-limbic gray matter morphology in adolescents with self-reported exposure to childhood maltreatment. *Archives of Pediatrics & Adolescent Medicine*, 165(12), 1069–1077. doi:10.1001/archpediatrics.2011.565
- Eluvathingal, T. J., Chugani, H. T., Behen, M. E., Csaba, J., Muzik, O., Maqbook, M., ... Makki, M. (2006). Abnormal brain connectivity in children after early severe socioemotional deprivation: A diffusion tensor imaging study. *Pediatrics*, 117(6), 2093–2100. doi:10.1542/peds.2005-1727
- Eriksson, M., Räikkönen, K., & Eriksson, J. G. (2014). Early life stress and later health outcomes—Findings from the Helsinki Birth Cohort Study. *American Journal of Human Biology*, 26, 111–116.
- Evans, G. W., Li, D., & Whipple, S. S. (2013). Cumulative risk and child development. *Psychological Bulletin*, 139, 1342–1396.
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... Marks, J. S. (1998). Household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245–258.
- Fisher, P. A., Burraston, B., & Pears, K. C. (2005). The Early Intervention Foster Care Program: Permanent placement outcomes from a randomized trial. *Child Maltreatment*, 10, 61–71.
- Fisher, P. A., & Kim, H. K. (2007). Intervention effects on foster preschoolers' attachment-related behaviors from a randomized trial. *Prevention Science*, 8, 161–170.
- Fisher, P. A., & Stoolmiller, M. (2008). Intervention effects on foster parent stress: Associations with child cortisol levels. *Development and Psychopathology*, 20, 1003–1021.
- Fisher, P. A., Stoolmiller, M., Gunnar, M. R., & Burraston, B. O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology*, 32(8–10), 892–905.
- Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, 30, 1010–1016. doi:10.1016/j.psyneuen.2005.04.006
- Gatt, J. M., Nemeroff, C. B., Schofield, P. R., Paul, R. H., Clark, C. R., Gordon, E., & Williams, L. M. (2010b). Early life stress combined with serotonin 3A receptor and brain-derived neurotrophic factor valine 66 to methionine genotypes impacts emotional brain and arousal correlates of risk for depression. *Biological Psychiatry*, 68(9), 818–824.

- Gatt, J. M., Williams, L. M., Schofield, P. R., Dobson-Stone, C., Paul, R. H., Grieve, S. M., ... Nemeroff, C. B. (2010). Impact of the HTR3A gene with early life trauma on emotional brain networks and depressed mood. *Depression and Anxiety*, 27(8), 752–759. doi:10.1002/da.20726
- Giedd, J. N. (2008). The teen brain: insights from neuroimaging. *Journal of Adolescent Health*, 42(4), 335–343.
- Goff, B., Gee, D. G., Telzer, E. H., Humphreys, K. L., Gabard-Durnam, L., Flannery, J., & Tottenham, N. (2013). Reduced nucleus accumbens reactivity and adolescent depression Following early-life stress. *Journal of Neuroscience*, 249, 129–138.
- Govindan, R. M., Behen, M. E., Helder, E., Makki, M. I., & Chugani, H. T. (2010). Altered water diffusivity in cortical association tracts in children with early deprivation identified with Tract-Based Spatial Statistics (TBSS). *Cerebral Cortex*, 20(3), 561–569.
- Grimm, S., Pestke, K., Feeser, M., Aust, S., Weigand, A., Wang, J., ... Bajbouj, M. (2014). Early life stress modulates oxytocin effects on limbic system during acute psychosocial stress. *Social Cognitive and Affective Neuroscience*, 9, 1828–1835. doi:10.1093/scan/nsu020
- Gunn, B. G., Cunningham, L., Cooper, M. A., Corteen, N. L., Seifi, M., Swinny, J. D., ... Belelli, D. (2013). Dysfunctional astrocytic and synaptic regulation of hypothalamic glutamatergic transmission in a mouse model of early-life adversity: Relevance to neurosteroids and programming of the stress response. *The Journal of Neuroscience*, 33(50), 19534–19554.
- Gunnar, M. R., & Cheatham, C. L. (2003). Brain and behavior interfaces: stress and the developing brain. *Infant Mental Health Journal*, 24, 195–211.
- Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, 27, 199–220. doi:10.1016/S0306-4530(01)00045-2
- Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009). Developmental changes in hypothalamus-pituitary-adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology*, 21, 69–85.
- Guyer, A. E., Kaufman, J., Hodgdon, H. B., Masten, C. L., Jazbec, S., Pine, D. S., & Ernst, M. (2006). Behavioral alterations in reward system function: The role of childhood maltreatment and psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry*, 45(9), 1059–1067.
- Hair, N. L., Hanson, J. L., Wolfe, B. L., & Pollak, S. D. (2015). Association of child poverty, brain development, and academic achievement. *JAMA Pediatrics*, 169(9), 822–829. doi:10.1001/jamapediatrics.2015.1475
- Halligan, S. L., Herbert, J., Goodyer, I., & Murray, L. (2007). Disturbances in morning cortisol secretion in association with maternal postnatal depression predict subsequent depressive symptomatology in adolescents. *Biological Psychiatry*, 62, 40–46.
- Hammen, C., Marks, T., Mayol, A., & de Mayo, R. (1985). Depressive self-schemas, life stress, and vulnerability to depression. *Journal of Abnormal Psychology*, 94, 308–319. doi:10.1037/0021-843X.94.3.308
- Hanson, J. L., Adluru, N., Chung, M. K., Alexander, A. L., Davidson, R. J., & Pollak, S. D. (2013). Early neglect is associated with alterations in white matter integrity and cognitive functioning. *Child Development*, 84(5), 1566–1578. <http://doi.org/10.1111/cdev.12069>
- Hanson, J. L., Bos, W., Roeber, B. J., Rudolph, K. D., Davidson, R. J., & Pollak, S. D. (2017). Early adversity and learning: Implications for typical and atypical behavioral development. *Journal of Child Psychology and Psychiatry*, 58(7), 770–778. doi:10.1111/jcpp.12694
- Hanson, J. L., Chung, M. K., Avants, B. B., Shirtcliff, E. A., Gee, J. C., Davidson, R. J., & Pollak, S. D. (2010). Early stress is associated with alterations in the orbitofrontal cortex: A tensor-based morphometry investigation of brain structure and behavioral risk. *The Journal of Neuroscience*, 30(22), 7466–7472. <http://doi.org/10.1523/JNEUROSCI.0859-10.2010>
- Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., Shirtcliff, E. A., Pollak, S. D., & Davidson, R. J. (2015). Behavioral problems after early life stress: contributions of the hippocampus and amygdala. *Biological psychiatry*, 77(4), 314–323. <https://doi.org/10.1016/j.biopsych.2014.04.020>
- Harms, M. B., Birn, R., Provencal, N., Wiechmann, T., Binder, E. B., Giakas, S. W., Roeber, B. J., & Pollak, S. D. (2017). Early life stress, FK506 binding protein 5 gene (FKBP5) methylation, and

- inhibition-related prefrontal function: A prospective longitudinal study. *Development and psychopathology*, 29(5), 1895–1903.
- Harms, M. B., Shannon-Bowen, K., Hanson, J. D., & Pollak, S. D. (2017). Instrumental learning and cognitive flexibility are impaired in children exposed to early life stress. *Developmental Science*, 21(4), e12596. doi:10.1111/desc.12596
- Hart, H., & Rubia, K. (2012). Neuroimaging of child abuse: a critical review. *Frontiers in Human Neuroscience*, 6(52). doi:10.3389/fnhum.2012.00052 eCollection 2012
- Heim, C., & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Biological Psychiatry*, 49, 1023–1039.
- Hodel, A. S., Hunt, R. H., Cowell, R. A., van den Heuvel, S. E., Gunnar, M. E., & Thomas, K. M. (2015). Duration of early adversity and structural brain development in post-institutionalized adolescents. *NeuroImage*, 105, 112–119.
- Humphreys, K. L., & Zeanah, C. H. (2015). Deviations from the expectable environment in early childhood and emerging psychopathology. *Neuropsychopharmacology Reviews*, 40, 154–170. doi:10.1038/npp.2014.165
- Isgor, C., Kabbaj, M., Akil, H., & Watson, S. J. (2004). Delayed effects of chronic variable stress during peripubertal-juvenile period on hippocampal morphology and on cognitive and stress axis functions in rats. *Hippocampus*, 14, 636–648.
- Ivy, A. S., Rex, C. S., Chen, Y., Dube, C., Maras, P. M., Grigoriadis, D. E., ... Baram, T. Z. (2010). Hippocampal dysfunction and cognitive impairments provoked by chronic early-life stress involve excessive activation of CRH receptors. *Journal of Neuroscience*, 30(39), 13005–13015. https://doi.org/10.1523/JNEUROSCI.1784-10.2010
- Jaffee, S. R. (2017). Child maltreatment and risk for psychopathology in childhood and adulthood. *Annual Review of Clinical Psychology*, 13, 525–551. https://doi.org/10.1146/annurev-clinpsy-032816-045005
- Johnson, J. L., & Leff, M. (1999). Children of substance abusers: Overview of research findings. *Pediatrics*, 103(2), 1085–1099.
- Kaplan, G. A., Turrell, G., Lynch, J. W., Everson, S. A., Helkala, E. L., & Salonen, J. T. (2001). Childhood socioeconomic position and cognitive function in adulthood. *International Journal of Epidemiology*, 30, 256–263.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., ... National Comorbidity Survey Replication. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *JAMA*, 289, 3095–3105.
- Kim, P., Evans, G. W., Angstadt, M., Shaun Ho, S., Sripada, C. S., Swain, J. E., ... Phan, K. L. (2013). Effects of childhood poverty and chronic stress on emotion regulatory brain function in adulthood. *PNAS*, 110(46), 18442–18447. https://doi.org/10.1073/pnas.1308240110
- Klengel, T., Mehta, D., Anacker, C., Rex-Haffner, M., Pruessner, J. C., Pariante, C. M., ... Binder, E. B. (2013). Allele-specific *FKBP5* DNA demethylation mediates gene–childhood trauma interactions. *Nature Neuroscience*, 16, 33–41. https://doi.org/10.1038/nn.3275
- Labonté, B., Suderman, M., Maussion, G., Navaro, L., Yerko, V., Mahar, I., ... Turecki, G. (2012). Genome-wide epigenetic regulation by early-life trauma. *Archives of General Psychiatry*, 69(7), 722–731. doi:10.1001/archgenpsychiatry.2011.2287
- Larsson, L., Thorbert-Mros, S., Rymo, L., & Berglundh, T. (2012). Influence of epigenetic modifications of the interleukin-10 promoter on IL10 gene expression. *European Journal of Oral Sciences*, 120, 14–20.
- Leeb, R. T., Paulozzi, L., Melanson, C., Simon, T., & Arias, I. (2008). *Child Maltreatment Surveillance: Uniform Definitions for Public Health and Recommended Data Elements, Version 1.0*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Levine, S. (1994). The ontogeny of the hypothalamic-pituitary-adrenal axis. The influence of maternal factors. *Annals of the New York Academy of Sciences*, 746, 275–288.
- Levine, S., & Ursin, H. (1991). What is stress? In M. R. Brown, G. F. Koob, & C. Rivier (Eds.), *Stress: Neurobiology and neuroendocrinology* (Vol. 1991, pp. 3–21). New York: Marcel Dekker.

- Loman, M. M., Johnson, A. E., Quevedo, K., Lafavor, T. L., & Gunnar, M. R. (2014). Risk-taking and sensation-seeking propensity in postinstitutionalized early adolescents. *Journal of Child Psychology and Psychiatry*, 55, 1145–1152.
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, 10, 434–445. doi:10.1038/nrn2639
- Lyons, D. M., Afarian, H., Schatzberg, A. F., Sawyer-Glover, A., & Moseley, M. E. (2002). Experience-dependent asymmetric variation in primate prefrontal morphology. *Behavioural brain research*, 136(1), 51–59. [https://doi.org/10.1016/s0166-4328\(02\)00100-6](https://doi.org/10.1016/s0166-4328(02)00100-6)
- MacMillan, H. L., Fleming, J. E., Streiner, D. L., Lin, E., Boyle, M. H., Jamieson, E., ... Beardslee, W. R. (2001). Childhood abuse and lifetime psychopathology in a community sample. *The American Journal of Psychiatry*, 158, 1878–1883.
- Malter Cohen, M., Jing, D., Yang, R. R., Tottenham, N., Lee, F. S., & Casey, B. J. (2013). Early-life stress has persistent effects on amygdala function and development in mice and humans. *Proceedings of the National Academy of Sciences of the United States of America*, 110(45), 18274–18278.
- Maxfield, M. G., & Widom, C. S. (1996). The cycle of violence: Revisited 6 years later. *Archives of Pediatrics & Adolescent Medicine*, 150(4), 390–395. doi:10.1001/archpedi.1996.02170290056009
- McCormick, C. M., & Mathews, I. Z. (2007). HPA function in adolescence: role of sex hormones in its regulation and the enduring consequences of exposure to stressors. *Pharmacology, Biochemistry, and Behavior*, 86, 220–233.
- McEwen, B. S. (1999). Stress and hippocampal plasticity. *Annual Review of Neuroscience*, 22, 105–122.
- McEwen, B. S. (2012). Brain on stress: How the social environment gets under the skin. *Proceedings of the National Academy of Sciences, USA*, 109, 17180–17185.
- McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood adversity. *Current Directions in Science*, 25(4), 239–245.
- McLaughlin, K. A., Sheridan, M. A., Tibu, F., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2015). Causal effects of the early caregiving environment on development of stress response systems in children. *Proceedings of the National Academy of Sciences*, 112(8), 5637–5642.
- McLaughlin, K. A., Sheridan, M. A., Winter, W., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2014). Widespread reductions in cortical thickness following severe early-life deprivation: a neurodevelopmental pathway to attention-deficit/hyperactivity disorder. *Biological psychiatry*, 76(8), 629–638. <https://doi.org/10.1016/j.biopsych.2013.08.016>
- Mehta, M. A., Gore-Langton, E., Golembo, N., Colvert, E., Williams, S. C., & Sonuga-Barke, E. (2010). Hyporesponsive reward anticipation in the basal ganglia following severe institutional deprivation early in life. *Journal of Cognitive Neuroscience*, 22(10), 2316–2325. <http://doi.org/10.1162/jocn.2009.21394>
- Milner, J. S., & Robertson, K. R. (1989). Inconsistent response patterns and the prediction of child maltreatment. *Child Abuse & Neglect*, 13(1), 59–64.
- Molet, J., Maras, P. M., Avishai-Eliner, S., & Baram, T. Z. (2014). Naturalistic rodent models of chronic early-life stress. *Developmental Psychobiology*, 56(8), 1675–1688. doi:10.1002/dev.21230
- Nederhof, E., & Schmidt, M. V. (2012). Mismatch or cumulative stress: Toward an integrated hypothesis of programming effects. *Physiology & Behavior*, 106, 691–700. doi:10.1016/j.physbeh.2011.12.008
- Nelson, E. C., & Winslow, J. T. (2009). Non-human primates: Model animals for developmental psychopathology. *Neuropsychopharmacology Reviews*, 34, 90–105.
- Nelson, C. A., III, Zeanah, C. H., Fox, N. A., Marshall, P. J., Smyke, A. T., & Guthrie, D. (2007). Cognitive recovery in socially deprived young children: The Bucharest Early Intervention Project. *Science*, 318(5858), 1937–2940.
- Noble, K. G., McCandliss, B. D., & Farah, M. J. (2007). Socioeconomic gradients predict individual differences in neurocognitive abilities. *Developmental Science*, 10(4), 464–480.

- O'Neal, C. R., Brotman, L. M., Huang, K. Y., Gouley, K. K., Kamboukos, D., Calzada, E. J., & Pine, D. S. (2010). Understanding relations among early family environment, cortisol response, and child aggression via a prevention experiment. *Child Development*, 81, 290–305.
- Ovtscharoff, W., & Braun, K. (2001). Maternal separation and social isolation modulate the postnatal development of synaptic composition in the infralimbic cortex of *Octodon degus*. *Neuroscience*, 104(1), 33–40.
- Pagliaccio, D., Luby, J. L., Bogdan, R., Agrawal, A., Gaffrey, M. S., Belden, A. C., ... Barch, D. M. (2014). Stress-system genes and life stress predict cortisol levels and amygdala and hippocampal volumes in children. *Neuropsychopharmacology*, 39(5), 1245–1253. doi:10.1038/npp.2013.327
- Papale, L., Seltzer, L. J., Madrid, A., Pollak, S. D., & Alisch, R. S. (2018). Differentially methylated genes in saliva are linked to childhood stress. *Scientific Reports*, 8, 10785. doi:10.1038/s41598-018-29107-0
- Paul, R., Henry, L., Grieve, S. M., Guilmette, T. J., Niaura, R., Bryant, R., ... Gordon, E. (2008). The relationship between early life stress and microstructural integrity of the corpus callosum in a non-clinical population. *Neuropsychiatric Disease and Treatment*, 4(1), 103–201.
- Pechtel, P., & Pizzagalli, D. A. (2011). Effects of early life stress on cognitive and affective function: An integrated review of human literature. *Psychopharmacology*, 214(1), 55–70.
- Pitzer, M., Jennen-Steinmetz, C., Esser, G., Schmidt, M. H., & Laucht, M. (2011). Prediction of preadolescent depressive symptoms from child temperament, maternal distress, and gender: Results of a prospective, longitudinal study. *Journal of Developmental and Behavioral Pediatrics*, 32(1), 18–26. doi:10.1097/DBP.0b013e3181f4a474
- Poeggel, G., Helmeke, C., Abraham, A., Schwabe, T., Friedrich, P., & Braun, K. (2003). Juvenile emotional experience alters synaptic composition in the rodent cortex, hippocampus, and lateral amygdala. *Proceedings of the National Academy of Sciences*, 100(26), 16137–16142. doi:10.1073/pnas.2434663100
- Pollak, S. D. (2015). Multilevel developmental approaches to understanding the effects of child maltreatment: Recent advances and future challenges. *Development and Psychopathology*, 27(4, Pt. 2), 1387–1397.
- Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. *Developmental Psychology*, 36(5), 679–688. doi:10.1037/0012-1649.36.5.679
- Provencal, N., & Binder, E. B. (2015). The effects of early life stress on the epigenome: From the womb to adulthood and even before. *Experimental Neurology*, 268, 10–20. doi:10.1016/j.expneurol.2014.09.001
- Pryce, C. R., Bettschen, D., Nanz-Bahr, N. I., & Feldon, J. (2003). Comparison of the effects of early handling and early deprivation on conditioned stimulus, context, and spatial learning and memory in adult rats. *Behavioral Neuroscience*, 117, 883–893. doi:10.1037/0735-7044.117.5.883
- Repetti, R. L., Robles, T. F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology*, 23(3), 921–938.
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, 128(2), 330–366.
- Reuben, A., Moffitt, T. E., Caspi, A., Belsky, D. W., Harrington, H., Schroeder, F., ... Danese, A. (2016). Lest we forget: comparing retrospective and prospective assessments of adverse childhood experiences in the prediction of adult health. *Journal of Child Psychology and Psychiatry*, 57(10), 1103–1112.
- Romens, S. E., McDonald, J., Svaren, J., & Pollak, S. D. (2015). Associations between early life stress and gene methylation in children. *Child Development*, 86, 303–309. doi:10.1111/cdev.12270
- Saltzman, W., Hogan, B. K., & Abbott, D. H. (2006). Diminished cortisol levels in subordinate female marmosets are associated with altered central drive to the hypothalamic-pituitary-adrenal axis. *Biological psychiatry*, 60(8), 843–849. https://doi.org/10.1016/j.biopsych.2005.12.006
- Sanchez, C., Arnt, J., Hyttel, J., & Moltzen, E. K. (1993). The role of serotonergic mechanisms in inhibition of isolation-induced aggression in male mice. *Psychopharmacology*, 110, 53–59.

- Sanchez, M. M. (2006). The impact of early adverse care on HPA axis development. Nonhuman primate models. *Hormones and Behavior*, 50, 623–631.
- Sandi, C., & Haller, J. (2015). Stress and the social brain: Behavioural effects and neurobiological mechanisms. *Nature Reviews. Neuroscience*, 16, 290–304. doi:10.1038/nrn3918
- Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1986). The neuroendocrinology of stress and aging: The glucocorticoid cascade hypothesis. *Endocrinology Review*, 7(3), 284–301.
- Scott, K. M., Smith, D. R., & Ellis, P. M. (2010). Prospectively ascertained child maltreatment and its association with DSM-IV mental disorders in young adults. *Archives of General Psychiatry*, 67(7), 712–719. doi:10.1001/archgenpsychiatry.2010.71
- Shackman, J. E., Fatani, S., Camras, L. A., Berkowitz, M. J., Bachorowski, J., & Pollak, S. D. (2010). Emotion expression among abusive mothers is associated with their children's emotion processing and problem behaviours. *Cognition and Emotion*, 24(8), 1421–1430. <https://doi.org/10.1080/02699930903399376>
- Shackman, J. E., & Pollak, S. D. (2014). Impact of physical maltreatment on the regulation of negative affect and aggression. *Development and Psychopathology*, 26, 1021–1033. doi:10.1017/S0954579414000546
- Shackman, J. E., Shackman, A. J., & Pollak, S. D. (2007). Physical abuse amplifies attention to threat and increases anxiety in children. *Emotion*, 7(4), 838–852.
- Sheridan, M. A., Fox, N. A., & Zeanah, C. H. (2012). Variation in neural development as a result of exposure to institutionalization early in childhood. *Proceedings of the National Academy of Sciences*, 109(32), 12927–12932. <http://doi.org/10.1073/pnas.1200041109/-/DCSupplemental>
- Spinelli, S., Chefer, S., Suomi, S. J., Higley, J. D., Barr, C. S., & Stein, E. (2009). Early-life stress induces long-term morphologic changes in primate brain. *Archives of General Psychiatry*, 66(6), 658–665. doi:10.1001/archgenpsychiatry.2009.52
- Sterlemann, V., Ganea, K., Liebl, C., Harbich, D., Alam, S., Holsboer, F., ... Schmidt, M. V. (2008). Long-term behavioral and neuroendocrine alterations following chronic social stress in mice: Implications for stress-related disorders. *Hormones and Behavior*, 53(2), 386–394.
- Sujan, A. C., Humphreys, K. L., Ray, L. A., & Lee, S. S. (2014). Differential association of child abuse with self-reported versus laboratory-based impulsivity and risk-taking in young adulthood. *Child Maltreatment*, 19, 145–155.
- Teicher, M. H., Anderson, C. M., Ohashi, K., & Polcari, A. (2014). Childhood maltreatment: Altered network centrality of cingulate, precuneus, temporal pole and insula. *Biological Psychiatry*, 76(4), 297–305. <http://doi.org/10.1016/j.biopsych.2013.09.016>
- Teicher, M. H., Tomoda, A., & Andersen, S. L. (2006). Neurobiological consequences of early stress and childhood maltreatment: Are results from human and animal studies comparable? *Annals of the New York Academy of Sciences*, 1071, 313–323.
- Thornberry, T. P., Henry, K. L., Ireland, T. O., & Smith, C. A. (2010). The causal impact of childhood-limited maltreatment and adolescent maltreatment on early adult adjustment. *The Journal of Adolescent Health*, 46(4), 359–365.
- Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., Millner, A., Galvan, A., Davidson, M. C., Eigsti, I. M., Thomas, K. M., Freed, P. J., Booma, E. S., Gunnar, M. R., Altemus, M., Aronson, J., & Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Developmental science*, 13(1), 46–61. <https://doi.org/10.1111/j.1467-7687.2009.00852.x>
- Tottenham, N., & Sheridan, M. A. (2010). A review of adversity, the amygdala and the hippocampus: A consideration of developmental timing. *Frontiers in Human Neuroscience*, 3, 68. doi:10.3389/neuro.09.068.2009 eCollection 2009
- Tyrka, A. R., Ridout, K. R., & Parade, S. H. (2016). Childhood adversity and epigenetic regulation of glucocorticoid signaling genes: Associations in children and adults. *Development and Psychopathology*, 28, 1319–1331. <https://doi.org/10.1017/S0954579416000870>
- UN World Health Organization (WHO). (2002). World report on violence and health, ISBN 92 4 154561 5. Retrieved from <https://www.refworld.org/docid/54aa8f74.html>

- Vachon, D. D., Krueger, R. F., Rogosch, F. A., & Cicchetti, D. (2015). Assessment of the harmful psychiatric and behavioral effects of different forms of child maltreatment. *JAMA Psychiatry*, 72(11), 1135–1142. doi:10.1001/jamapsychiatry.2015.1792
- Van Bodegom, M., Homberg, J. R., & Henckens, M. J. A. G. (2017). Modulation of the hypothalamic-pituitary-adrenal axis by early life stress exposure. *Frontiers in Cellular Neuroscience*, 11, 87. doi:10.3389/fncel.2017.00087
- Weller, J. A., & Fisher, P. A. (2013). Decision-making deficits among maltreated children. *Child Maltreatment*, 18(3), 184–194.
- Widom, C. S., & Morris, S. (1997). Accuracy of adult recollections of childhood victimization, part 2: childhood sexual abuse. *Psychological Assessment*, 9(1), 34–46. <https://doi.org/10.1037/1040-3590.9.1.34>
- Yates, T. M., Tracy, A. J., & Luthar, S. S. (2008). Nonsuicidal self-injury among “privileged” youths: Longitudinal and cross-sectional approaches to developmental process. *Journal of Consulting and Clinical Psychology*, 76(1), 52–62.
- Zannas, A., Wiechmann, T., Gassen, N., & Binder, E. B. (2016). Gene–stress–epigenetic regulation of *FKBP5*: Clinical and translational implications. *Neuropsychopharmacology*, 41, 261–274. <https://doi.org/10.1038/npp.2015.235>
- Zannas, A. S., & Binder, E. B. (2014). Gene–environment interactions at the *FKBP5* locus: sensitive periods, mechanisms and pleiotropism. *Genes, Brain, and Behavior*, 13, 25–37.
- Zeanah, C. H., Nelson, C. A., Fox, N. A., Smyke, A. T., Marshall, P., Parker, S. W., & Koga, S. (2003). *Development and Psychopathology*, 15(4), 885–907.

Suggested Reading

- Heim, C., & Binder, E. B. (2012). Current research trends in early life stress and depression: Review of human studies on sensitive periods, gene–environment interactions, and epigenetics. *Experimental Neurology*, 233(1), 102–111. doi:10.1016/j.expneurol.2011.10.032
- Hostinar, C. E., & Gunnar, M. R. (2013). The developmental effects of early life stress: An overview of current theoretical frameworks. *Current Directions in Psychological Science*, 22, 400–406. doi:10.1177/0963721413488889
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behavior and cognition. *Nature Reviews Neuroscience*, 10, 434–445. doi:10.1038/nrn2639

Alcohol-Related Brain Disorders

Rebecca N. Preston-Campbell¹, Stephanie A. Peak²,
Paola García-Egan³, and Robert H. Paul^{1,3}

¹*Missouri Institute of Mental Health, St. Louis, MO, USA*

²*Battelle Memorial Institute, Columbus, OH, USA*

³*Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA*

Introduction

Problematic alcohol consumption is a major contributor to disability-adjusted life years worldwide (World Health Organization [WHO], 2014). Much of the burden is due to neuropsychiatric disorders, primarily alcohol use disorders, causing disability and mortality compared with other chronic diseases (WHO, 2014). It is well established that excessive alcohol consumption directly impacts brain structure and function (de la Monte & Krill, 2014; Geibprasert, Gallucci, & Krings, 2010; Keil, Greschus, Schneider, Hadizadeh, & Schild, 2015; Kim, Lee, Young, Shin, & Kim, 2014; Zahr, Kaufman, & Harper, 2011). The mechanisms are varied and include the direct neurotoxic effects of alcohol on neurons and secondary effects of hepatic dysfunction and/or nutritional deficiency. Collectively, the neuropathological, morphological, and biochemical changes are referred to as alcohol-related brain damage (ARBD) (Zahr et al., 2011). In this chapter, we describe the clinical characteristics, neuropathological features, and neuroimaging signatures of ARBD.

Alcohol Consumption and Brain Dysfunction

The long-term effects of chronic heavy drinking, frequent binge drinking episodes (five drinks for men, four drinks for women on 5 or more days in the past month) (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2018), produce structural brain changes and impairment of cognitive function in some, but not all, individuals (de la Monte & Krill, 2014; Geibprasert et al., 2010; Keil et al., 2015; Kim et al., 2014; Zahr et al., 2011). Neuroimaging studies report a strong interdependence between lifetime alcohol consumption and loss of

white and gray brain volumes. Literature has suggested that 5–6 drinks per day over an extended period of time correspond to mild cognitive impairment and the consumption of 10 or more drinks per day results in moderate cognitive deficits (Ridley, Draper, & Withall, 2013). However, the long-term effects of light-to-moderate drinking, defined as up to one drink per day for women and up to two drinks per day for men, (2015–2020 Dietary Guidelines for Americans, 2015) on the human brain are poorly understood.

Some studies report an association between recent moderate alcohol use and reduced total brain volume, gray matter and hippocampal atrophy, and impaired white matter microstructure, while other studies show no such association (Ridley et al., 2013; Topiwala et al., 2017). These inconsistencies are likely due to (a) disproportional use of cross-sectional research designs; (b) confounds related to comorbid illicit drug use, including nicotine; (b) inaccurate estimates of alcohol intake from self-report; (d) heterogeneity of sample demographics; and (e) insufficient focus on sex-specific outcomes.

Mechanisms of Alcohol-Induced Brain Damage

Although not completely understood, several mechanisms have been proposed to explain ARBD. For example, ethanol-specific mechanisms underlying ARBD have been attributed to oxidative stress and the accumulation of toxic metabolites. Oxidative stress occurs due to an accumulation of free radicals (reactive oxygen species) causing DNA damage, the inhibition of gene expression, and neuronal death. Toxic metabolites of ethanol, such as acetaldehyde and fatty acid ethyl esters, can accumulate and lead to adduct formation, interrupting mitochondrial formation inducing neuronal damage (Crews & Nixon, 2009; Zahr et al., 2011). Further, immune-related inflammatory responses and subsequent neural degeneration may be caused by liver damage due to the synergistic effects of acetaldehyde protein adduct formation and oxidative stress (Zahr et al., 2011). A mechanistic role for implicating neuroinflammation in the pathogenesis and progression of ARBD links neuroimmune signaling and glutamate excitotoxicity to alcohol-related neurodegeneration (Crews & Vetreno, 2014). Studies report that lifetime alcohol consumption correlates with neuroimmune gene expression and signs of neuroinflammation in the hippocampus, amygdala, and ventral tegmental area among individuals with alcohol use disorder (Crews & Vetreno, 2014; Zahr et al., 2011). Readers are referred to the following publications for more detailed reviews (Crews & Nixon, 2009; Crews & Vetreno, 2014; Fadda & Rossetti, 1998; Harper, 2009).

Chronic heavy alcohol consumption also produces thiamine deficiency via decreased thiamine absorption in the gastrointestinal tract, inadequate nutritional thiamine intake (due to poor diet), and impaired thiamine utilization in the cells. Low levels of thiamine pyrophosphate disrupts the following processes: amino acid metabolism interrupting the release of glucose-derived neurotransmitters and carbohydrate metabolism producing a cytotoxic effect through reduction in α -KGDH activity (de la Monte & Krill, 2014; Zahr et al., 2011). The synergistic effects of thiamine deficiency and heavy alcohol use are associated with the loss of neural plasticity and reduction in what matter volume due to a reduction in GABAergic inhibition and increase in glutamatergic excitation (Costin & Miles, 2014; Zahr et al., 2011).

Wernicke–Korsakoff Syndrome

Wernicke–Korsakoff syndrome (WKS), composed of Wernicke’s encephalopathy (WE) and Korsakoff syndrome (KS), is one of the most frequently seen neurological consequences

associated with heavy, long-term alcohol use (Kim et al., 2014; Kopelman, Thomson, Guerrini, & Marshall, 2009). While the two conditions share some overlapping pathogenesis, WE and KS produce unique clinical phenotypes.

Wernicke's Encephalopathy

WE, an acute, potentially reversible neuropsychiatric condition, is a medical emergency caused by thiamine deficiency (Costin & Miles, 2014; Kopelman et al., 2009). It is marked by a clinical triad of symptoms including ataxia, global confusion, and ophthalmoplegia (Kim et al., 2014). However, most patients present with nonspecific cognitive changes, ocular motor abnormalities, and cerebellar dysfunction. Altered mental state is the most prevalent symptom, which may present as mental sluggishness, impaired awareness of surroundings, difficulty concentration, apathy, and confusion or agitation (Kim et al., 2014; Kopelman et al., 2009). The use of thiamine or thiamine-containing products for the treatment of WKS should be implemented as soon as possible beginning with parenteral or intravenous administration of thiamine for several days, followed by daily oral administration prevents mortality, but does not protect individuals from residual neurological symptoms (Costin & Miles, 2014). Even with prompt treatment, global confusion persists for up to three months, and chronic memory deficits remain thereafter (Costin & Miles, 2014).

Neuropathological features of both acute and chronic WE include global brain atrophy with disproportionate white matter volume loss. Symmetrical hemorrhagic lesions in the mammillary bodies, thalamus, brain stem, hypothalamus, and cerebellum are seen in acute WE, whereas atrophy and brownish discoloration of the mammillary bodies, anterior nuclei, and medial-dorsal nuclei of thalami are more common in chronic cases (de la Monte & Krill, 2014). Magnetic resonance (MR) findings in WE include edema hyperintensities in the gray matter of the periventricular region, mammillary bodies, medial thalamus, and superior/inferior colliculi (Keil et al., 2015; Kim et al., 2014). In the chronic stage, MR findings show brain atrophy and diffuse signal intensity changes in affected areas and cerebral white matter (Kim et al., 2014).

Korsakoff Syndrome

Approximately 80% of patients with acute WE will progress to KS (Costin & Miles, 2014; Keil et al., 2015; Kopelman et al., 2009). As many as 25% of affected cases require long-term psychiatric care (Kopelman et al., 2009). The most characteristic clinical symptom of KS is mental status change (Costin & Miles, 2014; Keil et al., 2015; Kopelman et al., 2009). Memory impairments in KS are characterized by marked inability to form declarative memories (explicit) with less disruption to semantic memory and procedural memory (implicit). Oftentimes, affected individuals exhibit confabulations (involuntary, false or erroneous memories or intrusion errors) (Kopelman et al., 2009).

Morphological changes such as cortical thinning, sulcal widening, mammillary body atrophy, and ventricular enlargement are more severe in KS than WE. A significant decrease in Purkinje cell density in the cerebellar vermis and micro-hemorrhages and gliosis in the periventricular and periaqueductal gray matter are also present and relate to ataxia/unsteadiness and memory dysfunction (Kopelman et al., 2009). Neuroimaging abnormalities are seen in the mammillary bodies, posterior midbrain, medial thalamus, hypothalamus, pons, and hippocampus (Keil et al., 2015) and focal cerebral edema detected on MRI. Anterograde memory deficits are

associated with reduced thalamus/hippocampus volume, whereas retrograde and working memory deficits are attributed to frontal lobe dysfunction. Further, cortical damage in the frontocerebellar and limbic circuits (Kopelman et al., 2009) relates to level of impairment in episodic memory.

Marchiafava–Bignami Disease

Originally called the “red wine drinker’s encephalopathy,” Marchiafava–Bignami disease (MBD) is a rare neurologic disorder associated with chronic heavy alcohol use seen in only 0.01% of patients with alcohol-related diseases (Costin & Miles, 2014). Most prevalent in men between 40 and 60 of age, MBD results in progressive demyelination and necrosis of the corpus callosum (Carrilho, Santos, Piasecki, & Jorge, 2013). Deficiency in thiamine and deficiency in all eight B vitamins have been implicated as possible cofactors, but the exact etiology of MBD is unknown. Although there is no standard clinical presentation of MBD, symptoms include depression, psychosis, hemiparesis, apraxia, and seizures. Untreated symptoms can be fatal or coma inducing (Carrilho et al., 2013; Costin & Miles, 2014).

In the acute phase, individuals with MBD exhibit severe impairment of consciousness, confusion, muscle rigidity, and gait disturbance. Signs of interhemispheric disconnection syndrome are possible (Carrilho et al., 2013; Costin & Miles, 2014). By contrast, the chronic phase is characterized by dementia and gait impairment that can progress over time (Costin & Miles, 2014). Treatment options are limited to replacement of depleted B vitamins and thiamine, corticosteroids, and amantadine with variable response (Kim et al., 2014). Patients who survive should receive rehabilitation and alcohol counseling.

The primary neuropathological features associated with MBD include degeneration and necrosis of the corpus callosum, predominantly at the body, genu, and splenium. Imaging features, consistent with pathology, are edema, demyelination, and later necrosis of the corpus callosum with formation of cystic cavities (Carrilho et al., 2013). Computed tomography (CT) scans reveal diffuse periventricular low density and focal areas of low density in genu and splenium of corpus callosum, and MRI show hyperintense signals within the genu and splenium and adjacent white matter (Kim et al., 2014). The splenium has more myelin than any other part of the corpus callosum, so intramyelinic edema has been proposed as the cause of more frequent lesions in this structure, and cytotoxic edema has also been proposed as a possible mechanism for early stage demyelination (Carrilho et al., 2013; Kim et al., 2014).

Osmotic Demyelination Syndrome (Central Pontine or Extra Pontine Myelinolysis)

Osmotic demyelination syndrome (ODMS) is a disease involving the central portion of the base of the pons (Turnbull, Lumsden, Siddiqui, Lin, & Lim, 2013). Chronic alcoholism is the most frequent underlying condition, yet the exact pathophysiology is not understood (Keil et al., 2015; Turnbull et al., 2013). Typically, ODMS presents with generalized encephalopathy with a period of recovery prior to a second phase of neurological deterioration (Keil et al., 2015; Turnbull et al., 2013). Associated symptoms such as decreased voluntary muscle control, acute changes in mental status, gaze paresis, and the inability to control facial movements reflect areas of demyelination. A locked-in syndrome, in which the patient is aware but cannot move or communicate due to paralysis of voluntary muscles,

may occur in more severe cases; progression to coma or death occurs in 50% of cases (Keil et al., 2015).

Neuropathological findings show demyelination of nerve fibers in the pons without destruction of axons. Extrapontine, symmetrical areas of myelin disruption involving the thalamus, putamen, and cerebellum have also been described. MRI shows abnormal signal in central pons, basal ganglia, thalami, cerebellum, and lateral geniculate; CT reveals low-density lesions in the pons (Keil et al., 2015; Turnbull et al., 2013).

Hepatic Encephalopathy

Hepatic encephalopathy (HE) is a reversible metabolic encephalopathy characterized by psychiatric, cognitive, and motor abnormalities, occurring in patients with advanced liver failure and cirrhosis (Geibprasert et al., 2010; Keil et al., 2015). Pathogenesis has been attributed to the inadequate removal of nitrogenous compounds resulting in the accumulation of ammonia, manganese, and mercaptans (Geibprasert et al., 2010). Hypermanganesemia can induce reactive gliosis and selective neuronal loss in the basal ganglia and midbrain (Geibprasert et al., 2010).

HE presents with various reversible clinical symptoms such as deficits in attention, personality changes (apathy, irritability, disinhibition), motor incoordination, and alterations in consciousness. Frequent disturbances to the sleep–wake cycle with excessive daytime sleepiness, inappropriate behavior, progressive disorientation/acute confusion, and stupor that progresses to coma may occur in severe cases (Geibprasert et al., 2010; Keil et al., 2015). The central nervous system (CNS)-related cognitive changes in HE are attributed to an accumulation of manganese in the globus pallidus, hypothalamus, and anterior midbrain. Edema of the gray matter, in particular the insular cortex, cingulate, and basal ganglia, occurs in the early stages and relates to sleep disturbances and subtle behavioral changes (Geibprasert et al., 2010; Keil et al., 2015). Imaging findings show bilateral symmetrical signal hyperintensity in the basal ganglia (e.g., caudate, putamen, substantia nigra, red nucleus) (Geibprasert et al., 2010; Keil et al., 2015).

Alcoholic Cerebellar Degeneration

Alcoholic cerebellar degeneration is characterized by slowly progressive ataxia and broad irregular gait; other symptoms such as slurred speech, impaired muscle control (dysarthria), and horizontal nystagmus are also reported. Postmortem pathology reveals a loss of Purkinje cells. Imaging studies show both gray and white matter deficits and hypometabolism in the anterior superior cerebellar vermis and evidence of vermal atrophy (Fitzpatrick, Jackson, & Crowe, 2008).

Alcohol-Related Dementia

Alcohol-related dementia (ARD) is a controversial clinical entity. Some argue that ARD is nonspecific neuropathological disorder given the lack of distinct etiologic mechanisms, suggesting symptoms are due to ethanol neurotoxicity, other underlying pathology, or combination of factors (Ridley et al., 2013; Schmidt et al., 2005). Diagnostic criteria were

developed to improve the classification of ARD as a syndrome (Oslin, Atkinson, Smith, & Hendrie, 1998). Yet, clinical judgment continues to be a popular approach to diagnosis. Controversy aside, empirical studies reveal a correspondence between cognitive decline and the chronicity/severity of alcohol use (Oslin *et al.*, 1998) caused by glutamate excitotoxicity, oxidative stress, and disruption of neurogenesis (Ridley *et al.*, 2013).

Cognitive deficits in ARD are linked to quantity and recency of use and duration of abstinence, with binge drinking episodes and multiple withdrawals from alcohol exacerbating impairment (Sachdeva, Chandra, Choudhary, Dayal, & Anand, 2016). The neuropsychological profile is characterized by deficits in executive functioning, working memory, and motor speed, with sparing of language abilities (Ridley *et al.*, 2013). ARD is rarely progressive, is most common in middle-aged individuals, and may resolve/stabilize following abstinence from alcohol (Sachdeva *et al.*, 2016). The pattern and rate of cognitive recovery is not fully understood; however, recovery of cognitive functioning appears to be related to the amount of recent alcohol use and duration of abstinence (Ridley *et al.*, 2013).

Cognitive deficits typically persist after cessation of drinking as a result of impaired fronto-cortico-cerebellar functional networks. Neuroimaging evidence shows sulcal swelling, dilation of the ventricles, and prominent gray and white matter loss in the prefrontal cortex, corpus callosum, and cerebellum. Consistent with these findings, results from a single-photon emission CT study report hypometabolism in the frontal cortices, thalami, and basal ganglia. (Sachdeva *et al.*, 2016). However, some partial structural and functional recovery after abstinence has been reported (Ridley *et al.*, 2013; Schmidt *et al.*, 2005).

Conclusions and Future Directions

Although it is well established that excessive alcohol consumption leads to structural and functional damage of the brain, many questions remain concerning long-term effects of alcohol use on brain health. The complex interaction between predisposing traits, environmental factors, and neurological impairments due to the synergistic effects of thiamine deficiency to ethanol neurotoxicity results in neurocognitive impairment and a number of clinical conditions. The complex and often overlapping pathologies make it challenging to achieve an exact diagnosis. Classical syndromes rarely appear isolated, and often overlap, incompletely or atypically. Indeed, multiple patterns of alcohol use, personal and lifestyle factors, and the vulnerability of specific brain regions make research about this issue particularly challenging.

Prospective studies of alcohol use on the brain in both men and women are needed to clarify whether there are sex differences in ARBD and to determine graded risks associated with alcohol consumption. Additionally, further clarification is needed to elucidate the interaction between early onset of alcohol consumption, chronic alcohol exposure, and neurodegeneration. The identification of specific mechanisms underlying ARBD will aid in accurate diagnosis and inform development of disorder-specific interventions.

Author Biographies

Rebecca N. Preston-Campbell, PhD, is a research scientist at the Missouri Institute of Mental Health. Her research interests focus on emotional stressors and impaired self-regulatory behaviors that may both predate and maintain substance use disorders, with an emphasis on sex differences.

Stephanie A. Peak, PhD, is a researcher at Battelle Memorial Institute where she contributes to government projects related to the understanding and improvement of public health. Dr. Peak's research interests focus on attitudinal and health consequences of loss, maladaptive behaviors, and threat (broadly defined).

Paola García-Egan is a PhD candidate at the University of Missouri–St. Louis. She has published articles on standardization of neuropsychological tests for Latin American population and parental ethnotheories and book chapters on vascular dementia and myasthenia gravis. Her current research includes early life stress and aging.

Dr. Robert H. Paul is director of the Missouri Institute of Mental Health and professor of psychological sciences at University of Missouri–St. Louis. His research program is focused on delineating the underlying mechanisms of cognitive phenotypes in adult and pediatric neurologic disease.

References

- 2015–2020 Dietary Guidelines for Americans. (2015). In (8th Edition ed.): U.S. Department of Health and Human Services and U.S. Department of Agriculture.
- Carrilho, P. E., Santos, M. B., Piasecki, L., & Jorge, A. C. (2013). Marchiafava-Bignami disease: A rare entity with a poor outcome. *Revista Brasileira de Terapia Intensiva*, 25(1), 68–72.
- Costin, B. N., & Miles, M. F. (2014). Molecular and neurologic responses to chronic alcohol use. *Handbook of Clinical Neurology*, 125, 157–171. doi:10.1016/B978-0-444-62619-6.00010-0
- Crews, F. T., & Nixon, K. (2009). Mechanisms of neurodegeneration and regeneration in alcoholism. *Alcohol and Alcoholism*, 44(2), 115–127. doi:10.1093/alcalc/agn079
- Crews, F. T., & Vetreno, R. P. (2014). Neuroimmune basis of alcoholic brain damage. *International Review of Neurobiology*, 118, 315–357. doi:10.1016/B978-0-12-801284-0.00010-5
- de la Monte, S. M., & Krill, J. J. (2014). Human alcohol-related neuropathology. *Acta Neuropathologica*, 127(1), 71–90.
- Fadda, F., & Rossetti, Z. L. (1998). Chronic ethanol consumption: From neuroadaptation to neurodegeneration. *Progress in Neurobiology*, 56(4), 385–431.
- Fitzpatrick, L. E., Jackson, M., & Crowe, S. F. (2008). The relationship between alcoholic cerebellar degeneration and cognitive and emotional functioning. *Neuroscience and Biobehavioral Reviews*, 32(3), 466–485. doi:10.1016/j.neubiorev.2007.08.004
- Geibprasert, S., Gallucci, M., & Krings, T. (2010). Alcohol-induced changes in the brain as assessed by MRI and CT. *European Radiology*, 20(6), 1492–1501. doi:10.1007/s00330-009-1668-z
- Harper, C. (2009). The neuropathology of alcohol-related brain damage. *Alcohol and Alcoholism*, 44(2), 136–140. doi:10.1093/alcalc/agn102
- Keil, V. C., Greschus, S., Schneider, C., Hadizadeh, D. R., & Schild, H. H. (2015). The whole spectrum of alcohol-related changes in the CNS: Practical MR and CT imaging guidelines for daily clinical use. *Röfo*, 187(12), 1073–1083. doi:10.1055/s-0035-1553509
- Kim, T. E., Lee, E. J., Young, J. B., Shin, D. J., & Kim, J. H. (2014). Wernicke encephalopathy and ethanol-related syndromes. *Seminars in Ultrasound, CT, and MR*, 35(2), 85–96. doi:10.1053/j.sult.2013.09.004
- Kopelman, M. D., Thomson, A. D., Guerrini, I., & Marshall, E. J. (2009). The Korsakoff syndrome: Clinical aspects, psychology and treatment. *Alcohol and Alcoholism*, 44(2), 148–154. doi:10.1093/alcalc/agn118
- National Institute on Alcohol Abuse and Alcoholism. (2018). Alcohol Facts and Statistics. Retrieved from <https://pubs.niaaa.nih.gov/publications/AlcoholFacts&Stats/AlcoholFacts&Stats.pdf>
- Oslin, D., Atkinson, R. M., Smith, D. M., & Hendrie, H. (1998). Alcohol related dementia: Proposed clinical criteria. *International Journal of Geriatric Psychiatry*, 13(4), 203–212.
- Ridley, N. J., Draper, B., & Withall, A. (2013). Alcohol-related dementia: An update of the evidence. *Alzheimer's Research & Therapy*, 5(1), 3. doi:10.1186/alzrt157

- Sachdeva, A., Chandra, M., Choudhary, M., Dayal, P., & Anand, K. S. (2016). Alcohol-related dementia and neurocognitive impairment: A review study. *International Journal of High Risk Behaviors and Addiction*, 5(3), e27976. doi:10.5812/ijhrba.27976
- Schmidt, K. S., Gallo, J. L., Ferri, C., Giovannetti, T., Sestito, N., Libon, D. J., & Schmidt, P. S. (2005). The neuropsychological profile of alcohol-related dementia suggests cortical and subcortical pathology. *Dementia and Geriatric Cognitive Disorders*, 20(5), 286–291. doi:10.1159/000088306
- Topiwala, A., Allan, C. L., Valkanova, V., Zsoldos, E., Filippini, N., Sexton, C., ... Ebmeier, K. P. (2017). Moderate alcohol consumption as risk factor for adverse brain outcomes and cognitive decline: Longitudinal cohort study. *BMJ*, 357, j2353. doi:10.1136/bmj.j2353
- Turnbull, J., Lumsden, D., Siddiqui, A., Lin, J. P., & Lim, M. (2013). Osmotic demyelination syndrome associated with hypophosphataemia: 2 cases and a review of literature. *Acta Paediatrica*, 102(4), e164–e168. doi:10.1111/apa.12143
- World Health Organization. (2014). *Global status report on alcohol and health*. Geneva, Switzerland: Author.
- Zahr, N. M., Kaufman, K. L., & Harper, C. G. (2011). Clinical and pathological features of alcohol-related brain damage. *Nature Reviews. Neurology*, 7(5), 284–294. doi:10.1038/nrneurol.2011.42

Suggested Reading

- Crews, F. T., Lawrimore, C. J., Walter, T. J., & Coleman, L. G. (2017). The role of neuroimmune signaling in alcoholism. *Neuropharmacology*, 122, 56–73.
- Cui, C., & Koob, G. F. (2017). Titrating tipsy targets: The neurobiology of low-dose alcohol. *Trends in Pharmacological Sciences*, 38(6), 556–568.
- Volkow, N. D., Wiers, C. E., Shokri-Kojori, E., Tomasi, D., Wang, J.-G., & Baler, R. (2017). Neurochemical and metabolic effects of acute and chronic alcohol in the human brain: Studies with positron emission tomography. *Neuropharmacology*, 122, 175–188.
- Zahr, N. M., Pfefferbaum, A., & Sullivan, E. V. (2017). Perspectives on fronto-fugal circuitry from human imaging of alcohol use disorders. *Neuropharmacology*, 122, 189–200.

Index

- [1-¹³C] acetate infusion, usage 153
- 3 Tesla scanner, usage 92
- ¹¹C-PiB retention, increase 305
- 11 β -hydroxysteroid de-hydrogenase-2 (11 β -HSD-2), impact 283
- 11 β -hydroxysteroid dehydrogenase type 2 (Hsd11B2), decrease 84
- [(18)F]-florbetapir 231

- accomplishment (construct) 328
- acetylation 83
- acetylcholine 8, 10
- acetyl groups, HDAC removal 75
- achievement (well-being sub-measure) 328
- Action for Health in Diabetes clinical trial 255
- action potential 7
- acute BDNF activation 38–39
- acute psychosis, treatment 54
- adenosine triphosphate (ATP), hydrolysis 7
- adrenocorticotrophic hormone (ACTH) 362
 - formation 78
 - release 26t
 - increase 51
- adulthood, brain development 1
- Adverse Childhood Events Scale (ACES) 376
- affective empathy 70
- aggression 70, 230
- aging 102–103
 - hippocampal subfields, structural neuroimaging 99

- agrammatism
 - emergence 210
 - features 213
- alcohol consumption, brain dysfunction (relationship) 395–396
- alcoholic cerebellar degeneration 3299
- alcohol-induced brain damage, mechanisms 396
- alcohol-related brain disorders (ARBDs) 395
 - mechanisms, proposal 396
- alcohol-related dementia (ARD) 399–400
- alexithymia 69–70
- allocortex 99
- alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) 8
- alphabet knowledge 356
- alpha-KGDH activity, reduction 396
- Alzheimer's disease (AD) 205
 - [1-¹³C] glucose, usage 153
 - biological studies, ML (application) 165
 - biomarkers 163–164, 301
 - cognitive impairment, association 237
 - development 253
 - risk (increase), APOE ϵ 4 allele (impact) 301–302
 - diagnosis, computer aid (question) 163
 - etiology 120
 - hippocampal subfields, structural neuroimaging 99
 - impact 120
 - MCI conversion 164

- Alzheimer's disease (AD) (*cont'd*)
- mild cognitive impairment, relationship 301–302
 - neuropathological evidence 293
 - pathology, evidence 304–305
 - patients, classification 165
 - risk, increase 254
 - volume loss, pattern 103
- amino acids 8
- amygdala
- amygdala-insula connectivity 347
 - MPFC inhibitory pathway, asymmetry (impact) 364
 - neuroticism, relationship 318–319
 - projections 362
 - reward processing, relationship 364–365
 - volume
 - increase 381
 - reduction 302
- amyloid-beta deposition/plaque formation 163
- amyloid PET data, collection 199
- amyloid precursor protein (APP), beta-amyloid burden (relationship) 197–198
- Angelman Syndrome 76–77
- anisotropic environment, spherical representation 92
- anisotropy, measures 93
- anterior cingulate cortex (ACC) 17
- dorsal ACC, activation 366
- anterior temporal lobes (ATL), svPPA localization 211
- anthropometric characteristics, Met allele risk factor 335–336
- antisocial behavior 70
- anxiety disorders
- characterization, ERPs (usage) 145
 - connectomics 183–184
 - glutamate, presence 153–154
- anxiety, GABA (impact) 155–156
- anxiety-related disorders, epigenetic mechanisms (impact) 86
- apathy 210
- apolipoprotein E (APOE)
- age-related cognitive impairment, risk factor (neuropsychological/neuroimaging findings) 299
 - APOE-4 genotype 254
 - brain integrity, relationship 302–305
 - cerebral perfusion 304
 - functional brain networks 303–304
 - genotype
 - cognition, relationship 300–302
 - presence 229
 - volumetric differences, absence 303
 - magnetic resonance imaging (MRI) 302–304
 - neuroimaging 302–305
 - neuropsychological findings 302, 305
 - regional brain volumes 302–303
 - task-based functional MRI 303
 - white matter integrity 304
 - ε4 allele 228, 299
 - gene-dose relationship 305
 - presence, impact 301–302
- apolipoprotein E (ApoE) gene
- alleles, types 67–68
 - epsilon 4 (ε4) isoform, influence 113–114
- apoptosis, impact 276
- apparent diffusion coefficient (ADC)
- calculation 110
 - values, expression 110
- archicortex 99
- arginine vasopressin (AVP), release 378
- argyrophilic grain disease 238
- arterial spin labeling (ASL) 302
- artificial intelligence (AI), usage 163
- Asperger, Hans 187
- Asperger syndrome (AS) 187
- ADI/ADOS 189–190
 - adult outcome 194
 - behavioral modification 194
 - clinically defined autistic individual, AS
 - individual (contrast) 190–191
 - developmental course 193
 - differential diagnoses 192
 - discovery 187–188
 - educational issues 193
 - employment 194
 - epidemiology 191
 - genetic issues 191–192
 - hidden cohort issue 194
 - history/ages 187–188
 - official disappearance 189–190
 - official entity, transient life 189
 - phenotype 192
 - rediscovery 188
 - resistance 189–190
 - social impairment, basis 192–193
 - treatment/support issues 194–195
- astrocytes 11
- attention-deficit hyperactivity disorder (ADHD) (TDAH) 192
- 15-HTTLPR L allele (relationship) 69
 - risk alleles 199–200
- attention, difficulties 246

- auditory attention processes, stimulus onset reaction 143
- autism 70
 - etiology, SHANK3 (relationship) 77–78
 - syndromic autism 192
- autism spectrum disorders (ASDs), clinical features 77–78
- autistic diagnostic observation schedule (ADOS) 189–190
- autistic language features, absence 188
- autistic psychopathy 187–188
- autistic strategy, building 189
- Automated Segmentation of Hippocampal Subfields (ASHS)
 - protocol, usage 104
 - toolbox 102
- autonomic nervous system (ANS) 24–25
 - heart, acceleration 365
 - HPA, interplay 362
- autonomy (PWB dimension) 327
- AV-1451 231
- avoidance symptoms 287
- Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort study 53
- axial diffusivity (AD) (parallel diffusivity) 92, 95, 110
 - representation 111
- axial kurtosis 118
- axonal growth 282
- axonal integrity 334
- basal circadian activity rhythm 378–379
- basal ganglia 13–15
- Bayesian approach 112
- Beck Depression Inventory, usage 37
- behavior
 - antisocial behavior 70
 - development, prenatal stress (impact) 278–281
 - epigenetics, relationship 83
 - polymorphisms, relationship 61–71
- behavioral disturbances 230
- behavioral modification 1904
- behavioral variant (FTD) (bvFTD) 209–210
- benzodiazepines (BZDs)
 - administration, effects 155–156
 - cessation, impact (meta-analysis) 262–263
 - half-life metabolism 263
 - usage 155
 - use/abuse 262–263
 - use effects, meta-analyses 262
- beta-adrenergic receptor expression, downregulation 33
- Beta-amyloid burden, amyloid precursor protein (APP) relationship 197–198
- beta-amyloid (A β) plaques, extracellular deposition 304–305
- bilateral dorsomedial prefrontal cortex, functional connectivity strength (reduction) 183
- bilateral temporal cortex, first-episode psychosis 157
- bioactivity, variables 333
- biofeedback procedures 222
- biogenic amines 8
- biomarkers, usage 163–166
- bipolar disorder (BD) 144
 - connectomics studies 183
 - GABA, impact 156
 - Glu MRS 154
 - glutamate, presence 154
 - glutathione, impact 158
 - NAA, presence 151–152
- bipolar disorder (BPD) 49
 - inflammation, relationship 52
- bipolar II disorder (BDII) 144
- bipolar neurons 5
- blood-brain barrier (BBB)
 - CORT, passage 27
 - methamphetamine, passage 267
 - proinflammatory cytokines, transport 50
- blood, fluid biomarker 231
- blood oxygenation level-dependent (BOLD)
 - activation 127
 - measurement, fMRI (usage) 303
- blood oxygenation level-dependent (BOLD)
 - signal 125–127
 - components 126
 - fMRI, correlation 182
 - low-frequency BOLD signal, isolation 132
 - PTSD symptom presentations, relationships 344–345
- book reading, literacy (correlation) 355
- borderline personality disorder (BPD) 70–71
 - BDNF methylation patterns, problems (rescue) 86
- bradykinesias 15–16
- brain
 - alcohol-induced brain damage, mechanisms 396
 - alcohol-related brain disorders (ARBDs) 395
 - biochemistry, objective measure 150
 - brain-behavior links, *in vivo* examination 237–238
 - chemicals, MRS (relationship) 150–152
 - cytokines, expression 50

- brain (*cont'd*)
 - diffusional kurtosis images 118
 - dysfunction, alcohol consumption (relationship) 395–396
 - gray matter, development 1–2
 - health 253
 - injury, types 220–221
 - integrity, APOE (relationship) 302–305
 - maturation 276
 - reserve, cognitive reserve (contrast) 293–294
 - structural abnormalities 198–199
 - structure, BDNF Val66Met SNP (relationship) 334
 - vesicles, organ precursor 276
 - white matter, development 1–2
- brain-derived neurotrophic factor (BDNF) 331
 - antidepressant efficacy, relationship 39
 - BDNF Val66Met SNP, brain structure (relationship) 334
 - bioactivity, variables 333
 - biology 332–333
 - cognition, relationship 336–337
 - DRD2/ANKK1 gene, interaction 70
 - expression
 - patterns 333
 - SSRI upregulation 51
 - genetic structure 332–333
 - glucocorticoids, contrast 38
 - hippocampal BDNF expression, investigation 333
 - hypermethylation 86
 - isoforms, conversion 332
 - mature BDNF, impact 332
 - methylation 84, 85, 86
 - neuropsychiatric function, relationship 335–336
 - peripheral BDNF, examination 336
 - pro-BDNF, components 332
 - serum concentrations, low levels 69
 - serum levels, assessment 254
 - TrkB, binding 332
 - truncated BDNF, cognitive performance predictor 336
 - upregulation 257
- brain-derived neurotrophic factor (BDNF) Val66Met
 - DRD2/ANKK1, interactions 69
 - four-way interactions 71
 - polymorphism 67–69
- brain development
 - critical time windows 2
 - detrimental effects 354
 - immune system, involvement 50
 - moderating factors 2–3
 - prenatal environment, impact 2
 - stress exposure, impact 382–383
- Brief International Cognitive Assessment for MS (BICAMS) 45
- broaden-and-build theory 365
- Brownian motion 91
- Bucharest Early Intervention Project 374
- bullying, depression (relationship) 194
- Cajal-Retzius cells 276
- California Verbal Learning Test second edition (CVLT-II), usage 45
- caloric restriction 253
 - cerebrovascular health, relationship 257
 - epidemiological data 254
 - mechanisms 256
 - neurohumoral/growth factor modulation, relationship 257
 - neuroinflammation 256
 - oxidative stress 257–258
 - randomized trials 254–256
- cAMP-regulated phosphoprotein (DARPP-32) gene 60, 67
- cannabis 263–264
 - neurobiological effects, fMRI/PET (usage) 264
 - subspecies 263
 - users, neuropsychological performance 264
- Cannon, Walter 21
- cardiovascular disease, WMH burden increase (correlation) 206
- caregiver
 - abuse, deprivation-related stressors (contrast) 375
 - burnout 214
- catecholamines, derivation 8
- catechol-*O*-methyltransferase (COMT)
 - impact 60, 68
 - Val158Met polymorphism 64, 67
- cavum septum pellucidum 231
- CCL11 gene, increase 231
- cells, types 5
- central executive network (CEN) 133, 134
- central nervous system (CNS)
 - astrocytes, functions 11
 - BDNF expression 333
 - BZD, impact 262
 - cannabis, psychoactive effects 263
 - CNS-related cognitive changes 399
 - development 1

- fetal development 275
- human prenatal CNS development,
 - organization 276
- inflammation 50
- lesions, inflammatory response 44
- neurons 5
- neurotransmitters, delineation 8
- central pontine myelinolysis 398–399
- central stress response neural network 27–30
 - elements 28
 - modulatory regulation 29
 - responsibility 29–30
- cerebral autosomal dominant arteriopathy
 - with subcortical infarcts and leukoencephalopathy (CADASIL) 205–206
- cerebral blood volume, exercise-induced
 - increase 333
- cerebral glucose metabolic rate (CGMR),
 - increase 1–2
- cerebral hypoperfusion 231
- cerebral perfusion 304
- cerebral tractography model, coronal section 112
- cerebral white matter, structural integrity 111
- cerebrospinal fluid (CSF)
 - anti-inflammatory cytokines, increase 53
 - circulation 11
 - fluid biomarkers 231
 - isotropic diffusion 93
- cerebrovascular disease 294
- cerebrovascular health, caloric restriction
 - (relationship) 257
- Chalder Fatigue Scale 47
- chemical synapse 6
- childhood maltreatment
 - cortisol response decreases, association 379
 - severity, amygdala-insula connectivity (negative relationship) 347
- Childhood Trauma Questionnaire (CTQ) 376
- cholinesterase inhibitors, usage 206
- chromatin, chemical modifications 83
- chromosome 6, dyslexia (correlation) 364
- chromosome 11, BDNF gene encoding 332
- chromosome 19, Notch-3 region (mutation) 205–206
- chromosome 21 variation 199
- chronic BDNF activation 38–39
- chronic traumatic encephalopathy (CTE) 227
 - biomarkers 231
 - clinical presentation 229–230
 - clinical research diagnostic criteria 230
 - diagnostic lesions, donation cases 229
 - early-stage CTE neuropathology 229
 - neuropathology 228
 - in vivo* detection 231
 - probable CTE/possible CTE diagnosis 230
 - p-tau PET ligands 231
 - RHI exposure, relationship 228–229
 - in vivo* biomarker 230
 - proposal 231
- cingulo-opercular/salience network 133
- circulating corticosteroids, sequestration 283
- clinically defined autistic individual, AS individual
 - (contrast) 190–191
- clinical research
 - connectomics 181
 - ERP method/applications 141
 - multimodal imaging techniques 181
- Clinician-Administered PTSD Scale
 - (CAPS-5) 245
- cocaine 265–266
 - acute effects 265
 - MRI studies, voxel-wise meta-analysis 265
 - use, deficits 265–266
 - user disorders, neuropsychological performance differences 265
- cognition
 - APOE genotype (relationship) 300–302
 - brain-derived neurotrophic factor (BDNF),
 - relationship 336–337
- cognitive behavioral therapy (CBT) 222
- cognitive deficits, prolongation 244
- cognitive empathy 70
- cognitive function 243
 - changes 255–256
- cognitive impairment, impact 44–45
- cognitive phenotyping, research opportunities 239–240
- cognitive processes, ERP components
 - (associations) 143–145
- cognitive reserve (CR) 293
 - active model 294
 - brain reserve, contrast 293–394
 - evidence 294
 - interventions 295
 - neural mechanisms 294–295
 - quantification 2904
- cognitive restructuring 223
 - treatment, importance 222
- comorbidity, diagnostic/neurobiological
 - signatures 243
- comorbid mild traumatic brain injury (comorbid mTBI), characteristics 246–247
- COMPAS-W scale 328

- composure, well-being sub-measure 328
- comprehension (information synthesis) 356
- compulsive behaviors, complexity 210
- conception, brain development 1
- concussion 243
 - pathophysiology 221
 - post-concussion syndrome (PCS) 222, 244
 - post-concussive disorder (PCD) 222
- connectivity strength, change 183
- connectome nodes, communication efficiency (measurement) 182
- connectomics 181
 - explanation 181–182
 - psychiatric disorders 182–184
- connexon 6
- context, hippocampus regulation 366
- contextual information, top-down processing 144
- contralesional cortical excitability, decrease 173
- contralesional hemisphere, inhibition (excess) 173
- contralesional M1 activity, suppression 175
- control cell survival, NAD-dependent deacetylases (impact) 258
- cornu Ammonis (CA) 99
- cornu Ammonis (CA) CA1
 - acute neuronal loss 103
 - network, information processing (impairment) 199
 - subiculum (boundary) 101
 - volume reductions 103
- cornu Ammonis (CA) CA3 101, 103
 - network, information process impairment 199
- corporal punishment, exposure (impact) 379
- cortical atrophy/thinning 231
- cortical cognitive phenotypes
 - neuropsychological assessment 237
 - neuropsychological performance, anatomical substrates 237–238
 - phenotype modeling, limitations 239
 - subcortical cognitive phenotypes, contrast 238–239
- cortical excitability
 - assessment, TMS (usage) 171–173
 - indexing/modulation, TMS (usage) 171
- cortical folding patterns, differences 142
- cortical gray matter regions, early involvement 238
- cortical surface area (SA), reduction 198
- cortical thickness (CT), reduction 198
- corticobasal syndrome (CBS) 209, 211
- corticospinal excitability 171
 - corticospinal function, interference 173
- corticosteroid-binding globulin (CBG), usage 283
- corticosteroids, glucocorticoid receptors (binding) 378–379
- corticosterone (CORT)
 - intercellular signal, importance 33
 - permissive effects 32
 - production 25–27
 - release 32
 - secretion 39
- corticotropin-releasing factor (CRF) 26–29
 - hypophysiotropic neurons, location 29
- corticotropin-releasing hormone (CRH), release 362
 - acute stressor, impact 378
 - increase 51
- coupling 277
- C-reactive protein levels
 - changes 255
 - decrease 254
- CSF A β 42, values (abnormality) 165
- cytokines
 - expression 50
 - proinflammatory cytokines, BBB transport 50
- DAT1 gene
 - dopaminergic function 67
 - ventromedial striatum activity 65
- declarative memories, formation (inability) 397
- default mode function, measurement 361–362
- default mode network (DMN) 133
 - activity, anticorrelation 303–304
 - connectivity, increase 304
 - differences 303–304
 - hyperconnectivity 134
 - normalization 136–137
 - results, rsFC results (comparison) 136
- default network (DN) 14
- delay discounting 66
- delta-9-tetrahydrocannabinol (Δ^9 -THC), psychoactive discovery 263
- δ -opioid receptors 61
- dementia
 - cause 120
 - glutamate, presence 153
 - glutathione, studies 157–158
 - NAA, presence 151
 - pathological processes, diffusional kurtosis imaging (usage) 117
 - risk, increase 254
- dendritic growth 282

- dendritic length/complexity, increase 279
- dentate gyrus (DG)
 - CA3/DG region, volume reductions 103
 - functional significance 37–38
 - neurobiology 199
- deoxyribonucleic acid (DNA)
 - methylation 75, 79
 - repair, NAD-dependent deacetylases (impact) 258
 - sequence variation, polymorphism 59
 - structure 60
- deoxyribonucleic acid methyltransferases (DNMTs) 75, 84
- level, elevation 84
- depression
 - 5-HTTLPR
 - association 316–318
 - meta-analyses 312–315
 - relationship 311
 - assessment 47
 - bullying, relationship 194
 - epigenetic mechanisms, impact 86
 - fatigue, relationship 46–47
 - GABA, impact 156
 - glutamate, presence 154
 - hippocampal subfield volume, effects 104
 - literature, examination 318
 - long-term depression (LTD)-like mechanisms, rTMS (mimic) 171
 - NAA, presence 151–152
 - neurogenic hypothesis 37
 - prediction, 5-HTTLPRxstress interaction (magnitude) 317
 - prenatal stress, relationship 281
 - traumatic brain injury (TBI), relationship 221–222
- deprivation-related stressor
 - caregiver abuse, contrast 375
 - effects 374
- developmental disorders, epigenetics (involvement) 75
- diathesis-stress theories 311
- Dietary Approaches to Stop Hypertension (DASH) diet 253
 - study 255, 256
- diffuse axonal injury (DAI), occurrence 221
- diffuse brain injury 221
- diffuse periventricular low density, CT scan display 398
- diffusion
 - analysis approaches 94
 - diffusion-weighted MR sequences 91
 - directionality, degree (index) 92
 - imaging, basics 91–94
 - measurement 91–92, 92
 - estimation 93
 - metrics, estimation 94
 - MRI 91, 117–118
 - probability distribution 118
 - tensor MRI, usage 220
 - tissue microstructure, relationship 119
- diffusional kurtosis, images 118
- diffusional kurtosis imaging (DKI) 117–119
 - usage 117
- diffusion tensor imaging (DTI) 109–110, 119, 302
 - advantages 244
 - factors, sensitivity 111
 - mTBI sensitivity 244
 - parameters 94–95
 - scalar metrics, derivation 110–113
 - sequence, parameters 92
 - studies 95
 - tractography metrics 109
 - usage 182, 198
- diffusion-weighted imaging (DWI) 91, 94–96
- dirty-appearing white matter (DAWM) 44
- disordered gambling 66
- distress, psychological response 278
- dopamine (DA) 8, 9
 - clearance, COMT (impact) 60
 - degradation, COMT (impact) 68
 - receptor candidate genes, investigation 65
 - release (stimulation), methamphetamine (impact) 267
 - system 59–60
 - transmission, mediation 60
- dopamine receptor D2 (DRD2) 67
 - DRD2 Taq1 allele, presence (impact) 65–66
 - gene 60
- dopamine receptor D4 (DRD4) 67
- dopamine transporter (DAT) density (reduction), methamphetamine (impact) 268
- dorsal anterior cingulate cortex (dorsal ACC)
 - activation 366
 - left anterior insula, functional connectivity (reduction) 347
- dorsolateral prefrontal cortex (DLPFC) 133
 - interhemispheric connectivity, demonstration 174–175
 - oculomotor loop signal source 17
 - seeds, usage 134

- Down syndrome (DS) (trisomy 21)
 - AD development risk 199
 - AD neuropathology, presentation 199
 - chromosome 21 variation 199
 - development, brain structure 198–200
 - neurobiology 197
- DRD2 Taq1A allele, presence 65
- Dutch Hunger Winter, PS exposure
 - (relationship) 280
- dysarthria 399
- dyslexia 193
 - chromosome 6, correlation 354
- early life deprivation, forms 374–375
- early life stress 373
 - animal models, limitations 376
 - behavioral sequelae 377–378
 - Bucharest Early Intervention Project 374
 - developmental timing 382–383
 - human studies 376
 - impact 377–378, 380–382
 - measurement 375–376
 - neurobiological sequelae 378–382
 - prevention/intervention, research 383–384
 - retrospective reporting, validity (debate) 376–377
 - reward process (abnormality), behavioral/neuroimaging studies (usage) 378
 - rodent models 376
 - sex differences 383
 - stress response, epigenetics 379–380
 - types, classification 373–375
- early-stage CTE neuropathology 229
- economic principle, application (failure) 191
- Einstein, Albert 91
- electrical synapse 6
- electroconvulsive therapy (ECT) treatment 156
 - response, structural connectome changes (association) 183
- electroencephalogram (EEG) 141
- electroencephalography (EEG)
 - asymmetry 382
 - usage 182
- electromyogram (EMG) 288
- electromyography (EMG), usage 171
- embryonic period 276
- emergent literacy skills 355
- emotion
 - emotion-perception relationships 193
 - regulation, development (abnormality) 378
- emotional development, programming 374
- emotional maternal withdrawal, exposure (impact) 379
- emotional Stroop test 346
- empathy
 - absence 188
 - increase 70
 - types 70
- engagement, construct 328
- entorhinal cortex volume, reduction 302
- environmental mastery (PWB dimension) 327
- ependymal cells 11
- epidural hematoma (EDH) 220–221
- epigenetic consequences stress 85
- epigenome
 - human epigenome, sensitivity 85
 - mental health, relationship 86
 - pharmacological alteration 85
- epigenomic remodeling, therapeutic implications 86
- epinephrine 8, 10
 - release 24
- episodic memory performance, ability 68
- epochs 142
- equifinality, developmental outcome (suggestion) 280
- eudaimonia (psychological well-being) 326
- event-related potential (ERP)
 - amplitude 142
 - amplitude, reduction 144–145
 - application (languages) 144
 - components 142–143
 - components, cognitive processes (associations) 143–145
 - data collection/analysis 142–143
 - index 377
 - latency 142
 - method/applications 141
 - post-acquisition data processing 142
 - scalp location 142
 - techniques, usage 143
 - usage 365
 - waveform, acquisition 142
- excitatory postsynaptic potential (EPSP) 7–8
- excitatory rTMS, application 173
- executive-associative loops 17
- executive control 366–367
- executive function
 - tests 255
 - variation 199–200
- executive functioning, detriments 245
- executive tasks, deficits 210
- experience, ML comprehension 164

- experimental autoimmune encephalomyelitis (EAE), MS animal model 43–44
- explosivity, CTE characterization 229
- externalizing problems, increase 280
- extra pontine myelinolysis 398–399
- extraversion 69
- facial electromyogram 288
- fasting insulin levels, decrease 254
- fast spin echo (FSE) scan, usage 100
- fatigue, depression (relationship) 46–47
- Fatigue Severity Scale (FSS) 47
- FDDNP 231
- fear conditioning dysregulation, disorder 245
- fear processing psychophysiology, traumatic stress (relationship) 289–290
- female interpersonal trauma survivors (PTSD presence), insula
 - functional connectivity 347
 - functional imaging 345–346
 - structural imaging 346
- fetal alcohol spectrum disorder (FASD) 78–79
 - gain-of-function studies 78–79
- fetal brain development
 - animal models 277
 - fetal physiological monitoring 277
 - study methods 277–278
- fetal heart rate (FHR) index, study 277
- fetal heart rate variability (FHRV) 277
- fetal magnetic resonance imaging (fetal MRI) 277–278
- fetal movement (FM) 277
- fetal period 276
- fetal physiological functioning/monitoring 277
- fetus, central nervous system (CNS)
 - development 275
- fiber bundle length (FBL)
 - DTI tractography metrics 109
 - mean FBL, age-related declines 113
 - metrics, computation 113
 - rationale 112–113
 - status, alteration 114
 - usage 113–114
- fight-or-flight response 289
- FK506 binding protein 5 (FKBP5), methylation (alteration) 85
- Flourishing Scale 326
- flow (construct) 328
- fludeoxyglucose (^{18}F -FDG) tracer, usage 305
- fluid-attenuated inversion recovery (FLAIR) 244–245
- fluid biomarkers 231
- fluorodeoxyglucose (FDG) PET data,
 - collection 199
- focal brain injury 220–221
- fractional anisotropy (FA) 92, 110
 - decrease/reduction 95, 111–112, 304
 - representation 111
 - TBSS projection 94
 - values, low level 93
- fragile X mental retardation 1 gene (FMR1),
 - hypermethylation 76
- fragile X syndrome 76
- free and cued selective reminding test (FCSRT) 164
- free nuclei, orientation 149
- FreeSurfer 5.3
 - automated hippocampus subfield segmentation protocol 102
 - usage 103
- FreeSurfer 6.0 segmentation algorithm,
 - usage 103–104
- FreeSurfer segmentation software, usage 101
- frontal cortex
 - alcohol-induced epigenetic modifications 79
 - methylation, decrease 86
- frontal lobe dementia 151
- frontal mismatch negativity (MMN),
 - reduction 144
- frontal parietal control network (FPN) 14
- frontoinsula regions, atrophy 210–211
- frontostriatal circuitry, dopaminergic
 - function 67
- frontotemporal dementia (FTD) 209
 - behavioral variant (FTD) (bvFTD) 209–210
 - caregiver burnout 214
 - clinical presentations 209–211
 - cognitive changes 210
 - epidemiology 212
 - frontal variant 238
 - neuropsychological assessment 212–213
 - PPA (subset) 210
 - safety considerations 213–214
 - syndromes 211–212
 - treatment planning 213–214
- frontotemporal lobar degeneration (FTLD) 209–212
- functional anatomy 5
- functional brain networks 303–304
- functional connectivity strengths, variance (increase) 182
- functional hypoactivity 231

- functional magnetic resonance imaging (fMRI)
 - 125, 302
 - clinical relevance 126–127
 - fMRI-derived connectome, defining 182
 - sequences, usage 245
 - task-based fMRI experiment/studies 125–126
- functional neuroanatomy, cortical-subcortical
 - distinctions/pathways 13
- future rewards/losses, prediction ability 64
- gain-of-function studies 78–79
- gamma-aminobutyric acid (GABA) (γ -aminobutyric acid) 8
 - binding, rapidity 262
 - concentrations, elevation 150
 - GABA/Cr ratios 155
 - GABAergic neurons 29
 - impact 155–156
 - release 15
 - signals 16
 - synthesis 2–3
 - target receptors 9
- gastrulation 276
- Gaussian diffusion 117
- Gaussian probability distribution 117
- gene polymorphisms, types 60
- generalization ability 164
- genetic polymorphisms 59
- genetics, study 59–61
- gene x environment interactions 331
- GIFT toolbox 133
- Glasgow Coma Scale (GCS) 220, 243
- glia 10–11
- globus pallidus, regions 14–15
- GluCEST 153
- glucocorticoid receptor (GR) 27
 - corticosteroids, binding 378–379
 - expression, decrease 379–380
 - gene (NR3C1), impact 380
 - methylation, increase 85
 - regulation, FKBP5 (impact) 85
- glucocorticoids
 - BDNF, relationship 38
 - release 378
- glucose homeostasis, improvement 257
- glucose metabolism
 - assessment, fludeoxyglucose (usage) 305
 - NAD-dependent deacetylases, impact 258
 - positron emission tomography (PET) 363
 - studies 364
- glutamate (Glu) 8–9
 - group-by-age associations 153
 - presence 152–154
- glutamate decarboxylase 1 (GAD1) (GAD67),
 - MeCP2 binding (increase) 77
- glutamine (Gln)
 - presence 152–154
 - storage 152
- glutathione (GSH)
 - concentrations, elevation 150
 - intracellular synthesis 156–157
 - LCModel measures 157
 - role 156–158
- glutathione disulfide (GSSG), formation 157
- glutathione peroxidase (GPx), catalysis 157
- glutathione reductase, usage 157
- G-protein-coupled receptors (GPRs), gene
 - expression action 8
- gray matter (GM)
 - volume, reduction 198
 - cocaine, impact 265
 - white matter junctions 238
- H¹ magnetic resonance spectroscopy,
 - methamphetamine studies 268
- happiness, measurement 326
- headache 246
- health, stress (impact/mechanisms) 32–34
- heart, acceleration 365
- heart rate (HR) 288
 - increase 362
 - measure, insensitivity 288
- heart rate variability (HRV) 288, 362
 - measure 289
- hepatic encephalopathy (HE) 399
 - CNS-related cognitive changes 399
- heroin use, report 268–269
- heterotypic stressor 30
- hexameric connexin proteins 6
- high-resolution MRI, usage 237–238
- high-resolution scans, ASHS protocol
 - (usage) 104
- high-resolution structural imaging 102
- high-resolution T2-weighted scan, coronal
 - slice 100
- hikikomoris 191
- hippocampal BDNF expression, investigation 333
- hippocampal body, high-resolution T2-weighted
 - scan (coronal slice) 100
- hippocampal functional connectivity strength,
 - increase 183
- hippocampal subfields
 - 3D reconstruction, Olsen-Amaral-Palombo
 - protocol (usage) 101
 - high-resolution structural imaging 102
 - structural neuroimaging 99

- hippocampus
 - alcohol-induced epigenetic modifications 79
 - anatomical survey 99–100
 - methylation, decrease 86
 - segmentation, gold standard 102
 - volume, reduction 102
- histone acetyltransferases (HATs) 84
- histone complex 83
- histone deacetylases (HDACs) 84
 - impact 75–76
 - silencing complex, protein interactions 77
- histone demethylases 84
- histone methyltransferases (HMTs) 84
- histones 75–76
 - methylation, effects 76
- Holocaust survivors, GR methylation
 - (increase) 85
- home literacy practices 355
- homotypic stressor 30
- HTR3A CC
 - genotype group, early life stress
 - (combination) 382
 - risk alleles, early life stress exposure 382
- hubness, measure 183
- hub nodes, influence (decrease) 182
- human BDNF gene, encoding 332
- human chromosomes, discovery 197–198
- human epigenome, sensitivity 85
- human immunodeficiency virus (HIV) 294
 - GABA, impact 156
 - glutamate, presence 154
 - infection 237
 - NAA, presence 152
- human prenatal CNS development,
 - organization 276
- humans, early life stress studies 376
- human tracer, accuracy 102
- Huntington's disease 238
- hydrogen-containing groups, proximity 150
- hyperarousal 134
 - symptoms 287
- hyperorality 210
- hyperphosphorylated tau protein, intracellular
 - accumulation 304–305
- hypothalamic-pituitary-adrenal (HPA)
 - dysregulation 282
 - sensitivity, reduction 379
- hypothalamic-pituitary-adrenal (HPA) axis
 - 25–27, 26 29,
 - activation 22
 - activity, increase 85
 - acute stressor activation 378
 - dysregulation 51
 - function 319–320
 - interruption 84
 - PTSD disruption 245–246
 - functioning, alterations 279
 - hyperreactivity, exhibition 31
 - neuronal component 26–27
 - response 31
 - stress response system, impact 33–34
- hypoxia (oxygen deficiency), prolongation 283
- IFITM genes, increase 54
- immune activation, differences 52
- immune markers, psychopathology (relationship)
 - 49, 50–51
- impulsivity 70, 230
 - CTE characterization 229
- independent component analysis (ICA) 132–133
 - usage 135
- inertia 210
- inflammation 49–50
 - psychiatric conditions, relationship 51–54
- inflammatory lesions, T cells (presence) 43–44
- inhibitory postsynaptic potential (IPSP) 7–8
- inhibitory rTMS, application 173
- insomnia 246
- institutionalization, neglect (comparison) 375
- insula
 - amygdala-insula connectivity 347
 - functions 343–344
 - left anterior insula, dorsal anterior cingulate
 - cortex (functional connectivity,
 - reduction) 347
 - level 341, 343
 - posttraumatic stress disorder (PTSD),
 - relationship 344–345
- insulin
 - resistance, changes 255
 - secretion, NAD-dependent deacetylases
 - (impact) 258
 - sensitivity, enhancement 257
- insulin-like growth factor (IGF-1) expression,
 - enhancement 257
- integrity
 - neuronal integrity, NAA measure (surrogate
 - marker) 153
 - structural coherence 109–110
- intelligent algorithms, AD diagnosis
 - suitability 165
- intentional weight loss, examination 255–256
- interferon gamma (IFN- γ), serum levels
 - (increase) 52
- interhemispheric competition model (IHCM)
 - 173, 175

- interhemispheric connectivity, demonstration 174–175
- interhemispheric inhibition (IHI) 171, 173
- interleukin-6 (IL-6)
 - BBB transport 50
 - concentrations, elevation (ALSPAC birth cohort) 53
 - injections, impact 284
 - levels, increase 51
 - serum levels, increase 52
- internalizing problems, increase 280
- interparental aggression, exposure (impact) 379
- intimate partner violence (IPV), PTSD examination 345
- intracortical excitability, alteration 171
- intracranial hematoma 221
- intracranial pressure, increase 220
- introversion 69
- intrusion symptoms 287
- ionotropic receptors 8
- ipsilesional cortex, neuroplasticity (interference) 173
- ipsilesional cortical excitability, increase 173
- ipsilesional hemisphere, cortical repair/ augmentation target location 174
- ipsilesional M1
 - excitability, decrease 173
 - obliteration 175
- ischemic stroke
 - kurtosis/diffusivity metrics, complementary characterization 119
 - occurrence 119
- j*-coupling 150
- kainate 8
- κ -opioid receptors 48 61,
- ketamine, studies 152–153
- KIAA0319 (chromosome 6), dyslexia (correlation) 354
- kiss and run fusion 7
- Korsakoff syndrome (KS) 396–398
- kurtosis
 - orientational dependence 118
- language
 - genetic/neurobiological factors (highlighting), etiological studies (usage) 354–355
 - language-action relationships 193
 - literacy development 353
 - literate language 356
 - processing, ERP application 144
 - proficiency, NICHD Early Child Care Research Network establishment 353
- large vessel cerebral infarcts, VaD case percentage 206
- latency jitter 142
- lateral geniculate nucleus (LGN) 14
- lateral superior parietal cortices 133
- LCModel, usage 157, 158
- learning, rewards (reinforcing role) 66–67
- left anterior insula, dorsal anterior cingulate cortex (functional connectivity, reduction) 347
- left dorsolateral prefrontal cortex (left DLPFC), activation 364
- left parietal lobe, blood oxygenation level-dependent (BOLD) activation 127
- left superior/middle frontal gyri, blood oxygenation level-dependent (BOLD) activation 127
- leptin, changes 255
- level of consciousness (LOC) 219
- Lewy body dementia 151
- life satisfaction (SWB component) 326
- limbic loop 17
- limbic system
 - functions 380
 - prefrontal cortex, connectivity 381
- lipid regulation, NAD-dependent deacetylases (impact) 258
- lipid transporter, APOE encoding 299
- lipopolysaccharide (LPS), injection 50, 55
- literacy
 - book reading, correlation 355
 - development 353
 - emergent literacy skills 355
 - home literacy practices 355
- literate language 356
- long-term depression (LTD)
 - LTD-like mechanisms, rTMS (mimic) 171
 - pro-BDNF facilitation 332
- long-term potentiation (LTP)
 - mimic 171
 - promotion 331
- LORETA techniques 364
- loss of consciousness (LOC) 243
- M1 function, control 174
- machine learning (ML) 164
 - research phase 165
- magnetic resonance imaging (MRI) 164
 - arterial spin labeling (ASL) 302
 - fetal magnetic resonance imaging (fetal MRI) 277–278

- functional magnetic resonance imaging (fMRI) 125–127, 245, 302
- high-resolution MRI, usage 237–238
- magnetic field (MF)/radiofrequency (FF) pulse, usage 277
- parameters 93
- resting-state functional magnetic resonance imaging (rs-fMRI) 131
- segmentation protocols, development 100
- technique 91
- magnetic resonance spectroscopic imaging (MRSI) 150
- magnetic resonance spectroscopy (MRS) 149
 - brain chemicals, relationship 150–152
 - methods 150
 - physics 149–150
 - studies 155
 - types 150
- major depression 133
- major depressive disorder (MDD) 49
 - diagnosis 37
 - ERP amplitudes, reduction 144–145
 - FKBP5, impact 85
 - glutamate, presence 154
 - hippocampal subfields, structural neuroimaging 99
 - inflammation, relationship 51–52
 - rs-fMRI, usage 134
- Marchiafava-Bignami disease (MBD) 398
- mastery (well-being sub-measure) 328
- maternal depressive symptoms, increase 281
- maternal endocrine system, PS effects
 - mechanism 282
- maternal immune system, PS effects mechanism 282–283
- maternal-placental-fetal unit, complexity 283
- maternal thyroid hormone (maternal TH), impact 282
- maternal vascular system, PS effects
 - mechanism 283
- mathematical representations, usage 111
- mature BDNF, impact 332
- MDMA (Ecstasy) 266–267
 - neuropsychological sequelae, meta-analyses 266–267
 - use initiation, effects 267
- mean diffusivity (MD) 92, 95, 110
 - defining 119
- mean FBL, age-related declines 113
- meaning (construct) 328
- mean kurtosis (MK) 118
- medial orbitofrontal cortex (mOFC), activation (increase) 65
- medial prefrontal cortex (MPFC) 133
 - inhibitory pathway, impact 364
 - projections 362
 - reactivity 290
- medial temporal lobe (MTL) 228
 - activation, increase 303
 - volume loss 302
- medicine, artificial intelligence (usage) 163
- MeDI diet, receiving 256
- Mediterranean diet, examination 253
- MEGA-PRESS 150, 155, 157
- memory
 - declarative memories, formation (inability) 397
 - impairment, exhibition 239
 - performance, age-related decline 68
 - procedural memory, disruption (reduction) 397
 - semantic memory, disruption (reduction) 397
 - subtypes (distinction), ERP techniques (usage) 143
 - tests, decline 67–68
- mental health
 - epigenetics, relationship 83
 - epigenome, relationship 86
 - stress, impact 33–34
- mental state, alteration 219
- mental well-being, neural networks 361–367
- metabotropic glutamate receptors (mGluRs) 8
- metabotropic receptors 8
- Met allele, risk factor identification 335–336
- methadone maintenance therapy (MMT) 269
- methamphetamine 267–268
 - acute effects 267
 - lipophilicity 267
 - long-term use, effects 267–268
- methionine (Met) substitution 332
- methylation 83
- methyl-binding domain genes, patterns (disruption) 77
- methyl-CpG-binding protein 2 (MeCP2)
 - binding 77
 - Sin3/HDAC complex, interaction 75
- Met/Met carriers, sensitivity 64
- microangiopathy, involvement 206
- microglia 11
 - role/function 3
- middle adulthood, APOE genotype 301
- middle temporal gyrus, right hippocampus (extension) 134
- mild cognitive impairment (MCI) 103, 151
 - Alzheimer's disease, relationship 301–302
 - classification 120
 - conversion 164
 - resting hyperperfusion 304

- mild traumatic brain injury (mTBI)
 - 135–136, 219
 - comorbidity, diagnostic/neurobiological signatures 243
 - diagnosis/characteristics 243–245
 - neuroimaging studies 247
 - pathophysiology 221
 - treatment 222–223
- military populations, mTBI/PTSD trauma
 - exposure (risk) 246
- mineralocorticoid receptor (MR) 27
 - expression 378–379
- Minimal Assessment of Cognitive Function in MS (MACFIMS) 45
- Mini-Mental State Examination (MMSE) 45
- mismatch negativity (MMN), reduction 144
- mode of anisotropy (MO) 92
- Modified Fatigue Impact Scale (MFIS) 47
- monoadenosine diphosphate-ribosyl transferases, impact 258
- monoamine oxidase (MAO)
 - 3-copy allele MAOA polymorphism, allelic frequency 66
 - deficiency 70
 - enzymes, types/impact 61
 - MAOA-uVNTR tandem repeats 70
- monoamines 8
- Montreal Cognitive Assessment (MoCA) 45
- mood
 - disorders, connectomics 183
 - functioning, serotonin (involvement) 51
 - stress/neurogenesis, relationship 37
- motor-evoked potential (MEP) 171
- motor neuron disease (MND) 209, 211
- motor recovery, improvement 171
- Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) 383–384
- multifinality, principle 280
- multimodal imaging techniques 181
- Multiple Automatically Generated Templates
 - Brain Segmentation Algorithm (MAGet-Brain) 102
- multiple sclerosis (MS)
 - cognitive changes, radiographic changes (correlation) 46
 - cognitive impairment 44–46
 - cognitive rehabilitation 46
 - disease-modifying therapies 46
 - inflammation, occurrence 43
 - neuropsychiatric symptoms/pathogenesis 43
 - pathophysiology 43–44
- multipolar neurons 5
- multivariate analysis 164
- multivariate ML techniques, usage 164
- muscarinic acetylcholine receptor (mAChR) 10
- muscle control, impairment (dysarthria) 399
- μ -opioid receptor gene (OPRM1), impact 61, 65
- myelin
 - changes, sensitivity 111
 - sheaths 6
- myelinated vagus 289
- myelination 282
 - problem 198, 276
- N400 component 142, 144
- N-acetylaspartate (NAA)
 - concentrations, reduction 268
 - distribution 150–151
 - NAA/Cr, presence 152
 - presence 151–152
- National Institute of Child Health and Human Development (NICHD) Early Child Care Research Network, language proficiency establishment 353
- N-back paradigm 126
- necrotic cell death 276
- negative feedback mechanisms, usage 379
- negative mood/cognitions 287
- negative personality traits 70
- neglect, institutionalization (comparison) 375
- neocortex 99
- neonatal stress exposure (rats/mice), impact 32
- nerve growth factor (NGF) 331
- network alterations, schizophrenia/hallucination
 - symptoms (relationship) 1832
- network strength, reduction 182
- neural circuitry, early life stress (impact) 380–382
- neural compensation 295
- neural migration 276
- neural networks 361
 - reward 363–366
 - threat 362–363
- neural plate 276
- neural progenitor cells, emergence 276
- neural reserve 294–295
- neural switchboard areas 366
- neural tube, formation 276
- neurite orientation dispersion and density
 - imaging (NODDI) 93, 93
- neurocognitive performance (improvements), weight loss (association) 255
- neurodegenerative conditions (FTD) 209
- neurodegenerative disease 228

- neurodevelopmental disorders, prevalence rates (increase) 280
- neuroendocrine system 61
- neuroexocytosis 6–7
- neurogenerators 142
- neurogenesis 276, 282
 - stress/mood, relationship 37
 - stress response, relationship 38
- neurohumoral/growth factor modulation, caloric restriction (relationship) 257
- neuroinflammation, implication 396
- neurological deficits 219
- neurometabolic cascade 221
- neuronal damage (etiology), cognitive testing (usage) 238
- neuronal disruption 238
- neuronal integrity, NAA measure (surrogate marker) 153
- neuronal migration 282
- neurons
 - classification 5
 - information transfer, interface 5–6
- neuroplasticity, glutamate (role) 154
- neuropsychiatric conditions, resting-state functional magnetic resonance imaging (usage) 133–136
- neuropsychiatric function, BDNF (relationship) 335–336
- neuropsychological functioning, cocaine (acute effects) 265
- neuroticism, amygdala (relationship) 318–319
- neurotransmitters (NTs) 8–10
 - genetic variations 65
 - synthesis 2–3
 - systems, genes (association) 62–64
- neurotrophin-3 (NT-3) 331
- neurotrophin-4/5 (NT-4/5) 331
- neurulation 276
- nicotinamide adenine dinucleotide (NAD), NAD-dependent deacetylases, impact 258
- nicotine dependence, 5-HTTLPR L allele (relationship) 69
- nicotinic acetylcholine receptor (nAChR) 10
- N*-methyl-*D*-aspartate (NMDA) 8–9, 46
 - receptor agonists, studies 152–153
 - receptors, dysfunction 152–153
- N*-methyl-*D*-aspartate (NMDA) glutamate receptor
 - inhibition 65
 - reversal potential 7–8
- nodes of Ranvier 6
- nonfluent variant PPA (nfvPPA), characterization 210–211
- non-Gaussianity, degree (measure) 117
- nonhuman primates, early life stress 376
- non-isotropic T2-weighted FSE scan, usage 100
- norepinephrine (NE) 10
 - production 2–3
- normal-appearing white matter (NAWM) 44
- normal pressure hydrocephalus 206
- nuclear spin, alteration 150
- nucleus accumbens (NAcc) 14, 17
 - DNMT3A, presence 85
- nucleus ambiguus (NA) 289
- obesity paradox 254
- obsessive-compulsive disorder (OCD)
 - BDNF levels, reduction 335
 - brain network characteristics, SSRI pharmacological treatment, impact 184
 - characterization, ERPs (usage) 145
 - connectomics measures treatment, impact (studies) 184
- oculomotor loop 16–17
- off-axis diffusivity 110
- official entity, transient life 189
- offspring
 - birth outcomes, PS effects 280
 - development, prenatal stress (impact) 275, 278–281
 - regulatory systems, PS effects mechanism 284
- older adulthood, APOE genotype 301
- older adults, FA decrease 95
- olfactory tubercle 14
- oligodendrocytes 11
 - differentiation, gene regulation 198
- Olsen-Amaral-Palombo protocol, usage 100, 101
- opiates 268–269
 - neuropsychological impairments 269
 - use, neuropsychological consequences 269
- opioid system 61
- orbital prefrontal cortex (OFC) 363
- orbitofrontal cortex (OFC), engagement (reduction) 67
- Osmotic demyelination syndrome (ODMS) 398–399
- otakus 191
- own-worth (well-being sub-measure) 328
- oxidative stress, caloric restriction (relationship) 257–258
- Oxycontin, usage (national crisis) 269
- oxygen deficiency, prolongation 273

- oxytocin receptor (OXTR) gene
 - epigenetic regulation 77
 - impact 61, 65
 - methylation 85
 - SNP rs53576 G allele, empathy (increase) 70
 - SNPs 70
- p75 receptor, activation 332
- Paced Auditory Serial Addition Test (PASAT) 47
- Paced Visual Serial Addition Test (PVSAT) 47
- paired helical filament (PHF) tau isoforms 231
- paired pulse rTMS
 - measures 172
 - usage 174
- panic disorder (PD), (characterization), ERPs
 - (usage) 145
- parallel diffusivity 92, 95, 110
- parasympathetic nervous system (PSNS) 24–25
- paraventricular nucleus (PVN) 26–29
- Parkinson's disease 206, 238, 294
- pathological processes (defining), diffusional
 - kurtosis imaging (usage) 117
- PATH Through Life project cohort 301
- performance IQ (PIQ) 191
- peripheral BDNF, examination 335
- perpendicular diffusivity 92, 95, 110
- personal growth (PWB dimension) 327
- personality traits, Met allele risk factor 335–336
- personalized therapeutic rTMS application 175
- phencyclidine, studies 152–153
- phenotype modeling, limitations 239
- phonemic awareness 356
- phonics 356
- phonological awareness 355
- phosphorylation 83
- physical/verbal violence, CTE
 - characterization 229
- physiological health, stress (impact) 32–33
- physiological reactivity 287
- physiological stress response
 - forebrain integrator 29
 - systems 23
- physiology, cell types 5
- physioneurosis 287
- placental corticotrophin-releasing hormone
 - (pCRH), production/impact 283
- placenta, PS effects mechanism 283–284
- plasma A β data, collection 199
- pleasant affect (SWB component) 326
- PMd, interhemispheric connectivity
 - (demonstration) 174–175
- polarity 142
- polymorphisms 59
 - behavior, relationship 61–71
 - gene polymorphisms 60, 62–64
 - variable number tandem repeat (VNTR)
 - polymorphisms 60
- polyvagal theory, traumatic stress
 - (relationship) 289
- positive emotion (construct) 328
- positive relations with others (PWB
 - dimension) 327
- positivity (well-being sub-measure) 328
- positron emission tomography (PET),
 - usage 237–238
- post-acquisition data processing 142
- post-concussion syndrome (PCS) 244
 - risk 222
 - symptoms 246–247
- post-concussive disorder (PCD)
 - behavioral health intervention 222
 - diagnostic use 222
 - symptom management 222
- posterior cingulate/retrosplenial cortex
 - (PCC) 133
- posterior cortical atrophy 238
- posterior parietal lobe, oculomotor loop signal
 - source 17
- postnatal stress, epigenetic consequences 85
- posttraumatic amnesia (PTA), alteration 243
- posttraumatic stress disorder (PTSD) 49, 133, 222
 - avoidance symptoms 346
 - characteristics 246–247
 - characterization, ERPs (usage) 145
 - comorbidity, diagnostic/neurobiological
 - signatures 243
 - conceptualization 288
 - development, conditional risk (increase) 342
 - diagnosis/characteristics 245–246
 - dissociative symptoms 135
 - explanatory models 288
 - FKBP5, association 85
 - frontoparietal CEN, focus 135
 - inflammation, relationship 54
 - insula
 - level 341, 343
 - relationship 344–345
 - learning conceptualization 290
 - neuroception, role 289
 - neuroimaging studies 247
 - psychophysiological studies, paradigms
 - (usage) 288
 - psychophysiology 287
 - developments 288

- rsFC, exhibit (diminishment) 135
- rs-fMRI, usage 134–135
- sex differences, scientific premise 342–343
- symptom presentations, BOLD signals (relationship) 344–345
- volumetric MRI studies 246
- posttraumatic stress disorder (PTSD) Symptom Scale—Interview (PSS-I-5) 245
- Prader-Willi Syndrome, clinical features 76–77
- prediction errors (PE) 66, 347
- prefrontal cortex (PFC) 362–363
 - activation 64
 - COMT expression 68
 - limbic system, connectivity 381
 - top-down/bottom-up connections 366
- PREMEDI study 256
- premotor cortices, impact 174
- prenatal stress (PS) 373
 - epigenetic consequences 84
 - impact 275
 - mental health outcomes, relationship 84
 - offspring sex/intensity/timing, impact 280–281
 - resilience/intervention 284
 - in utero* exposures 280
- prenatal stress (PS) effects 278–281
 - animal studies 278–279
 - correlational human studies 280–281
 - mechanisms 281–284
 - mediation, maternal thyroid hormone (impact) 282
 - quasi-experimental human studies 279–280
- PRESS, usage 157
- primary progressive aphasia (PPA) 165, 209
 - FTD subset 210
 - nonfluent variant PPA (nfvPPA), characterization 210–211
 - semantic variant PPA (svPPA) 211
- primary somatosensory cortex (S1), interhemispheric connectivity (demonstration) 174–175
- print concepts, knowledge 355–356
- probabilistic tractography, usage 111–112
- pro-BDNF, components 332
- procedural memory, disruption (reduction) 397
- prodynorphin (PDYN)
 - gene, impact 61, 65
 - nucleotide tandem repeat functional polymorphism 67
- progressive muscle relaxation procedures 222
- progressive supranuclear palsy (PSP) 211
- progressive supranuclear palsy (PSP-S) 209, 211
- proopiomelanocortin (POMC) gene, methylation 78
 - decrease 85
- proton magnetic resonance spectroscopy (^1H MRS) 149, 152
- pseudounipolar neurons 5
- psychiatric consultation, usage 222
- psychiatric diseases, magnetic resonance spectroscopy (MRS) 149
- psychiatric disorders, connectomics 182–184
- Psychiatric Genomics Consortium, Schizophrenia Working Group 71
- psychological stressors, response (ability) 22–23
- psychological well-being (PWB) 326–327
 - dimensions 327
- psychoneuroimmunology 49
- psychopathology, immune markers 49, 50–51
- p-tau PET ligands 231
- punch drunk, term (usage) 227
- purpose in life (PWB dimension) 327
- putamen, interconnection 14
- quality of life (QOL) measures, cognitive deficits (relationship) 45
- radial diffusivity (RD) (perpendicular diffusivity) 92, 95, 110
 - representation 111
- radial glial cells 10
- radial kurtosis 118
- radiofrequency (RF) volume head coil, patient location 149–150
- rape, PTSD development 342
- reactive oxygen species (ROS), reactions, catalysis 156
- readily releasable pool (RRP) 6–7
- reading
 - difficulties, heritable component 354
 - instruction, critical components 355–356
 - specificity, examples 353–354
- reelin (RELN) 276
 - MeCP2 binding, increase 77
 - methylation, increase 86
- reexperiencing 134
 - symptoms 287
- regional brain volumes, MRI (usage) 302–303
- region of interest (ROI)
 - analysis, tractography 94
 - anatomical ROI, three-dimensional images 118
 - seed ROIs, selection 133
- reinforcement learning models 66

- repetitive head impacts (RHIs)
 - chronic traumatic encephalopathy (CTE), relationship 228–229
 - long-term consequences 227
- repetitive transcranial magnetic stimulation (rTMS)
 - individualized approach, requirement 174–175
 - measures 172
 - personalized therapeutic rTMS application 175
 - therapeutic approach 173–174
 - therapeutic effects 169
- Research Domain Criteria (RDoC) 288
- respiratory sinus arrhythmia (RSA), noninvasive monitoring 289
- resting physiological state, paradigm (usage) 288
- resting-state functional connectivity (rsFC) 131
 - alteration 136
 - methods 132–133
 - outcome measure (clinical studies) 136–137
- resting-state functional magnetic resonance imaging (rs-fMRI) 220
 - background 132
 - methods/analytic techniques 132–133
 - neuropsychiatric conditions, relationship 131
 - preprocessing methods 132–133
 - task-based fMRI, comparison 133
 - usage 133–136
- restricted interests and repetitive behaviors (RIRB) 189, 191
- Rett syndrome, clinical features 77
- revised Brief Visuospatial Memory Test (BVMT-R), usage 45
- reward
 - neural networks 363–366
 - process (abnormality), behavioral/neuroimaging studies (usage) 378
 - processing 61–66
 - amygdala/PCC activity, relationship 364–365
 - development, abnormality 378
 - dopamine neurons, impact 363
 - system, well-being (associations) 363–364
- rich club nodes, density (reduction) 182
- right dorsolateral prefrontal cortex (DLPFC), positive images (engagement) 363–364
- right hippocampus volume, reduction 302
- right inferior prefrontal cortex (PFC) 363
- right medial orbital prefrontal cortex (OFC) 363
- rs25531 G allele, occurrence 316
- salience network (SN) 133
 - normalization 136–137
- saliva, fluid biomarker 231
- satisfaction (well-being sub-measure) 328
- Scale of Positive and Negative Experience (SPANE) 326
- schizoid personality 192
- schizophrenia 49, 70
 - connectomics 182–183
 - delusional and vague wooliness 192
 - development, risk 53
 - diagnosis, machine learning algorithm
 - ability 144
 - GABA, impact 155
 - glutamate, presence 152–153
 - glutathione, presence 157
 - hippocampal subfields, structural neuroimaging 99
 - hippocampus subfield volumetry studies 103–104
 - inflammation, relationship 52–54
 - Met allele risk factor 335–336
 - NAA, presence 151
- Schizophrenia Working Group (Psychiatric Genomics Consortium) 71
- secondary brain injury 221
- second-order tensor, ADC values (expression) 110
- seed-based connectivity 132–133
- seed-based rsFC analysis, dlPFC seeds (usage) 134
- selective serotonin reuptake inhibitors (SSRIs) 10
 - pharmacological treatments 184
 - treatments 156
 - upregulation (BDNF expression) 51
- self-acceptance (PWB dimension) 327
- self-reported empathy 70
- Selye, Hans 21–22
- semantic dementia 238
- semantic memory, disruption (reduction) 397
- semantic variant PPA (svPPA) 210, 211
- sensory impairments 193
- serotonin (5-HT) (5-hydroxytryptamine) 8, 9–10
 - mood functioning involvement 51
 - system 60–61
- serotonin transporter gene (SLC6A4) 85, 315
 - expression
 - comparison 316
 - decrease 318
 - methylation, increase 86
- serotonin transporter-linked polymorphic region (5-HTTLPR)
 - alexithymia, relationship 70
 - depression

- association 316–318
 - meta-analyses 312–315
- DRD2/ANKK1, interaction 69
- gene 61
- literature, examination 318
- neural development 319
- orthologue, cross-species investigation 315
- polymorphism 67, 71, 315–316
 - stress/depression, relationship 311
- short allele 316
- stress interaction, meta-analyses 312–315
- stress-related depression, associations (neural mechanisms) 318–319
- serotonin transporter (5-HTT), reuptake protein function 315
- SERPINA3 genes, increase 54
- serum spinal fluid (CSF) anti-inflammatory cytokines, increase 53
- SHANK3, autism etiology (relationship) 77–78
- SIN3 gene, transcriptional regulatory protein encoding 75
- single nucleotide polymorphisms (SNPs) 59, 70, 299
 - investigations 332
- single-photon emission computed tomography (SPECT) 247
- single rTMS
 - measures 172
 - usage 174
- single-voxel MRS (SV MRS) 150–151
- skeletomotor loop 15–16
- skin conductance (SC) 288
 - measure, insensitivity 288
- SLC6A4 (serotonin transporter gene)
 - impact 85
 - methylation, increase 86
- slowed/foggy thinking 246
- social anxiety disorders 70
- social development, programming 374
- social functioning, deficits 70
- social impairment, basis 192–193
- social rules, misunderstanding 193
- soluble *N*-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) 6
- sortilin binding 332
- source-localization LORETA techniques 364
- source modeling, usage 365
- speech
 - apraxia 210
 - comprehension, agrammatism (impact) 213–214
- speech onset delay (SOD) 190–191
- startle reactivity 288
 - increase 287
- statistical analysis 94
- STEAM 157
- stratum radiatum, lacunosum, and molecular (SRLM) 102
 - visualization 100
- streamtubes 111
- street drugs, substance abuse 261
- stress
 - 5-HTTLPR interaction, meta-analyses 312–315
 - 5-HTTLPR, relationship 305
 - animal studies, focus 376
 - body response, neurobiology 23–30
 - concept 21–22
 - conceptualization 278
 - developmental/lifespan factors 31–32
 - developmental timing 382–383
 - diathesis-stress theories 311
 - early life stress 373
 - exposure 317–318
 - literature, examination 318
 - neurogenesis/mood, relationship 37
 - reactivity (increase), neonatal stress exposure (impact) 32
 - sex differences 31
 - stress-health relationships, neurobiology 21
 - stress-related depression, 5-HTTLPR association (neural mechanisms) 318–319
- stress hyporesponsive period (SHRP) 382
- stressors
 - defining 373
 - global exposure 374
 - types 22–23, 30
- stress response 378–379
 - adaptation 30–31
 - epigenetics 37–380
 - habituation, impairment 30–31
 - neural pathways 24
 - neurogenesis, relationship 38
 - regulation 30–32
 - negative feedback mechanisms, usage 379
 - types 23
- striatum 14
 - activation 64
 - positive images, engagement 363–364
- stroke
 - cortical excitability/connectivity (assessment), TMS (usage) 171–173
 - ischemic stroke, occurrence 119

- stroke (*cont'd*)
- pathological processes, diffusional kurtosis imaging (usage) 117
 - personalized therapeutic rTMS application 175
 - recovery research, M1 focus 174
 - repetitive transcranial magnetic stimulation (rTMS), therapeutic effects/approaches 169–175
 - research, DKI (contributions) 119–120
 - upper limb impairment, change 173
- structural brain abnormalities 198–199
- structural coherence (integrity) 109–110
- Structured Clinical Interview for DSM-5 (SCID-5) 245
- study inclusion, usage 317
- subcortical cognitive phenotypes
- cortical cognitive phenotypes, contrast 238–239
 - memory impairment, exhibition 239
 - neuropsychological assessment 237
 - neuropsychological performance, anatomical substrates 237–238
 - phenotype modeling 239
- subdural hematoma (SDH) 220–221
- subgenual cingulate distinguished rTMS responders, centrality 183
- Subjective Happiness Scale (SHS) 326
- subjective well-being (SWB) 326
- components 326
- substance abuse, street drugs 261
- substance use disorders, Met allele risk factor 335–336
- substance users, characteristics 261–262
- substantia nigra 14
- subthalamic nucleus (STN) 14, 15
- SUMOylation 83
- superior frontal gyrus, CR-related benefits 295
- supplementary motor area (SMA) 174
- surface deformation analysis 102
- surface electromyography (EMG), usage 171
- susceptibility-weighted imaging (SWI), usage 244–245
- Sylvian fissures, operculum 221
- symbol digit modalities test (SDMT), usage 45
- sympathetic nervous system (SNS) 24–25, 29
- activation, impact 33
 - activity, increase 23
 - homeostasis function 21–22
 - output neurons, direct neural control 28
 - physiologic response 23
 - preganglionic neurons, primary inputs 25
 - stress response system, impact 33–34
- synapse 5–8, 276
- interfaces, types 6
 - synaptic transmission 6–8
 - synaptic vesicle exocytosis/recycling 6–7
- synaptic pruning 276
- synaptic serotonin, duration/magnitude (5-HTT regulation) 315
- synaptogenesis 282
- syndromic autism 192
- T2-weighted clinical imaging, visualization threshold 112–113
- TAR DNA-binding protein 43 (TDP-43), deposition (excess) 228
- task-based fMRI
- experiment/studies 125–126
 - rs-fMRI, comparison 133
- tau-protein neurodegeneration 163
- Tcl mouse model 199
- TDAH. *See* attention-deficit hyperactivity disorder.
- TE-averaged PRESS 154
- TE-averaged SV MRS 150
- teenagers, GR gene methylation (increase) 85
- ten-eleven translocation (TET), protein family 84
- tensor-based approaches 112
- tetrahydrocannabinol (THC), psychoactive effects 263
- thalamus 13–14
- dysregulation 17
- theory of mind 213
- thiamine, use 397
- thoracolumbar spinal cord preganglionic neurons 28
- thought-action repertoire 365
- threat
- neural networks 362–363
 - perception, early life stress (impact) 377–378
 - sensitivity, development (abnormality) 378
 - threat-related stressors, effects 374
- three-dimensional diffusivity, usage 92
- thyroid hormone (TH), PS effect mediation 282
- time-limited cognitive behavioral therapy (time-limited CBT) 222
- tract-based spatial statistics (TBSS) 94
- tractography 94
- accomplishment 111–112
 - color-coding convention 111
 - DTI tractography metrics 109
 - model, generation 112–113
- tract, structural integrity 112
- trait empathy 70

- transcranial magnetic stimulation (TMS) 169, 170–171
 - repetitive transcranial magnetic stimulation 169
 - schematic 170f
 - usage 171–173
- transcriptional regulatory protein, encoding 75
- trauma
 - cues, responses 288
 - trauma-related stimuli, avoidance 134
- traumatic axonal injury (TAI)
 - occurrence 221
 - shearing forces 244
- traumatic brain injury (TBI) 133, 219, 230
 - cognitive restructuring 223
 - definition/classification 219–220
 - depression, relationship 221–222
 - evaluation, tools 220
 - evidence 220
 - functional neuroanatomy 220–221
 - mechanisms 220
 - mild traumatic brain injury (mTBI) 136
 - motor vehicle crashes, impact 220
 - severity indicators 220
 - symptom management 223
- traumatic encephalopathy syndrome (TES) 230
- traumatic stress
 - fear processing psychophysiology, relationship 289–290
 - polyvagal theory, relationship 289
 - psychophysiology 287
 - developments 288
- tropomyosin receptor kinase B (TrkB), BDNF binding 332
- truncated BDNF, cognitive performance predictor 336
- Ts65Dn mouse model 197, 199
- tumor necrosis factor- α (TNF- α) 50
 - levels, increase 51
 - serum levels, increase 52
- turbo spin echo (TSE) scan, usage 100
- two-dimensional (2D) MRS 150
- ubiquitin-protein ligase E3A (UBE3A), silencing 76
- ubiquitylation 83
- UCLA Life Stress Interview 276
- UK Biobank, brain imaging data collection goal 96
- Uncinate fasciculus, white matter tract development 281
- undoing hypothesis 365
- unmyelinated vagus 289
- unpleasant affect (SWB component) 326
- unsaturated fatty acids (UFAs), intake (increase) 254
- untranslated regions (UTRs) 332
- vagal break 289
- vagal systems, identification 289
- vagus nerve 362
- Val66Met 67–71, 332
- Val66Met SNP
 - brain structure, relationship 334
 - focus 333
 - mediator status, determination 333
 - meta-analyses, failure 335
- valine (Val)
 - involvement 332
 - Val allele carriers, fiber bundles 334
- variable number tandem repeat (VNTR)
 - polymorphisms 60, 65
- vascular dementia (VaD) 151, 205
 - cholinesterase inhibitors, usage 206
 - large vessel cerebral infarct, percentage 206
 - risk, increase 254
 - subtypes 205–206
 - symptoms 206
 - treatment 206
- venous collagenosis, involvement 206
- ventral cingulum bundle (VCB), integrity (examination) 114
- ventral pallidum 14
- ventral striatum, nuclei (involvement) 14
- ventral tegmental area (VTA), reward (processing) 363
- ventromedial PFC (VMPFC), bilateral lesions 363
- verbal IQ (VIQ)
 - presentation 191
 - variation 199–200
- verbal memory scores, increase 254
- vesicle soluble *N*-ethylmaleimide-sensitive factor attachment protein receptor (v-SNARE) 6
- Vicodin, usage (national crisis) 268–269
- violence 70
- visual attention processes, stimulus onset reaction 143
- visual word form area (VWFA) 354
- vocabulary 356
- voltage-gated calcium channels, activation 6
- volumetric MRI
 - data, collection 199
 - studies 246

- voxel-based method (VBM) 94
- voxel-based morphometric study, usage 198
- Wallerian degeneration, involvement 206
- warrior gene 70
- water diffusion
 - directional restriction, increase 110
 - kurtosis, measurement 118
- water molecule, translational motion/
 - movement 109
- weight loss, neurocognitive performance
 - improvements (association) 255
- well-being
 - composite perspective 327–329, 361
 - conceptual definitions/
 - measurements 326–329
 - eudaimonic perspective 326–327, 361
 - hedonic perspective 326, 361
 - mental well-being, neural networks
 - 361–367
 - neuroscience
 - conceptual definitions 325
 - neural networks 361
 - PERMA theory 328
 - reward system, association 363–364
 - subjective well-being (SWB) 326
- Wernicke-Korsakoff syndrome (WKS)
 - 396–397
 - thiamine, usage 397
- Wernicke's encephalopathy (WE) 396–397
- white matter 6
 - alterations 231
 - development 2
 - development/decline, trajectory (DTI
 - demonstration) 95
 - diffusion, usage 93
 - fractional anisotropy 125
 - gray matter junctions 238
 - integrity 304
 - impairment 183
 - reduction, DTI studies (factors) 95
 - maturation (changes), DTI parameter
 - sensitivity 94–95
 - structure
 - changes 95–96
 - stress, impact 381–382
 - tract
 - development 281
 - structural connectivity (evaluation), DTI
 - (usage) 381–382
 - tractography 125
 - volume, loss 397
- white matter hyperintensity (WMH)
 - burden, mediation 183
 - MRI evidence 206
- whole diet interventions 256
- WHO Quality of Life Scale (WHOQOL) 326
- Wisconsin Registry for Alzheimer's Prevention
 - (WRAP) 301
- women
 - female interpersonal trauma survivors (PTSD
 - presence), insula
 - functional connectivity 347
 - functional imaging 345–346
 - structural imaging 346
 - posttraumatic stress disorder (PTSD)
 - development, conditional risk
 - (elevation) 342
 - insula level 341
 - stress-related mood disorders, prevalence 31
- working memory (WM) 59
 - microstructural properties 302
 - N-back paradigm 126
 - updating, learning effects 66
- young adulthood, APOE genotype 300

The Wiley Encyclopedia
of Health Psychology

The Wiley Encyclopedia of Health Psychology

Volume 2
The Social Bases of Health Behavior

Edited by

Kate Sweeny
Megan L. Robbins

Editor-in-Chief

Lee M. Cohen

WILEY Blackwell

This edition first published 2021
© 2021 John Wiley & Sons Ltd

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by law. Advice on how to obtain permission to reuse material from this title is available at <http://www.wiley.com/go/permissions>.

The right of Lee M. Cohen to be identified as the author of the editorial material in this work has been asserted in accordance with law.

Registered Offices

John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Office

111 River Street, Hoboken, NJ 07030, USA

For details of our global editorial offices, customer services, and more information about Wiley products visit us at www.wiley.com.

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Limit of Liability/Disclaimer of Warranty

While the publisher and authors have used their best efforts in preparing this work, they make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives, written sales materials or promotional statements for this work. The fact that an organization, website, or product is referred to in this work as a citation and/or potential source of further information does not mean that the publisher and authors endorse the information or services the organization, website, or product may provide or recommendations it may make. This work is sold with the understanding that the publisher is not engaged in rendering professional services. The advice and strategies contained herein may not be suitable for your situation. You should consult with a specialist where appropriate. Further, readers should be aware that websites listed in this work may have changed or disappeared between when this work was written and when it is read. Neither the publisher nor authors shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

Library of Congress Cataloging-in-Publication Data

Names: Paul, Robert D., editor.

Title: The Wiley encyclopedia of health psychology / edited by Robert Paul,
St. Louis MO, USA ; editor-in-chief, Lee Cohen, The University of
Mississippi, MS, USA.

Description: First edition. | Hoboken : Wiley-Blackwell, 2021. | Includes
index. | Contents: volume. 1. Biological bases of health behavior.

Identifiers: LCCN 2020005459 (print) | LCCN 2020005460 (ebook) | ISBN
9781119057833 (v. 1 ; cloth) | ISBN 9781119057833 (v. 2 ; cloth) | ISBN
9781119057833 (v. 3 ; cloth) | ISBN 9781119057833 (v. 4 ; cloth) | ISBN
9781119675785 (adobe pdf) | ISBN 9781119675761 (epub)

Subjects: LCSH: Clinical health psychology—Encyclopedias.

Classification: LCC R726.7 .W554 2021 (print) | LCC R726.7 (ebook) | DDC
616.001/903—dc23

LC record available at <https://lcn.loc.gov/2020005459>

LC ebook record available at <https://lcn.loc.gov/2020005460>

Cover image and design: Wiley

Set in 10/12.5pt ITC Galliard by SPi Global, Pondicherry, India

10 9 8 7 6 5 4 3 2 1

Contents

List of Contributors	xi
Foreword	xxix
Preface	xxxix
Preface Volume 2: Social Bases of Health Behavior	xxxiii
Editor-in-Chief Acknowledgments	xxxv
Acculturation and Substance Use <i>Alan Meca and Seth J. Schwartz</i>	1
Accurate Interpersonal Perception <i>Lauren J. Human</i>	13
Affective Forecasting <i>Elinor Flynn, Arpine Hovasapian, and Linda J. Levine</i>	21
Attachment Processes and Health <i>Onawa P. LaBelle, Britney M. Wardecker, William J. Chopik, and Robin S. Edelstein</i>	31
Bereavement <i>Mary-Frances O'Connor</i>	43
Conflicting Health Information <i>Delesha M. Carpenter and Paul K. J. Han</i>	47
Coping Strategies <i>Ellen Stephenson and Anita DeLongis</i>	55
Coping with Chronic Illness <i>Aliza A. Panjwani and Tracey A. Revenson</i>	61

Counterfactual Thought <i>Rachel Smallman, Amy Summerville, Ryan J. Walker, and Brittney Becker</i>	71
Couple-Relationships and Cancer Adaptation <i>Hoda Badr and Zeba Ahmad</i>	81
Direct-to-Consumer Testing <i>Angela M. Legg and May Reinert</i>	89
Disease-Specific Worry <i>Kevin D. McCaul, Renee E. Magnan, and Michael P. Mead</i>	97
Ecological Momentary Assessment for the Psychosocial Study of Health <i>Doerte U. Jungphaenel and Arthur A. Stone</i>	105
The Effects of Health-Related Policy <i>Robert García and Steve Sussman</i>	113
Embodied Health <i>Ariel J. Mosley, Trevor James Swanson, and Mark J. Landau</i>	121
Emotion Regulation <i>Emily W. Shih and Elizabeth L. Davis</i>	131
Everyday Coping Behaviors <i>Megan L. Robbins, Robert C. Wright, and Alexander Karan</i>	141
Expressive Writing <i>Vanessa Juth, Danielle Arigo, and Joshua M. Smyth</i>	149
Failed Control of Health Behavior <i>Melissa V. Auerbach and Marci Lobel</i>	159
The Geography of Health <i>Arseny A. Ryazanov, Kirstie K. Russell, and Nicholas J. S. Christenfeld</i>	167
Habits <i>Martin S. Hagger and Amanda L. Rebar</i>	177
Health Behavior Change <i>Paul T. Fuglestad, Jody S. Nicholson, and Lauren James</i>	183
Health Behavior Interventions <i>Casey K. Gardiner, Arielle S. Gillman, Courtney J. Stevens, and Angela D. Bryan</i>	193
Health Behavior Maintenance <i>Lisa Auster-Gussman, Rachel J. Burns, and Alexander J. Rothman</i>	203
The Health Belief Model <i>Edward C. Green, Elaine M. Murphy, and Kristina Gryboski</i>	211

Health Consequences and Correlates of Life Stories <i>William L. Dunlop, Tara P. McCoy, and Grace E. Hanley</i>	215
Health Consequences and Correlates of Social Justice <i>Todd Lucas</i>	223
Health Consequences of Prejudice and Discrimination <i>Tessa L. Dover, Jeffrey M. Hunger, and Brenda Major</i>	231
The Health Correlates and Consequences of Friendship <i>Robert G. Kent de Grey and Bert N. Uchino</i>	239
Health Correlates and Consequences of Social Comparison <i>Dana M. Litt, Anne M. Fairlie, Melissa A. Lewis, and Michelle L. Stock</i>	247
Health Effects of Traumatic Events <i>Judith Andersen and Roxane Cohen Silver</i>	255
Health Implications of Gratitude <i>Kristin Layous</i>	261
The Health Implications of Resilience <i>Kristen L. Rudd and Tuppett M. Yates</i>	269
Health Information Avoidance <i>Jennifer L. Howell, Nikolette P. Lipsey, and James A. Shepperd</i>	279
Health Literacy <i>Amber S. Emanuel and Kristel M. Gallagher</i>	287
Healthcare Treatment Seeking and Avoidance <i>Nils Olsen, Phillip J. Moore, Steven Hyde, and Lauren A. Fowler</i>	297
Health-Related Uncertainty <i>Jada G. Hamilton, Margaux C. Genoff, and Paul K. J. Han</i>	305
Commonsense Modeling (CSM) of Health Behaviors <i>Howard Leventhal, L. Alison Phillips, Edith Burns, and Jenna Herold Cohen</i>	315
Implicit Processes and Health Behavior Change <i>Aya Avishai and Paschal Sheeran</i>	329
Intimate Relationships and Physical Health <i>Fiona Ge, Jana Lembke, and Paula R. Pietromonaco</i>	337
Medical Decision Making <i>Meng Li and Gretchen B. Chapman</i>	347
Message Framing <i>Scout N. McCully and John A. Updegraff</i>	355

Message Tailoring <i>Mia Liza A. Lustria and Juliann Cortese</i>	363
Naturalistic Observation of Social Interactions <i>Angela L. Carey, Kelly E. Rentscher, and Matthias R. Mehl</i>	373
Optimism and Physical Health <i>Maria G. Mens, Michael F. Scheier, and Charles S. Carver</i>	385
Patient Adherence <i>Kelly B. Haskard-Zolnieriek and Briana Cobos</i>	395
Patient Satisfaction <i>Summer L. Williams and Tricia A. Miller</i>	403
Personality and Coping <i>Jamie M. Jacobs and Charles S. Carver</i>	411
Personality and Health Behaviors <i>Christopher S. Nave and Michelle R. Dixon</i>	421
Personality and Health Outcomes <i>Dietlinde Heilmayr and Howard S. Friedman</i>	429
Personality Hardiness <i>Salvatore R. Maddi</i>	439
Physical Activity Monitoring <i>Birte von Haaren-Mack, Johannes B.J. Bussmann, and Ulrich W. Ebner-Priemer</i>	447
Physical and Social Pain: Twin Challenges to Well-Being in Adult Life <i>John A. Sturgeon, Alex J. Zautra, and Beth D. Darnall</i>	459
Physician–Patient Communication <i>Tricia A. Miller and M. Robin DiMatteo</i>	469
Placebo and Nocebo Effects <i>Andrew L. Geers and Fawn C. Caplandies</i>	475
Positive Affect <i>Brooke N. Jenkins, Amanda M. Acevedo, and Sarah D. Pressman</i>	485
The Precaution Adoption Process Model <i>Neil D. Weinstein, Peter M. Sandman, and Susan J. Blalock</i>	495
Prejudice and Stereotyping in Healthcare <i>Elizabeth S. Focella</i>	507

The Prototype-Willingness Model <i>Frederick X. Gibbons, Michelle L. Stock, and Meg Gerrard</i>	517
Psychosocial Factors in Coronary Heart Disease <i>Elizabeth J. Vella</i>	529
Relationship Dissolution and Health <i>Karen Hasselmo, Kyle J. Bourassa, and David A. Sbarra</i>	537
Risk Perception <i>Vera Hoorens</i>	547
Rumination <i>Andrew W. Manigault and Peggy M. Zoccola</i>	557
Screening Behavior <i>Leona S. Aiken</i>	569
Selective Exposure in the Domain of Health <i>Zachary Fetterman and William Hart</i>	579
Self-Affirmation and Health <i>Peter R. Harris, Donna C. Jessop, and Philine S. Harris</i>	587
Self-Awareness and Health <i>Melissa Spina and Jamie Arndt</i>	597
Self-Efficacy and Health <i>Lisa Marie Warner and Ralf Schwarzer</i>	605
Self-Esteem and Health <i>Danu Anthony Stinson and Alexandra N. Fisher</i>	615
Self-Regulation <i>Richie L. Lenne and Traci Mann</i>	623
Sex Differences and Health <i>Vicki S. Helgeson and Katilyn Mascatelli</i>	631
Social-Evaluative Threat <i>Sally S. Dickerson</i>	641
Social Factors in Diet and Obesity <i>Gbolahan O. Olanubi and A. Janet Tomiyama</i>	649
Social Factors in Neuroendocrine Function <i>Theodore F. Robles and Ben W. Shulman</i>	659

Social Factors in Sleep <i>Zlatan Krizan and Garrett Hisler</i>	669
Social Identity <i>S. Alexander Haslam, Catherine Haslam, Jolanda Jetten, Tegan Cruwys, and Genevieve A. Dingle</i>	679
Social Influence <i>Karen S. Rook, Dara H. Sorkin, and Kelly Biegler</i>	689
Social Isolation and Health <i>Mona Moieni and Naomi I. Eisenberger</i>	695
Social Support and Health <i>Marci E. J. Gleason and Jerica X. Bornstein</i>	703
Spirituality/Religiosity and Health <i>Kevin S. Masters and Stephanie A. Hooker</i>	709
Stress and Resilience in Pregnancy <i>Alyssa C. D. Cheadle, Isabel F. Ramos, and Christine Dunkel Schetter</i>	717
Subjective Health Norms <i>Allecia E. Reid, Molly B. Hodgkins, Carly A. Taylor, and Alexandra A. Belzer</i>	725
Temptation <i>David A. Kalkstein and Kentaro Fujita</i>	733
Terror Management Health Model <i>Patrick Boyd and Jamie L. Goldenberg</i>	741
The Role of Persuasion in Health-Related Attitude and Behavior Change <i>Colin A. Zestcott and Jeff Stone</i>	747
The Theories of Reasoned Action and Planned Behavior <i>Christina Nisson and Allison Earl</i>	755
Thriving in Life <i>Margaret L. Kern and Jessie Sun</i>	763
Unrealistic Optimism and Health <i>James A. Shepperd and Jennifer L. Howell</i>	773
Waiting for Health News <i>Angelica Falkenstein, Michael D. Dooley, and Kate Sweeny</i>	781
Index	789

List of Contributors

- Amanda M. Acevedo**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Zeba Ahmad**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- Leona S. Aiken**, Department of Psychology, Arizona State University, Tempe, AZ, USA
- Olusola Ajilore**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Damla E. Aksen**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Kristi Alexander**, Department of Psychology, University of Central Florida, Orlando, FL, USA
- Andrea G. Alioto**, Palo Alto University, Palo Alto, CA, USA
Department of Neurology, University of California, Davis, CA, USA
- Michael L. Alosco**, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA
- Christina M. Amaro**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Judith Andersen**, Department of Psychology, University of Toronto, Mississauga, ON, Canada
- Aviva H. Ariel**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Danielle Arigo**, Psychology Department, The University of Scranton, Scranton, PA, USA
- Jamie Arndt**, Psychology Department, University of Missouri System, Columbia, MO, USA
- Robert C. Arnold**, Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
- Melissa V. Auerbach**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Lisa Auster-Gussman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Aya Avishai**, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Hoda Badr**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- David E. Balk**, Department of Health and Nutrition Sciences, Brooklyn College of the City University of New York, Brooklyn, NY, USA

- Kirk Ballew**, Department of Communication, Fontbonne University, St. Louis, MO, USA
 Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
 Department of Communication, University of Illinois at Urbana-Champaign College of
 Liberal Arts and Sciences, Urbana, IL, USA
- Diletta Barbiani**, Department of Neuroscience, University of Turin Medical School, Turin,
 Italy
 Hypoxia Medicine, Plateau Rosà, Switzerland
- Jeffrey E. Barnett**, College of Arts and Sciences, Loyola University Maryland, Baltimore,
 MD, USA
- Petronilla Battista**, Istituti Clinici Scientifici Maugeri IRCCS, Pavia, Institute of Bari, Italy
- Brittney Becker**, Psychology Department, Texas A&M University System, College Station,
 TX, USA
- Alexandra A. Belzer**, Colby College, Waterville, ME, USA
- Fabrizio Benedetti**, Department of Neuroscience, University of Turin Medical School,
 Turin, Italy
 Hypoxia Medicine, Plateau Rosà, Switzerland
- Danielle S. Berke**, Massachusetts Veterans Epidemiology Research and Information Center
 (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Mamta Bhatnagar**, Department of Medicine, University of Pittsburgh Medical Center,
 Pittsburgh, PA, USA
- Kelly Biegler**, Department of Psychological Science, University of California, Irvine, CA,
 USA
- Susan J. Blalock**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill,
 Chapel Hill, NC, USA
- Brittany E. Blanchard**, Department of Psychological Sciences, Texas Tech University,
 Lubbock, TX, USA
- Jennifer B. Blossom**, Clinical Child Psychology Program, University of Kansas, Lawrence,
 KS, USA
- Ryan Bogdan**, BRAIN Lab, Psychological and Brain Sciences, Washington University in
 St. Louis, St. Louis, MO, USA
- Jacob Bolzenius**, Missouri Institute of Mental Health, University of Missouri-St. Louis,
 St. Louis, MO, USA
- Bruce Bongar**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Dianna Boone**, Clinical Psychology Program, Department of Psychological Sciences, Texas
 Tech University, Lubbock, TX, USA
- Jerica X. Bornstein**, Department of Human Development and Family Sciences, University of
 Texas at Austin, Austin, TX, USA
- Joaquín Borrego Jr.**, Department of Psychological Sciences, Texas Tech University, Lubbock,
 TX, USA
- Kyle J. Bourassa**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Lara A. Boyd**, Department of Physical Therapy, University of British Columbia, Vancouver,
 BC, Canada
 Djavad Mowafaghian Centre for Brain Health, University of British Columbia, Vancouver,
 BC, Canada
- Patrick Boyd**, Department of Psychology, University of South Florida, Tampa, FL, USA
- Adam M. Brickman**, Columbia University Medical Center, New York, NY, USA
- Athena Brindle**, Department of Psychology, College of Science, The University of Texas at
 Arlington, Arlington, TX, USA

- Jennifer L. Brown**, Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati College of Medicine, Cincinnati, OH, USA
- Kathleen S. Brown**, Private Practice, Fort Myers, FL, USA
- Wilson J. Brown**, Pennsylvania State University, The Behrend College, Erie, PA, USA
- Steven E. Bruce**, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Angela D. Bryan**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Katherine R. Buchholz**, VA Boston Healthcare System, Boston, MA, USA
National Center for Post Traumatic Stress Disorder, White River Junction, VT, USA
- Claire Burgess**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Edith Burns**, Medical College of Wisconsin, Milwaukee, WI, USA
- Rachel J. Burns**, Douglas Mental Health University Institute, McGill University, Montreal, QC, Canada
- Johannes B.J. Bussmann**, Erasmus MC University Medical Center Rotterdam, CA Rotterdam, South Holland, The Netherlands
- Fawn C. Caplandies**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Angela L. Carey**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Brandon L. Carlisle**, Department of Psychiatry, University of California, San Diego, CA, USA
- Delesha M. Carpenter**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Allison J. Carroll**, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL, USA
- Charles S. Carver**, Department of Psychology, University of Miami, Coral Gables, FL, USA
Center for Advanced Study in the Behavioral Sciences, Stanford University, Stanford, CA, USA
- Isabella Castiglioni**, Dipartimento di Fisica “G. Occhialini”, Università degli Studi di Milano-Bicocca – Piazza della Scienza, Milan, Italy
- Gretchen B. Chapman**, Department of Psychology, Rutgers University–New Brunswick, New Brunswick, NJ, USA
- Alyssa C.D. Cheadle**, Psychology Department, Hope College, Holland, MI, USA
- Rebecca E. Chesher**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- William J. Chopik**, Psychology Department, Michigan State University, East Lansing, MI, USA
- Nicholas J.S. Christenfeld**, Department of Psychology, University of California San Diego, La Jolla, CA, USA
- L.E. Chun**, University of Colorado, Boulder, CO, USA
- Briana Cobos**, Department of Psychology, Texas State University, San Marcos, TX, USA
- Alex S. Cohen**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Jenna Herold Cohen**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Lee M. Cohen**, Department of Psychology, College of Liberal Arts, The University of Mississippi, Oxford, MS, USA
- Catherine Cook-Cottone**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA

- Sarah A. Cooley**, Department of Neurology, School of Medicine, Washington University in Saint Louis, St. Louis, MO, USA
- Stephen Correia**, Department of Psychiatry and Human Behavior, Alpert Medical School, Brown University, Providence, RI, USA
- Patrick Corrigan**, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
- Juliann Cortese**, School of Information, Florida State University, Tallahassee, FL, USA
- Riley Cropper**, Stanford University Counseling and Psychological Services, Stanford Hospital and Clinics, Stanford, CA, USA
- Robert T. Croyle**, National Cancer Institute, Bethesda, MD, USA
- Tegan Cruwys**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Sasha Cukier**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Gregory Dahl**, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Beth D. Darnall**, Department of Anesthesiology, Perioperative, and Pain Medicine, Stanford University School of Medicine, Stanford, CA, USA
- Savannah Davidson**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Elizabeth L. Davis**, Department of Psychology, University of California, Riverside, CA, USA
- Rachael L. Deardorff**, Center for Biomedical Imaging, Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Janet Deatrick**, Department of Family & Community Health, University of Pennsylvania School of Nursing, Philadelphia, PA, USA
- Lauren Decaportale-Ryan**, Departments of Psychiatry, Medicine, & Surgery, University of Rochester Medical Center, Rochester, NY, USA
- Jordan Degelia**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Anita Delongis**, Department of Psychology, University of British Columbia, Vancouver, BC, Canada
- Naiara Demnitz**, Department of Psychiatry, University of Oxford, Oxford, UK
- Eliot Dennard**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Kristine M. Diaz**, Walter Reed National Military Medical Center, Bethesda, MD, USA
- Sally S. Dickerson**, Department of Psychology, Pace University, New York, NY, USA
- M. Robin DiMatteo**, Department of Psychology, University of California Riverside, Riverside, CA, USA
- Sona Dimidjian**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Genevieve A. Dingle**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Laura J. Dixon**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Michelle R. Dixon**, Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Tiffany S. Doherty**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Michael D. Dooley**, Department of Psychology, University of California, Riverside, Riverside, CA, USA

- Tessa L. Dover**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Colleen Doyle**, Institute of Child Development, College of Education and Human Development, University of Minnesota, Minneapolis, MN, USA
- Ira Driscoll**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Claudia Drossel**, Department of Psychology, Eastern Michigan University College of Arts and Sciences, Ypsilanti, MI, USA
- Sean P.A. Drummond**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Christine Dunkel Schetter**, Department of Psychology, University of California, Los Angeles, CA, USA
- William L. Dunlop**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Jacqueline C. Duperrouzel**, Florida International University, Miami, FL, USA
- Allison Earl**, Department of Psychology, University of Michigan, Ann Arbor, MI, USA
- Amanda C. Eaton**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Ulrich W. Ebner-Priemer**, Karlsruhe Institute of Technology, Karlsruhe, Germany
- Robin S. Edelstein**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Jamie Edgin**, Department of Psychology, The University of Arizona, Tucson, AZ, USA
- Bradley A. Edwards**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Naomi I. Eisenberger**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Amber S. Emanuel**, Psychology Department, University of Florida, Gainesville, FL, USA
- Patrick England**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Ipek Ensari**, Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA
- Emily Evans**, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
- James Evans**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Anne M. Fairlie**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Angelica Falkenstein**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Zachary Fetterman**, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
- Christopher Fink**, Durham Veterans Affairs Medical Center, Durham, NC, USA
- North Carolina State University, Raleigh, NC, USA
- Alexandra N. Fisher**, Psychology Department, University of Victoria, Victoria, BC, Canada
- Tabitha Fleming**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Tasheia Floyd**, Washington University School of Medicine, St. Louis, MO, USA
- Lauren A. Fowler**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA

- Elinor Flynn**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Heather Walton Flynn**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Elizabeth S. Focella**, Department of Psychology, University of Wisconsin Oshkosh, Oshkosh, WI, USA
- Amy Frers**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA
- Howard S. Friedman**, Psychology Department, University of California Riverside, Riverside, CA, USA
- Paul T. Fuglestad**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Kentaro Fujita**, Department of Psychology, The Ohio State University, Columbus, OH, USA
- Kristel M. Gallagher**, Thiel College, Greenville, PA, USA
- Andrea M. Garcia**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Paola García-Egan**, Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA
- Robert García**, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA
- Casey K. Gardiner**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Robert J. Gatchel**, Endowment Professor of Clinical Health Psychology Research, Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA
- Justine Megan Gatt**, Neuroscience Research Australia, Sydney, NSW, Australia
School of Psychology, University of New South Wales, Sydney, NSW, Australia
- Nicole K. Gause**, Department of Psychology, University of Cincinnati, Cincinnati, OH, USA
- Ashwin Gautam**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Fiona Ge**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Andrew L. Geers**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Margaux C. Genoff**, Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, NY, USA
- James I. Gerhart**, Department of Psychosocial Oncology, Cancer Integrative Medicine Program, Rush University Medical Center, Chicago, IL, USA
- Meg Gerrard**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Frederick X. Gibbons**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Arielle S. Gillman**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Marci E.J. Gleason**, Department of Human Development and Family Sciences, University of Texas at Austin, Austin, TX, USA
- Teresa Gobble**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA

- Katharina Sophia Goerlich**, Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany
- Jamie L. Goldenberg**, Department of Psychology, University of South Florida, Tampa, FL, USA
- Cesar A. Gonzalez**, Mayo Clinic Minnesota, Rochester, MN, USA
- Raul Gonzalez**, Florida International University, Miami, FL, USA
- Brittany F. Goodman**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Janna R. Gordon**, San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- Edward C. Green**, Department of Anthropology, The George Washington University, Washington, DC, USA
- Joseph P. Green**, Department of Psychology, Ohio State University at Lima, Lima, OH, USA
- Michael G. Griffin**, Department of Psychological Sciences and Center for Trauma Recovery, University of Missouri–St. Louis, St. Louis, MO, USA
- Steven Grindel**, Medical College of Wisconsin, Milwaukee, WI, USA
- Kristina Gryboski**, Project Hope, Millwood, VA, USA
- Suzy Bird Gulliver**, Baylor and Scott and White Hospitals and Clinics, Waco, TX, USA
School of Public Health, Texas A&M University, College Station, TX, USA
- Megan R. Gunnar**, Institute of Child Development, College of Education and Human Development, University of Minnesota, Minneapolis, MN, USA
- Jenna Haddock**, Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA
- Martin S. Hagger**, Health Psychology and Behavioral Medicine Research Group, School of Psychology and Speech Pathology, Faculty of Health Sciences, Curtin University, Perth, WA, Australia
Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland
School of Applied Psychology and Menzies Health Institute Queensland, Behavioural Bases for Health, Griffith University, Brisbane, QLD, Australia
Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia
- Jada G. Hamilton**, Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, NY, USA
- Paul K.J. Han**, Center for Outcomes Research and Evaluation, Maine Medical Center Research Institute, Portland, ME, USA
- Grace E. Hanley**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Madeline B. Harms**, Department of Psychology, University of Wisconsin-Madison, Madison, WI, USA
- Adam Harris**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA
- Lauren N. Harris**, VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA
- Peter R. Harris**, School of Psychology, University of Sussex, Brighton & Hove, UK
- Philine S. Harris**, School of Psychology, University of Sussex, Brighton & Hove, UK
- William Hart**, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
- M.J. Hartsock**, University of Colorado, Boulder, CO, USA

- Kelly B. Haskard-Zolnierrek**, Department of Psychology, Texas State University, San Marcos, TX, USA
- S. Alexandar Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Catherine Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Karen Hasselmo**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Kathryn S. Hayward**, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada
- Stroke Division, Florey Institute of Neuroscience and Mental Health, University of Melbourne, Melbourne, VIC, Australia
- NHMRC Centre for Research Excellence in Stroke Rehabilitation and Recovery, Parkville, VIC, Australia
- Jodi M. Heaps-Woodruff**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Dietlinde Heilmayr**, Psychology Department, Moravian College, Bethlehem, PA, USA
- Vicki S. Helgeson**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Joseph A. Helpern**, Center for Biomedical Imaging, Department of Neuroscience, Department of Neurology, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Garrett Hisler**, Department of Psychology, Iowa State University, Ames, IA, USA
- Molly B. Hodgkins**, Department of Psychological and Brain Sciences, University of Maine, Orono, ME, USA
- Michael Hoerger**, Department of Psychology, Tulane University, New Orleans, LA, USA
- Jeanne S. Hoffman**, US Department of the Army, Washington, DC, USA
- Stephanie A. Hooker**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Vera Hoorens**, Center for Social and Cultural Psychology, Katholieke Universiteit Leuven, Leuven, Belgium
- Arpine Hovasapian**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jennifer L. Howell**, Psychology Department, Ohio University, Athens, OH, USA
- Robert M. Huff**, Department of Health Sciences, California State University, Northridge, Morgan Hill, CA, USA
- Jacob Huffman**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Department of Psychiatry, Washington University in St. Louis School of Medicine, St. Louis, MO, USA
- Lauren J. Human**, Department of Psychology, McGill University, Montreal, QC, Canada
- Jeffrey M. Hunger**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Tanya Hunt**, Department of Clinical Psychology, Palo Alto University, Palo Alto, California, USA
- Steven Hyde**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- Christina Di Iorio**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA

- Jamie M. Jacobs**, Department of Psychiatry, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA
- Lauren James**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Rebekah Jazdzewski**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Brooke N. Jenkins**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jens H. Jensen**, Center for Biomedical Imaging, Department of Neuroscience, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Donna C. Jessop**, School of Psychology, University of Sussex, Brighton & Hove, UK
- Jolanda Jetten**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Kimberly Johnson**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Doerte U. Junghaenel**, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA
- Vanessa Juth**, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA
- Samantha Kahn**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- David A. Kalkstein**, Department of Psychology, New York University, New York, NY, USA
- Alexander Karan**, Department of Psychology, University of California, Riverside, CA, USA
- Natalia M. Kecala**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri–St. Louis, St. Louis, MO, USA
- Robert G. Kent de Grey**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- Margaret L. Kern**, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia
- Nathan A. Kimbrel**, Department of Veterans Affairs' VISN 6 Mid-Atlantic MIRECC, Durham, NC, USA
Durham Veterans Affairs Medical Center, Durham, NC, USA
Psychiatry and Behavioral Sciences, Duke University School of Medicine, Durham, NC, USA
- Tricia Z. King**, The Neuroscience Institute, Georgia State University, Atlanta, GA, USA
- Nancy Kishino**, West Coast Spine Restoration, Riverside, CA, USA
- Ekaterina Klyachko**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Brian Konecky**, Boise VAMC Virtual Integrated Multisite Patient Aligned Care Team Hub, Boise, ID, USA
- Gerald P. Koocher**, Department of Psychology, DePaul University, Chicago, IL, USA
- Laura E. Korthauer**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Abigail O. Kramer**, Palo Alto University, Palo Alto, CA, USA
Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Joel H. Kramer**, Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Zlatan Krizan**, Department of Psychology, Iowa State University, Ames, IA, USA

- Monica F. Kurylo**, Departments of Medicine and Public Health, University of Kansas Medical Center, Kansas City, KS, USA
- Onawa P. LaBelle**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa H. Laitner**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Mark J. Landau**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Shane Landry**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Kevin T. Larkin**, Department of Psychology, West Virginia University, Morgantown, WV, USA
- Jason M. Lavender**, Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA
- Kristin Layous**, Department of Psychology, California State University East Bay, Hayward, CA, USA
- Thanh Le**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Thad R. Leffingwell**, Department of Psychology, Oklahoma State University, Stillwater, OK, USA
- Angela M. Legg**, Department of Psychology, Pace University, New York, NY, USA
- Jana Lembke**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Elizabeth Lemon**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Richie L. Lenne**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Alex Leow**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Department of Bioengineering, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Howard Leventhal**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Linda J. Levine**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Melissa A. Lewis**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Meng Li**, Department of Health and Behavior Sciences, University of Colorado Denver, Denver, CO, USA
- Huijun Liao**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Alexander P. Lin**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Wendy P. Linda**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Nikolette P. Lipsey**, Psychology Department, University of Florida, Gainesville, FL, USA
- Dana M. Litt**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

- Andrew K. Littlefield**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Brett Litz**, Department of Psychology, Boston University & VA Boston Healthcare System, Boston, MA, USA
- Marci Lobel**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Todd Lucas**, C. S. Mott Endowed Professor of Public Health Associate Professor of Epidemiology and Biostatistics Division of Public Health College of Human Medicine Michigan State University, Wayne State University, Detroit, MI, USA
- Mia Liza A. Lustria**, School of Information, Florida State University, Tallahassee, FL, USA
- Steven Jay Lynn**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Salvatore R. Maddi**, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
- Renee E. Magnan**, Washington State University Vancouver, Vancouver, WA, USA
- Brenda Major**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Vanessa L. Malcarne**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
UC San Diego Moores Cancer Center, San Diego, CA, USA
Department of Psychology, San Diego State University, San Diego, CA, USA
- Kelsey A. Maloney**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Andrew W. Manigault**, Psychology Department, Ohio University, Athens, OH, USA
- Traci Mann**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Soe Mar**, Neurology and Pediatrics, Division of Pediatric Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Shannon M. Martin**, Department of Psychology, Central Michigan University, Mount Pleasant, MI, USA
- Allison Marziliano**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Katilyn Mascatelli**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Tyler B. Mason**, Department of Psychology, University of Southern California, Los Angeles, CA, USA
Neuropsychiatric Research Institute, Fargo, ND, USA
- Kevin S. Masters**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Kevin D. McCaul**, North Dakota State University, Fargo, ND, USA
- Tara P. McCoy**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Michael McCrea**, Brain Injury Research Program, Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA
- Scout N. McCully**, Department of Psychological Sciences, Kent State University, Kent OH, USA
- Don McGeary**, Department of Psychiatry, Department of Family and Community Medicine, The University of Texas Health Science Center San Antonio, San Antonio, TX, USA

Jessica McGovern, Department of Psychology, Louisiana State University College of Science,
Baton Rouge, LA, USA

Robert E. McGrath, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA

Michael P. Mead, North Dakota State University, Fargo, ND, USA

Alan Meca, Department of Psychology, Old Dominion University, Norfolk, VA, USA

Matthias R. Mehl, Psychology Department, University of Arizona, Tucson, AZ, USA

Timothy P. Melchert, Department of Psychology, Marquette University, Milwaukee,
WI, USA

Paola Mendoza-Rivera, Department of Clinical Psychology, Palo Alto University, Palo Alto,
CA, USA

Maria G. Mens, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA,
USA

Sai Merugumala, Department of Radiology, Center for Clinical Spectroscopy, Brigham and
Women's Hospital, Harvard Medical School, Boston, MA, USA

Rebecca A. Meza, Department of Communication, Fontbonne University, St. Louis,
MO, USA

Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA

Dara Mickschl, Medical College of Wisconsin, Milwaukee, WI, USA

Tricia A. Miller, Santa Barbara Cottage Hospital, Santa Barbara, CA, USA

Department of Psychology, University of California Riverside, Riverside, CA, USA

Kyle R. Mitchell, Department of Psychology, Louisiana State University College of Science,
Baton Rouge, LA, USA

Mona Moieni, Psychology Department, University of California Los Angeles, Los Angeles,
CA, USA

Phillip J. Moore, Department of Psychological and Brain Sciences, The George Washington
University, Washington, DC, USA

DeAnna L. Mori, VA Boston Healthcare System, West Roxbury, MA, USA

Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA

Sandra B. Morissette, Department of Psychology, The University of Texas at San Antonio
College of Sciences, San Antonio, TX, USA

Amber Morrow, Department of Psychological Sciences, Texas Tech University, Lubbock,
TX, USA

Ariel J. Mosley, Department of Psychology, University of Kansas, Lawrence, KS, USA

Robert W. Motl, Department of Physical Therapy, University of Alabama at Birmingham,
Birmingham, AL, USA

Laurent Mottron, Department of Psychiatry, Faculty of Medicine, Universite de Montreal,
Montreal, QC, Canada

Anne Moyer, Department of Psychology, Stony Brook University, Stony Brook, NY, USA

Elaine M. Murphy, Population Reference Bureau, Washington, DC, USA

Carolyn B. Murray, Psychology, University of California, Riverside, CA, USA

Christopher S. Nave, Department of Psychology, University of Pennsylvania, Philadelphia,
PA, USA

Department of Psychology, Rutgers University Camden, Camden, NJ, USA

Eve-Lynn Nelson, KU Center for Telemedicine & Telehealth, University of Kansas Medical
Center, University of Kansas, Kansas City, KS, USA

Steven M. Nelson, VISN 17 Center of Excellence for Research on Returning War Veterans,
Waco, TX, USA

Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA

Center for Vital Longevity, School of Behavioral and Brain Sciences, University of Texas at Dallas, Dallas, TX, USA

Elizabeth Neumann, Medical College of Wisconsin, Milwaukee, WI, USA

Jason L. Neva, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada

Jody S. Nicholson, Department of Psychology, University of North Florida, Jacksonville, FL, USA

Christina Nisson, Department of Psychology, University of Michigan, Ann Arbor, MI, USA

Mary-Frances O'Connor, Psychology Department, University of Arizona, Tucson, AZ, USA

Laura O'Hara, Department of Communication, Fontbonne University, St. Louis, MO, USA

Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA

Gbolahan O. Olanubi, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA

Nils Olsen, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA

Yimi Omofuma, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA

Elizabeth Ortiz-Gonzalez, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Ileana Pacheco-Colón, Florida International University, Miami, FL, USA

Aliza A. Panjwani, Graduate School and University Center, City University of New York, New York, NY, USA

Mike C. Parent, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA

Thomas J. Parkman, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Robert H. Paul, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

Missouri Institute of Mental Health, St. Louis, MO, USA

Stephanie A. Peak, Battelle Memorial Institute, Columbus, OH, USA

Michael G. Perri, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

Laura Perry, Department of Psychology, Tulane University, New Orleans, LA, USA

Alyssa Phelps, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Carissa L. Philippi, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

L. Alison Phillips, Iowa State University, Ames, IA, USA

Paula R. Pietromonaco, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA

Craig P. Pollizi, Department of Psychology, Binghamton University, Binghamton, NY, USA

Seth D. Pollak, Department of Psychology, University of Wisconsin–Madison, Madison, WI, USA

David B. Portnoy, US Food and Drug Administration, Silver Spring, MD, USA

Rachel Postupack, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

- Matthew R. Powell**, Division of Neurocognitive Disorders, Department of Psychiatry & Psychology, Mayo Clinic Minnesota, Rochester, MN, USA
- Candice Presseau**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Sarah D. Pressman**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Rebecca N. Preston-Campbell**, Missouri Institute of Mental Health, St. Louis, MO, USA
- Isabel F. Ramos**, Department of Psychology, University of California, Los Angeles, CA, USA
- Christopher T. Ray**, College of Health Sciences, Texas Woman's University, Denton, TX, USA
- Amanda L. Rebar**, Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia
- Allecia E. Reid**, Colby College, Waterville, ME, USA
- May Reinert**, Department of Psychology, Pace University, New York, NY, USA
- Kelly E. Rentscher**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Tracey A. Revenson**, Department of Psychology, Hunter College, New York, NY, USA
- C. Steven Richards**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Laura River**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Megan L. Robbins**, Department of Psychology, University of California, Riverside, CA, USA
- Michael C. Roberts**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- John D. Robinson**, Department of Surgery and Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine/Hospital, Washington, DC, USA
- Theodore F. Robles**, Psychology Department, University of California Los Angeles, CA, USA
- Catherine Rochefort**, Department of Psychology, Southern Methodist University, Dallas, TX, USA
- Daniel G. Rogers**, Department of Psychology, University of Mississippi, MS, USA
- John L. Romano**, Department of Educational Psychology, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Karen S. Rook**, Department of Psychological Science, University of California, Irvine, CA, USA
- Ann Rosenthal**, Medical College of Wisconsin, Milwaukee, WI, USA
- J. Megan Ross**, Florida International University, Miami, FL, USA
- Tania L. Roth**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Alexander J. Rothman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Ronald H. Rozensky**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Kristen L. Rudd**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Kirstie K. Russell**, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada
- Arseny A. Ryazanov**, Department of Psychology, University of California San Diego, La Jolla, CA, USA

- Napapon Sailasuta**, Centre for Addiction and Mental Health, Toronto, ON, Canada
Stella Sakhon, Department of Psychology, The University of Arizona, Tucson, AZ, USA
Lauren E. Salminen, Imaging Genetics Center, University of Southern California, Marina del Rey, CA, USA
Christian Salvatore, Scuola Universitaria Superiore IUSS Pavia, Piazza della Vittoria, Pavia, Italy
DeepTrace Technologies S.R.L., Via Conservatorio, Milan, Italy
Peter M. Sandman, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
Keith Sanford, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
David A. Sbarra, Department of Psychology, University of Arizona, Tucson, AZ, USA
Michael F. Scheier, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
Adam T. Schmidt, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Elana Schwartz, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
Esther N. Schwartz, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
Seth J. Schwartz, Department of Public Health Sciences, University of Miami Miller School of Medicine, Miami, FL, USA
Ralf Schwarzer, Health Psychology, Freie Universität Berlin, Berlin, Germany
Health Psychology, Australian Catholic University, Melbourne, VIC, Australia
Richard J. Seime, Mayo Clinic Minnesota, Rochester, MN, USA
Claire E. Sexton, Department of Psychiatry, University of Oxford, Oxford, UK
Louise Sharpe, School of Psychology, Faculty of Science, The University of Sydney, Sydney, NSW, Australia
Lindsay Sheehan, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
Paschal Sheeran, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
Daniel Sheinbein, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
James A. Shepperd, Psychology Department, University of Florida, Gainesville, FL, USA
Brittany N. Sherrill, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
Molin Shi, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Emily W. Shih, Department of Psychology, University of California, Riverside, CA, USA
Ben W. Shulman, Psychology Department, University of California, Los Angeles, CA, USA
Elizabeth Silberman, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
Department of Neurology, Oregon Health and Science University, Portland, OR, USA
Roxane Cohen Silver, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
Colleen A. Sloan, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA

Rachel Smallman, Psychology Department, Texas A&M University System, College Station, TX, USA

Kathryn E. Smith, Neuropsychiatric Research Institute, Fargo, ND, USA
Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA

Patrick J. Smith, Duke University Medical Center, Durham, NC, USA

Todd A. Smitherman, Department of Psychology, University of Mississippi, University, MS, USA

Joshua M. Smyth, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA

Dara H. Sorkin, Department of Psychological Science, University of California, Irvine, CA, USA

R. L. Spencer, University of Colorado, Boulder, CO, USA

Melissa Spina, Psychology Department, University of Missouri System, Columbia, MO, USA

Bonnie Spring, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA

Annette L. Stanton, Department of Psychology, University of California, Los Angeles, Los Angeles, CA, USA

Ellen Stephenson, Department of Psychology, University of British Columbia, Vancouver, BC, Canada

Robert A. Stern, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Department of Neurosurgery and Anatomy & Neurobiology, Boston University School of Medicine, Boston, MA, USA

Angela K. Stevens, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Courtney J. Stevens, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA

Elizabeth M. Stewart, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia

Danu Anthony Stinson, Psychology Department, University of Victoria, Victoria, BC, Canada

Michelle L. Stock, Department of Psychological and Brain Sciences, George Washington University, Washington, DC, USA

Arthur A. Stone, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA

Department of Psychology, Dana and David Dornsife College of Letters Arts and Sciences, University of Southern California, Los Angeles, CA, USA

Jeff Stone, Psychology Department, University of Arizona, Tucson, AZ, USA

John A. Sturgeon, Center for Pain Relief, University of Washington, Seattle, WA, USA

Kieran T. Sullivan, Department of Psychology, Santa Clara University, Santa Clara, CA, USA

Amy Summerville, Psychology Department, Miami University, Oxford, OH, USA

Jessie Sun, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia

Department of Psychology, University of California, Davis, CA, USA

Steve Sussman, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

- Trevor James Swanson**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Allison M. Sweeney**, Department of Psychology, University of South Carolina, Columbia, SC, USA
- Kate Sweeny**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Sergey Tarima**, Medical College of Wisconsin, Milwaukee, WI, USA
- Carly A. Taylor**, Boston Medical Center, Boston, MA, USA
- George T. Taylor**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Interfakultäre Biomedizinische Forschungseinrichtung (IBF) der Universität Heidelberg, Heidelberg, Germany
- A. Janet Tomiyama**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Victoria A. Torres**, Department of Psychology, College of Liberal Arts, University of Mississippi, Oxford, MS, USA
- David R.M. Trotter**, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
- Bert N. Uchino**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- John A. Updegraff**, Department of Psychological Sciences, Kent State University, Kent, OH, USA
- Jay Urbain**, Milwaukee School of Engineering, Milwaukee, WI, USA
- Amy E. Ustjanauskas**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- UC San Diego Moores Cancer Center, San Diego, CA, USA
- Jason Van Allen**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Erika D. Van Dyke**, Department of Sport Sciences, College of Physical Activity and Sport Sciences, West Virginia University, Morgantown, WV, USA
- Department of Psychology, Springfield College, Springfield, MA, USA
- Judy L. Van Raalte**, Department of Psychology, Springfield College, Springfield, MA, USA
- Maryke Van Zyl-Harrison**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Rachel Vanderkruik**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Elizabeth J. Vella**, Department of Psychology, University of Southern Maine, Portland, ME, USA
- Birte von Haaren-Mack**, German Sport University Cologne, Köln, Nordrhein-Westfalen, Germany
- David Von Nordheim**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Mikhail Votinov**, Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany
- Institute of Neuroscience and Medicine (INM-10), Jülich, Germany
- Rebecca K. Vujnovic**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA
- Amy Wachholtz**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA

- Eric F. Wagner**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Ryan J. Walker**, Psychology Department, Miami University, Oxford, Ohio, USA
- Rachel A. Wamser-Nanney**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Britney M. Wardecker**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa Ward-Peterson**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Lisa Marie Warner**, Health Psychology, Freie Universitat Berlin, Berlin, Germany
- Neil D. Weinstein**, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
- Suzanne E. Welcome**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Kristen J. Wells**, San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- Amy Wenzel**, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, PA, USA
- Private Practice, Rosemont, PA, USA
- Summer L. Williams**, Department of Psychology, Westfield State University, Westfield, MA, USA
- Cynthia J. Willmon**, Reno VA Sierra Nevada Health Care System, Reno, NV, USA
- Sara M. Witcraft**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Stephen A. Wonderlich**, Neuropsychiatric Research Institute, Fargo, ND, USA
- Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA
- E.R. Woodruff**, University of Colorado, Boulder, CO, USA
- Patrick W. Wright**, Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Robert C. Wright**, Department of Psychology, University of California, Riverside, CA, USA
- Shawna Wright**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Tuppett M. Yates**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Julie D. Yeterian**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Lok-Kin Yeung**, Columbia University Medical Center, New York, NY, USA
- Alex J. Zautra**, Psychology Department, Arizona State University, Tempe, AZ, USA
- Colin A. Zestcott**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Peggy M. Zoccola**, Psychology Department, Ohio University, Athens, OH, USA

Foreword

Until the 1970s, there were no books, journals, or university courses on health psychology. Although the field's intellectual roots stretch back to the beginnings of psychology more than a century ago, its formal emergence depended on a convergence of influences (Friedman & Silver, 2007), including psychosomatic medicine, social-psychological and socio-anthropological perspectives on medicine, epidemiology, and medical and clinical psychology. Today, health psychology is a principal area of significant social science research and practice, with vital implications for the health and well-being of individuals and societies. Understanding the explosive trajectory of health psychology is useful to appreciating the strengths of the field and to approaching this new encyclopedia, the *Wiley Encyclopedia of Health Psychology*.

What is the nature of health? That is, what does it mean to be healthy? The way that this question is answered affects the behaviors, treatments, and resource allocations of individuals, families, health practitioners, governments, and societies. For example, if it is thought that you are healthy unless and until you contract a disease or suffer an injury, then attention and resources are primarily allocated toward “fixing” the problem through medications or surgical repairs. This is the traditional biomedical model of disease (sometimes called the “disease model”). Indeed, in the United States, the overwhelming allocation of attention and resources is to physicians (doing treatments) and to pharmaceuticals (prescription drugs and their development). In contrast, health psychology developed around a much broader and more interdisciplinary approach to health, one that is often termed the biopsychosocial model.

The biopsychosocial model (a term first formally proposed by George Engel in 1968) brings together core elements of staying healthy and recovering well from injury or disease (Stone et al., 1987). Each individual—due to a combination of biological influences and psychosocial experiences—is more or less likely to thrive. Some of this variation is due to genetics and early life development; some depends on the availability and appropriate applications of medical treatments; some involves nutrition and physical activity; some depends on preparations for, perceptions of, and reactions to life's challenges; and some involves exposure to or seeking out of healthier or unhealthier environments, both physically and socially. When presented in this way, it might seem obvious that health should certainly be viewed in this broader interdisciplinary way. However, by misdirecting its vast expenditures on health care, the United States gives its residents mediocre health at high cost (Kaplan, 2019). To approach these matters in a thorough manner, this encyclopedia includes four volumes, with Volume 1 focusing on the biological bases of health and health behavior, Volume 2 concentrating on the social bases, Volume 3 centering around the psychological and clinical aspects, and Volume 4 focused more broadly on crosscutting and applied matters.

Key parts of health depend on biological characteristics and how they interact with our experiences and environments. So, for example, in Volume 1, there are articles on injury to the brain, alcohol effects on the brain, nutrition, drug abuse, psychophysiology, and the tools and key findings of neuroscience. Note that even individuals with the same genes (identical twins) can and do have different health and recovery outcomes, and the articles delve into such complexities of health. Of course humans are also social creatures, and people's growth, development, and health behaviors take place in social contexts. So, in Volume 2, there are articles on such fundamental matters as social support, coping, spirituality, emotion, discrimination, communication, psychosocial stress, and bereavement.

Because our approaches to and conceptions of health are heavily influenced by society's institutions and structures revolving around medical care, much of health psychology derives from or intersects with clinical psychology and applied behavioral medicine. Volume 3 covers such clinical topics as psycho-oncology, depression, drug abuse, chronic disease, eating disorders, and the psychosocial aspects of coronary heart disease. Finally and importantly, there are a number of special and cross-cutting matters that are considered in Volume 4, ranging from relevant laws and regulations to telehealth and health disparities. Taken together, the articles triangulate on what it truly means to be healthy.

What are the most promising directions for the future that emerge from a broad and deep approach to health? That is, to where do these encyclopedia articles point? One key clue arises from a unique opportunity to enhance, extend, and analyze the classic Terman study of children who were followed and studied throughout their lives (Friedman & Martin, 2012). These studies revealed that there are lifelong trajectories to health, thriving, and longevity. Although anyone can encounter bad luck, a number of basic patterns emerged that are more likely to lead to good health. That is, for some individuals, certain earlier life characteristics and circumstances help propel them on *pathways* of healthier and healthier behaviors, reactions, relationships, and experiences, while others instead face a series of contingent stumbling blocks. There are multi-part but nonrandom pathways across time linking personalities, health behaviors, social groups, education, work environments, and health and longevity. The present encyclopedia necessarily is a compendium of summaries of the relevant elements of health and thriving, but one that would and can profitably be used as a base to synthesize the long-term interdependent aspects of health.

In sum, this encyclopedia is distinctive in its explicit embrace of the biopsychosocial approach to health, not through lip service or hand-waving but rather through highly detailed and extensive consideration of the many dozens of topics crucial to this core interdisciplinary understanding.

References

- Friedman, H. S., & Martin, L. R. (2012). *The longevity project: Surprising discoveries for health and long life from the landmark eight-decade study*. New York: Penguin Plume Press.
- Friedman, H. S., & Silver, R. C. (Eds.) (2007). *Foundations of health psychology*. New York: Oxford University Press.
- Kaplan, R. M. (2019). *More than medicine: The broken promise of American health*. Cambridge, MA: Harvard University Press.
- Stone, G., Weiss, M., Matarazzo, J. D., Miller, N. E., Rodin, J., Belar, C. D., ... Singer, J. E. (Eds.) (1987). *Health psychology: A discipline and a profession*. University of Chicago Press.

Howard S. Friedman
University of California, Riverside
August 15, 2019

Preface

The field of health psychology is a specialty area that draws on how biology, psychology, behavior, and social factors influence health and illness. Despite the fact that the formal recognition of the field is only about a half of a century old, it has established itself as a major scientific and clinical discipline. The primary reason for this is that there have been a number of significant advances in psychological, medical, and physiological research that have changed the way we think about health, wellness, and illness. However, this is only the tip of the iceberg.

We have the field of health psychology to thank for much of the progress seen across our current healthcare system. Let me provide some examples that illustrate the diversity of these important contributions. Starting with a broad public health perspective, health psychologists have been involved in how our communities are planned and how urban development has a significant and direct impact on our health behaviors. That is, if we live close to where we work, play, and shop, we are more likely to walk or bike to our destination rather than drive or take public transportation. Focusing on a smaller, but no less important, source of data, health psychologists are involved in using information obtained from our genes to help counsel individuals to make good, well-informed health decisions that could have an impact on the individual immediately or in the future. Being well-informed can direct a person toward the best possible path toward wellness, whether it be measures aimed at prevention, further monitoring, or intervention.

Of course, there are many other contributions. For instance, data indicates that several of the leading causes of death in our society can be prevented or delayed (e.g., heart disease, respiratory disease, cerebrovascular disease, and diabetes) via active participation in psychological interventions. Knowing that we can improve health status by changing our behaviors seems like an easy “fix,” but we know that behavior change is tough. As such, it is not surprising that health psychologists have been involved in trying to improve upon treatment success by examining patient compliance. When we better understand what motivates and discourages people from engaging in treatment or pro-health behaviors, we can improve upon compliance and help individuals adopt more healthy lifestyles. Health psychologists have also played a role in shaping healthcare policy via identifying evidence-based treatments. This work has direct effects on how individuals receive healthcare as well as what treatments are available/reimbursable by insurance companies. Finally, there are also factors beyond medical care to consider (e.g., economic, educational) that can lead to differential health outcomes. Thus, health psychologists likewise examine ways to reduce health disparities, ensuring that the public and government officials are made aware of the impact of the social determinants of health.

I could go on and on, however, examples like this illustrate the significant impact this broad and exciting field has had (and will continue to have) on our understanding of health and wellness. Given the constantly changing nature of the field, it is not possible to be all inclusive; however, the aim of these four volumes is to provide readers an up-to-date overview of the field. Each entry is written to stand alone for those who wish to learn about a specific topic, and if the reader is left wanting more, suggested readings are provided to expand one's knowledge. Volume I, *Biological Bases of Health Behavior*, includes entries that cover topics in the broad areas of neuroscience and biopsychology relevant to health behavior. General topics include degenerative and developmental conditions, emerging methodologies available in clinical research, functional anatomy and imaging, and gene×environment interactions. Volume II, *Social Bases of Health Behavior*, addresses topics related to theories and concepts derived from social psychology. Specifically, topics related to the self, social cognition, social perception, attitudes and attitude change, perception, framing, and pro-health behaviors are included. Volume III, *Clinical Health Psychology and Behavioral Medicine*, covers the applied aspects of the field of health psychology including practical topics that clinical health psychologists face in the workplace, behavioral aspects of medical conditions, the impact of unhealthy behaviors, and issues related to the comorbidity of psychiatric disorders and chronic health concerns. Finally, Volume IV, *Special Issues in Health Psychology*, contains a wide array of topics that are worthy of special consideration in the field. Philosophical and conceptual issues are discussed, along with new approaches in delivering treatment and matters to consider when working with diverse and protected populations.

It is my sincere hope that the *Wiley Encyclopedia of Health Psychology* will serve as a comprehensive resource for academic and applied psychologists, other health care professionals interested in the relationship of psychological and physical well-being, and students across the health professions. I would very much like to thank and acknowledge all those who have made this work possible including Michelle McFadden, the Wiley editorial and production teams, the volume editors, and of course all of the authors who contributed their outstanding work.

Lee M. Cohen, PhD

Preface Volume 2: Social Bases of Health Behavior

Social context plays numerous critical roles in health processes. Consider the utility of a medical diagnosis if patients do not trust their doctor or take their prescribed medication. Patients may also find that racial and gender biases can lead to different diagnoses and treatments because healthcare providers are influenced by social factors just like everyone else. Outside of medical contexts, researchers have discovered that personality predicts health and longevity and leads to different styles of coping with stressful situations. Turning to health behavior, imagine knowing what you need to do to manage a chronic illness but encountering significant barriers to changing your behavior. Finally, the presence and quality of close relationships can alter physical, as well as mental, health in myriad ways. Volume 2 of this encyclopedia aims to address the broad topic of how social influences get under the skin to affect health.

This volume is the result of an effort to provide a concise, yet comprehensive, overview of the social bases of health. We define “social” quite broadly, including factors that are clearly social in nature (e.g., communication, prejudice, romantic relationships, social support) as well as factors that are fundamentally intrapersonal and yet deeply entangled with one’s social context (e.g., emotion regulation, habits, self-esteem, attitudes). We conceptualized the field as loosely falling into the following topical areas (in no particular order): (a) health behavior, (b) social cognitive processes, (c) emotions and emotion regulation, (d) stress and coping, (e) self and identity, (f) healthcare and health communication, (g) health disparities, (h) relationship processes, and (i) social factors in illness. We also include entries that address broad theories of health and health behavior, and further entries address contemporary methodological issues that commonly arise in the study of health from a social perspective.

We were fortunate to amass a set of contributing authors that represents the best and brightest in the field, both rising stars and prominent luminaries. Although space does not allow us to summarize all of the outstanding entries, we highlight a few here in an effort to capture the breadth and quality of the volume that lies ahead. Regarding theoretical contributions, Howard Leventhal and his co-authors provide an overview of nearly a half-century of groundbreaking work on illness representations, and Rick Gibbons and Meg Gerard (with Michelle Stock) outline the prototype-willingness model, their influential model of adolescent health behavior. An entry by Leona Aiken reviews decades of important research on screening behavior, Anita DeLongis (led by Ellen Stephenson) summarizes the large and complex research literature on coping strategies, Roxane Cohen Silver (led by Judith Pizarro Andersen) considers the role of trauma in health outcomes, and Robin DiMatteo (led by Tricia Miller) highlights the importance of effective physician–patient communication. Other key contributors

include Alex Rothman (led by Lisa Auster-Gussman and Rachel Burns), who tackles the challenging issue of health behavior maintenance; Christine Dunkel Schetter (led by Alyssa Cheadle and Isabel Ramos), who addresses social bases of the pregnancy experience; Brenda Major (led by Tessa Dover and Jeffrey Hunger), who presents evidence for the negative health consequences of prejudice and discrimination; and Arthur Stone, who discusses the methodological contribution of ecological momentary assessment in health research.

Finally, we dedicate our volume to two contributors who passed away during the preparation of this encyclopedia, both enormous losses to the field of health psychology. Charles Carver co-authored two entries in our volume, one on dispositional optimism (with Maria Mens and long-time collaborator Michael Scheier) and one on the role of personality in coping (with Jamie Jacobs). Carver's work on dispositional optimism was inspiring, interdisciplinary, and highly influential, as evidenced by the nearly 125,000 citations to his work.

Alex Zautra co-authored an entry on physical and social pain (with John Sturgeon and Beth Darnall). Zautra's research illuminated the interplay between physical health and social aspects of life, and his work on resilience was transformative to the field of health psychology. We are indebted to Carver and Zautra for their influences on both our volume and the field, and we are deeply grateful to all of our authors for their role in what we believe to be an important contribution to the social side of health psychology.

**Kate Sweeny
Megan L. Robbins**

Editor-in-Chief Acknowledgments

I would like to publicly recognize and thank my family, Michelle, Ross, Rachel, and Becca for being supportive of my work life while also providing me with a family life beyond my wildest dreams. I love you all very much. I would also like to note my great appreciation and thanks to the faculty and staff of the Doctoral Training Program in Clinical Psychology at Oklahoma State University for providing me with an opportunity to expand my education and for providing extraordinary training. In particular, I would like to thank my late mentor Dr. Frank L. Collins Jr., Dr. Larry Mullins, Dr. John Chaney, and Patricia Diaz Alexander. Further, I am grateful to the University of Mississippi and Texas Tech University (and the colleagues and students I have had the privilege to work alongside) for affording me excellent working environments and the support to do the work I am honored to be a part of. Finally, I would like to recognize and thank the volume editors for their vision and perseverance to this project as well as to each of the contributors for their excellent entries and their dedication to this very important field.

Lee M. Cohen, Ph.D.
Editor-in-Chief

Acculturation and Substance Use

Alan Meca¹ and Seth J. Schwartz²

¹*Department of Psychology, Old Dominion University, Norfolk, VA, USA*

²*Department of Public Health Sciences, University of Miami Miller School of Medicine, Miami, FL, USA*

Since the 1970s, the foreign-born population in the United States has rapidly increased, reaching an all-time high of nearly 40 million (13% of the total population) in 2010 (Grieco et al., 2012). Today, the majority of foreign-born individuals residing in the United States originate from what is commonly known as the global south (i.e., Latin America and Asia). Immigrants from the global south typically hold collectivist-based beliefs (i.e., focus on the well-being of the family, clan, nation, or religion), whereas members of the receiving society typically emphasize individualism (i.e., focus on autonomy and self-sufficiency; Hofstede, 2001). The patterns of migration to the United States over the past 50 years have therefore produced cultural divides between immigrants and the society that receives them. As a result of the large and increasing flow of international migrants, coupled with these cultural divides, scholarly interest in acculturation has substantially increased over the last few decades (Schwartz, Unger, Zamboanga, & Szapocznik, 2010).

Acculturation deals with the process of blending into a new culture and refers primarily to the extent to which an individual retains his or her cultural heritage and/or adopts the new receiving culture (Berry, 1980). Acculturation and the stressors that often accompany it affect not only foreign-born individuals but also US-born individuals from immigrant families. Accordingly, there has been a growing interest in work that examines the relationship between acculturation and a wide range of positive and negative indicators of psychosocial functioning (Schwartz et al., 2010). This entry serves as a review of contemporary models of acculturation and the role of acculturation in substance use among adolescents from immigrant families. Our focus on adolescence is driven by our motivation to understand the role of acculturation in the early stages of vulnerability. In addition, this entry focuses particularly on identifying methodological and conceptual limitations that have been prominent in the field of acculturation and on pointing out how the field might best move forward.

Contemporary Models of Acculturation: Bidimensional and Multicomponent Process

Current conceptualizations of acculturation have largely grown out of what is termed the “unidimensional” view, a framework that places the acculturative experience along a single continuum ranging from “completely unacculturated” to “completely acculturated.” This continuum is used to describe an immigrant’s ability to blend with her/his new cultural surroundings following arrival. Although an individual could feasibly be placed anywhere on the continuum (from low to high), that person’s placement essentially assumes that “successful” acculturation involves complete adoption of the receiving culture and forfeiture of their cultural heritage (Gordon, 1964). In contrast to this unidimensional view, contemporary views have increasingly cast acculturation as a bidimensional process (Schwartz et al., 2010). According to Berry’s (1980) seminal acculturation model, receiving-culture acquisition and heritage-culture retention represent independent dimensions. In this respect, acquiring the culture of the receiving context does not imply that an individual will discard their own cultural heritage; rather, she/he may judiciously straddle a fence between two cultural worlds, where different cultural streams will be expressed depending on the situation at hand.

Several authors have extended Berry’s (1980) original conceptualization, suggesting that acculturation is multidimensional, not only because it contains numerous cultural streams but also because it stretches across at least three relevant components: *cultural practices*, *cultural values*, and *cultural identifications* (Schwartz et al., 2010). *Cultural practices* refer to behaviors such as language use, choice of friends, and cultural customs and traditions. The vast majority of studies and measures in the acculturation literature have focused on cultural practices, emphasizing language use in particular. Extending beyond cultural practices, contemporary conceptualizations of acculturation also emphasize the importance of *cultural values* or belief systems associated with a specific context or group. Cultural values can refer to broad, cross-ethnic group values (e.g., collectivism and individualism) as well as group-specific values such as familism (strength of family ties) and filial piety (bestowing honor upon one’s family). The *cultural identifications* component refers to the strength of commitment and attachment a person feels toward her/his cultural heritage and/or the culture of the receiving context.

Although early conceptualizations of acculturation examined at most one or two of these components, more recent arguments suggest that acculturation is best understood as the confluence of several components. Indeed, Schwartz et al. (2010) have suggested a model that crosses the three components of acculturation across two dominant cultural streams and yields a total of six facets of acculturation: US practices, heritage practices, individualist values, collectivist values, US identification, and heritage identification. This blending of all three components with both cultural streams provides a more complete picture that is faithful to the theoretical definitions and original conceptualizations of acculturation (Berry, 1980).

Acculturation and Substance Use

Empirical studies evaluating the relationship between acculturation and substance use among adolescent immigrants are quite limited. Moreover, despite evidence favoring bidimensional over unidimensional approaches to acculturation, the majority of studies linking acculturation to substance use have relied on unidimensional conceptualizations, demographic proxies, and/or measures that tap solely into one of three components of acculturation (Schwartz

et al., 2010). Although the findings from these studies have been inconsistent, many have reported that “greater” degrees of acculturation are associated with problematic health outcomes. However, this conceptualization of acculturation leaves unclear whether the effects of acculturation on health outcomes are due to adoption of the receiving culture or to loss/rejection of the heritage culture. As of the summer of 2015, our review indicated only a handful of studies employing a bidimensional conceptualization of acculturation among first- and second-generation adolescent immigrants. Moreover, all of these studies were conducted with Hispanic adolescents. Indeed, our review did not find a single study employing a bidimensional conceptualization of acculturation among immigrant youth in countries other than the United States.

Acculturation and Alcohol Use

Using a validated measure of cultural practices, Unger, Ritt-Olson, Soto, and Baezconde-Garbanati (2009) found that US orientation among Hispanic adolescents was protective against recent alcohol use at a single point in time. However, using growth curve analysis, Unger, Schwartz, Huh, Soto, and Baezconde-Garbanati (2014) found no relationship between US or Hispanic orientations and alcohol use. Similarly, Zamboanga, Schwartz, Jarvis, and Van Tyne (2009) found no significant relationship between US or Hispanic cultural practices and alcohol use initiation or frequency.

Acculturation and Cigarette Use

Using comprehensive measures of Hispanic and US cultural practices, Zamboanga et al. (2009) and Unger et al. (2009, 2014) found significant negative associations between adolescents’ Hispanic orientation and cigarette use. However, neither Zamboanga et al. (2009) nor Unger et al. (2009, 2014) found a significant relationship between US orientation and cigarette use.

Acculturation and Illicit Drug Use

For the most part, studies employing a bidimensional approach have found a Hispanic orientation was associated with a lower risk of marijuana use (Unger et al., 2009, 2014) and illicit drug use (a latent variable measured by marijuana, hard drug, inhalant, injecting, and prescription drugs; Schwartz et al., 2011).

Summary

As a whole, studies using bidimensional and multicomponent models of acculturation suggest that the loss of one’s heritage practices, values, and identifications poses a greater risk for substance use among Hispanic youth. Adoption of US cultural practices, values, and identifications does not appear to pose a risk for these outcomes. The unidimensional conclusion that “greater acculturation is a risk for substance use” appears to be overly simplistic and misguided. Although the field of acculturation has begun moving toward a bidimensional and multicomponent conceptualization of acculturation, research on the role of acculturation in substance use must still address substantial theoretical and methodological limitations. Next, we outline some of these limitations and discuss future directions for the field.

Understanding the Mechanisms Between Acculturation and Substance Use

To develop culturally based prevention programs, it is essential to understand the developmental mechanisms underlying the relationship between acculturation and substance use. Although several theories have been proposed, we focus here on two of the more promising theoretical models: cultural stress and family functioning/acculturation-gap theories. Below we review these theoretical models and identify future research within each of these areas.

Disentangling Cultural Stress from Acculturation and Other Stressors

A long tradition of researchers has suggested that it is the stress associated with acculturation, rather than acculturation per se, that contributes to wide range of dysfunctional behaviors. Indeed, several theoretical frameworks have attempted to account for the process through which acculturation promulgates psychosocial stress, proposing that conflict between one's heritage and receiving cultures produces perceived/experienced cultural stress. Cultural stress comprises a variety of experiences, including perceived discrimination, negative context of reception, and bicultural stress. As a whole, the literature has consistently found that indicators of cultural stress are linked to increased risk of substance use (e.g., Tran, Lee, & Burgess, 2010).

Studies attempting to flesh out these relationships have found that acculturation is linked with cultural stressors, which in turn are associated with impaired psychosocial functioning and increase the probability of engaging in health risk behaviors (e.g., Juang & Cookston, 2009). Although these studies provide preliminary support for the role of cultural stress as a key mediator of the relationship between acculturation and substance use, it is possible that cultural stress may also function as a moderator. Specifically, the acculturation process for youth experiencing greater cultural stress may lead to feelings of being overwhelmed. Alternatively, some have posited that acculturation may instead moderate the relationship between cultural stress and psychosocial functioning. People who try to assimilate into the dominant culture but are rejected may experience the most distress (Staerklé, Sidanius, Green, & Molina, 2010). As a consequence, immigrant youth who adopt US cultural orientations may experience the negative impacts of discrimination and living in an unwelcoming context of reception. On the other hand, youth who retain and value their cultural heritage may be more likely to experience the negative impacts of cultural conflict within their own ethnic group. For example, Deng, Kim, Vaughan, and Li (2010) found that the relationship between perceived discrimination and delinquent behavior was higher for adolescents who strongly endorsed their heritage culture. The reverse pattern was found for US cultural orientation.

Moving forward, to develop a more comprehensive model, an imperative first step is to separate the "process of acculturation" from the underlying "process of cultural stress" and determine whether both contribute independently or interdependently to maladaptive outcomes. It is also essential to identify the exact nature of their relationship with one another (i.e., mediation, moderation, or both). In addition, it is important to note that studies to date have yet to determine whether culturally specific stress is unique from other forms stress (e.g., normative developmental and family economic stress). Research has also yet to disentangle the effects of these various stressors on various forms of psychosocial functioning. Thus, an additional step for future research is to evaluate the relative contributions that cultural stress and other stressors, in conjunction with the process of acculturation, exert on youth adaptation.

Family as a Developmental System

A critical step for future research requires conceptualizing the family as a holistic and constantly evolving system. Of relevance to the current entry, research in the past decade has focused on clarifying the ways in which caregivers encourage children to gravitate toward specific aspects of their cultural heritage and to avoid aspects of the receiving cultural context (Portes & Rumbaut, 2006). Umaña-Taylor, Bhanot, and Shin (2006) have referred to this phenomenon as *familial ethnic socialization*. Several studies have found that the presence of heritage cultural symbols, such as activities in the home, increases the likelihood that children and adolescents will retain or adopt their heritage culture (Umaña-Taylor et al., 2006). Regarding acquisition/rejection of US culture, however, there is evidence that caregivers' socialization attempts are less effective in shaping youths' US cultural adoption. Peers, school, and media may play a greater role than youths' primary caregivers in shaping US cultural adoption. Although scholars have emphasized the role adolescents have in transmitting US values and culture (Padilla, 2006), studies have yet to empirically examine whether adolescents have an impact on their caregivers' acquisition/retention of US and heritage cultures.

Over and above focusing on the direct role caregivers may have in shaping their child's acculturation process, researchers have highlighted that caregiver-child gaps in acculturative processes can affect family processes, cultural stress, and mental health outcomes. For example, differential acculturation theory (DAT) (Szapocznik & Kurtines, 1993) proposes that gaps between the adolescents' and caregivers' acculturation disrupt the basic emotional climate of the family. The original iteration of DAT was unidimensional, positing these gaps as emerging as adolescents, who are encouraged to adopt US cultural practices, beliefs, and norms in school, typically adopt US culture more rapidly than their caregivers, who typically settle into ethnic enclaves and avoid having to adapt to US culture (Schwartz, Pantin, Sullivan, Prado, & Szapocznik, 2006). More recent conceptualizations of DAT have employed a bidimensional approach, suggesting that family problems can emerge (a) when adolescents adopt US culture more than their caregivers or (b) when caregivers retain the heritage culture more than their adolescents do. Regarding US culture acquisition, caregivers may perceive certain US-based behaviors and/or attitudes their children have adopted as disrespectful or as a rejection of traditional ways of behaving (Portes & Rumbaut, 2006). In contrast, with regard to heritage-culture retention, adolescents' loss of their cultural heritage may make it difficult for adolescents and caregivers to communicate with each other.

Although unidimensional studies have provided mixed support, studies employing bidimensional approaches have supported DAT. For example, Unger et al. (2009) found that greater caregiver-youth discrepancies in US cultural practices were associated with recent cigarette use, whereas greater caregiver-youth discrepancies in both US and Hispanic cultural practices were associated with greater recent and lifetime alcohol and marijuana use. The effect of US discrepancies on substance use was partially mediated by levels of family cohesion. Additionally, using latent growth curve modeling, Schwartz et al. (2016) found that larger, and increasing, caregiver-adolescent gaps in Hispanic practices, collectivist values, and ethnic identity predicted poorer family functioning and greater risk for binge drinking. In contrast, larger and increasing gaps in individualist values and US identity predicted *more favorable* family functioning (and lower odds of binge drinking) over time.

As a whole, future research should continue to conceptualizing the family as a developmental system and recognize that adolescents and their caregivers are not only concurrently undergoing acculturation (Berry, 1980) but are actively working to influence each other's acculturation process. Thus, it is essential for future research to follow at least three research directions. First, it is important to further explore adolescent-to-caregiver and caregiver-to-adolescent ethnic

and US socialization process by identifying the direction(s) of effects and identifying potential differences in family interactional patterns that may promote or inhibit ethnic and US socialization. Second, it is essential to provide further empirical evidence for DAT across different immigrant groups and contexts, including contexts outside the United States. Third, we should endeavor to understand the relationship between adolescent–caregiver and caregiver–adolescent ethnic and US socialization attempts over time and determine how these attempts relate to increasing adolescent–caregiver differences in US and heritage cultural adoption/retention.

Further Conceptual and Theoretical Considerations

Bicultural Integration

Although the acculturation process represents a critical task for first- and second-generation immigrants, recent research has suggested that the acculturation process extends beyond simply adopting (or not) the receiving culture and retaining (or not) the heritage culture. More specifically, first- and second-generation immigrants are also tasked with reconciling and integrating potentially conflicting heritage and receiving cultural streams across each cultural component (i.e., practices, values, identification). An unwillingness or inability to integrate across cultural streams into a cohesive mosaic may lead to problematic outcomes. Taken together, the contemporary conceptualization of acculturation paints an increasingly complex model that includes both endorsement of heritage and receiving cultural streams separately for identification, practices, and values as well as the capacity for integrating each component across cultural streams (see Figure 1).

Bicultural identity integration (BII), or the capacity and willingness to blend (vs. compartmentalize) and harmonize (vs. view as being in conflict) two potentially divergent cultures,

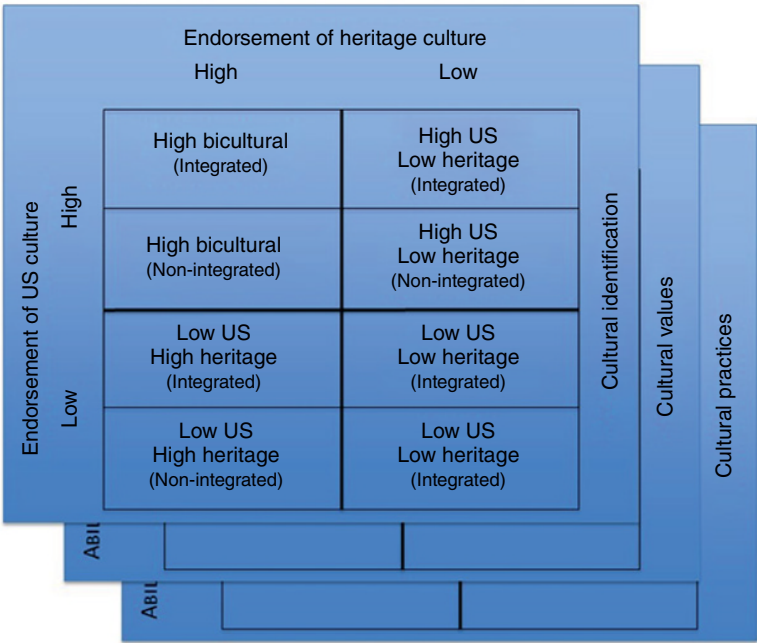


Figure 1 Multidimensional model of Acculturation. Note. The multiple layers represent a focus on several separate components of acculturation.

represents a domain of cultural integration that has received considerable attention, particularly in relation to psychosocial functioning. Indeed, a growing literature has evaluated the relationship between BII and mental health outcomes (for a review, see Huynh, Nguyen, & Benet-Martínez, 2011). Most recently, Schwartz et al. (2015) found that adolescents who maintained high levels of BII over time reported higher self-esteem, greater optimism, fewer depressive symptoms, and more favorable family relationships.

Despite this burgeoning area of research, there are a number of areas in which the links between BII and outcomes can be further extended and expanded. First, whether BII reflects an ability or capacity or a developmental construct remains a debated issue. Although recent evidence indicates that the ability to integrate multiple cultures may be stable over time (Schwartz et al., 2015), that study was limited to recently immigrated Hispanic adolescents. The findings may differ across ethnic groups, subgroups, and generation status. Additionally, and as previously stated, bicultural integration is not solely limited to cultural identification. Thus, a more complete conceptualization of acculturation will require research to extend beyond BII to cultural practices and values as well. Additionally, future studies should further explore the relationship between BII and cultural stressors. Indeed, biculturalism may represent a precarious situation where one feels pushed to adopt the receiving culture and pressured to retain one's heritage. Although experimental studies suggest individuals high in BII may be better equipped to handle cultural pressures (e.g., Mok & Morris, 2010), more research is necessary to evaluate the developmental relationship between acculturation, bicultural integration, cultural stress, and psychosocial functioning.

Acculturation as a "Multidimensional" Construct

Although contemporary models have shifted away from a unidimensional perspective and toward a bidimensional conceptualization of acculturation, some scholars have argued acculturation can involve more than two dimensions (Ferguson, Iturbide, & Gordon, 2014). Indeed, it is possible that individuals may draw on more than one heritage culture and/or immigrate to a context of reception that involves more than just one receiving culture, or a combination of the two. Scholars have typically embraced the term triculturalism to refer these situations; however, the number of distinct and recognizable cultural streams could extend beyond three. Triculturalism may emerge when ethnic-minority migrants (a) gravitate (or are directed) toward the receiving-society ethnic group to which they are most similar, (b) were part of a minority group within their country or region of origin, or (c) come to reside in a context where a hybrid "third" culture has emerged. For example, Chicano culture is a blend of Mexican and US cultures but is distinct from both, and British Indian culture is distinct from both traditional British and Indian cultural streams.

As a whole, triculturalism extends the bicultural conceptualization of acculturation. As such, for each component (practices, values, and identifications), the process of acculturation spreads across the multiple cultural streams in which the immigrant is contextually embedded. Concurrently, the immigrant is tasked with retention of one or more heritage cultures and adoption of one or more receiving cultures, as well as integrating these various cultural streams, all while coping with cultural stressors. Although triculturalism does introduce additional complexity for immigrants, preliminary research indicates that immigrants generally manage fairly well in these situations (Ferguson et al., 2014). However, additional research is necessary not only to identify the various cultural streams with which specific groups are confronted within specific contexts of reception but also to further explore how the increased difficulty and stressors involving three (or more) dimensional acculturation is associated with psychosocial functioning.

Methodological Concerns

Over and above these future theoretical and conceptual directions, it is critical for the field of acculturation to acknowledge (and deal with) prevalent methodological limitations that interfere with our efforts to understand the relationship between acculturation and drug use.

Understanding Effects of the Receiving Context

To begin with, as highlighted by Schwartz et al. (2010), many studies have made the often implicit assumption that each context of reception is equally welcoming and inviting. Context of reception refers to the individual's perception of the overall valence that the receiving society directs toward an immigrant group and the opportunity structure available to that group (Portes & Rumbaut, 2006). Although immigrants in a negative context of reception are likely to feel isolated, have difficulty finding jobs, and experience discrimination or perceive hostility, those in positive context of reception are able to aspire to succeed and achieve the "American Dream." Thus, immigration and acculturation can be conceptualized as the interaction between a specific immigrant group and the context in which they are received (Schwartz et al., 2010). Given the variability in the opportunity structure, degree of openness, hostility, and acceptance across receiving contexts (both within and across countries), it is important for research to evaluate and take into account the effects that different types communities have on immigrants.

Within-Group Diversity

Over and above the importance of context of reception, it is critical to acknowledge the high variability that exists within ethnic groups when it comes to examining acculturation. Inconsistent findings across studies may reflect a lack of consideration of these within-group differences. When studying Hispanic/Latino populations, the specific nationality of group members is important for many reasons. To begin with, immigrant groups such as Asians and Hispanics vary not just in terms of national origin but also in socioeconomic status, culture, dialect, history in the United States, skin tone, and ability to fit into mainstream US society, as well as many other factors. These factors may separately or jointly influence the acculturation process and/or the cultural stressors with which a person is likely to be confronted. Thus, these factors can result in very different immigrant experiences that may influence the acculturation process and therefore also influence psychosocial functioning, family relationships, and risks for substance use. Moreover, it is critical for future research to jointly take into account these within-group differences across various contexts. For example, the acculturation experience in Miami is likely different for Cubans, who account for the majority of the Hispanic population, have been aided by the "wet foot, dry foot" law that allows them to stay legally in the United States, and hold the majority of political and economic power (Stepick, Grenier, Castro, & Dunn, 2003), than for other Hispanic groups.

Measuring Substance Use

In addition to conceptualizing and measuring acculturation, there are important measurement considerations with regard to substance use. Specifically, it is essential to move beyond using simple dichotomous measures of drug use and composite scores that aggregate across different types of substances. For the most part, much of the literature looking at the relationship

between acculturation and substance use has relied on “yes/no” coding procedures for lifetime and/or current use. Although this often is done as a result of underreporting or low base rates, it is important to recognize that using solely dichotomized outcome variables obscures important developmental mechanisms. More specifically, using yes/no variables makes it impossible to differentiate between stages of substance use (e.g., initiation, experimental use, problematic). Additionally, it is essential for future research to avoid using “composite scores” that aggregate across multiple types of substances, thereby masking potentially theoretical and developmentally meaningful differences.

The Need for Longitudinal and Large Epidemiological Studies

Perhaps two of the greatest methodological concerns moving forward pertain to study design. Specifically, the majority of the acculturation literature has relied on cross-sectional studies and community samples. Given that acculturation, broadly, represents the process of change in immigrants’ cultural orientation as they adapt to life in a new cultural context, acculturation is inherently a developmental process (Schwartz et al., 2010). For this reason, it is essential for research to employ longitudinal studies that capture the progression of each component (across relevant cultural streams). Indeed, it is important to recognize that cultural components are interrelated but independent processes. Thus, change in each cultural component may proceed at different rates, in one component and not another, or in different directions (e.g., adoption of US cultural practices yet rejection of individualist values). Additionally, much of what we know about the relationship between acculturation and substance use emerges from disparate individual studies conducted in diverse parts of the United States. As a result, it is essential to design and implement large-scale epidemiological studies employing measures bidimensional, multicomponent, and well-validated measures of acculturation as opposed to unidimensional markers of acculturation and demographic proxies that previous epidemiological studies have traditionally employed (Schwartz et al., 2010).

Conclusion

As a whole, the acculturation literature suggests that losing one’s heritage practices, values, and identifications is more detrimental toward substance use than adopting US cultural practices, values, and identifications. However, the scarcity of empirical studies, coupled with conceptual and methodological problems outlined in this entry, has hampered our understanding of the processes linking acculturation with substance use. Although there is considerable work to be done, we hope that the present entry will serve to stimulate innovative and informative work that further illuminates the relationship between acculturation and substance use.

Author Biographies

Alan Meca is a postdoctoral associate in the Department of Public Health Sciences at the University of Miami Miller School of Medicine. His research is centered around personal and cultural identity, immigration and acculturation, health risk behaviors, and promoting positive and progressive developmental change during adolescence and emerging adulthood.

Seth J. Schwartz is professor of Public Health Sciences at the University of Miami Miller School of Medicine. His research interests are in identity, immigration and cultural adaptation,

family relationships, well-being, and health risk behaviors in adolescents and emerging adults. He is the incoming president of the Society for the Study of Emerging Adulthood and senior editor of the *Handbook of Identity Theory and Research* (2011) and of the forthcoming *Handbook of Acculturation and Health*.

References

- Berry, J. W. (1980). Acculturation as varieties of adaptation. In A. M. Padilla (Ed.), *Acculturation: Theory, models, and some new findings* (pp. 9–25). Boulder, CO: Westview.
- Deng, S., Kim, S. Y., Vaughan, P. W., & Li, J. (2010). Cultural orientation as a moderator of the relationship between Chinese American adolescents' discrimination experiences and delinquent behaviors. *Journal of Youth and Adolescence*, 39, 1027–1040. doi:10.1007/s10964-009-9460-6
- Ferguson, G. M., Iturbide, M. I., & Gordon, B. P. (2014). Tridimensional (3D) acculturation: Ethnic identity and psychological functioning of tricultural Jamaican immigrants. *International Perspectives in Psychology*, 3, 238–251. doi:10.1037/ipp0000019
- Gordon, M. M. (1964). *Assimilation in American life: The role of race, religion, and national origins* (pp. 117). New York, NY: Oxford University Press.
- Grieco, E. M., Acosta, Y. D., de la Cruz, G. P., Gambino, C., Gryn, T., Larsen, L. J., ... Walters, N. P. (2012). The foreign-born population in the United States: 2010. *American Community Survey Reports*, 19, 1–22.
- Hofstede, G. (2001). *Culture's consequences* (2nd ed.). London, UK: Sage.
- Huynh, Q.-L., Nguyen, A.-M. D., & Benet-Martínez, V. (2011). Bicultural identity integration. In S. J. Schwartz, K. Luyckx, & V. L. Vignoles (Eds.), *Handbook of identity theory and research* (pp. 827–844). New York, NY: Springer.
- Juang, L. P., & Cookston, J. T. (2009). Acculturation, discrimination, and depressive symptoms among Chinese American adolescents: A longitudinal study. *Journal of Primary Prevention*, 30, 475–496. doi:10.1007/s10935-009-0177-9
- Mok, A., & Morris, M. W. (2010). An upside to bicultural identity conflict: Resisting groupthink in cultural ingroups. *Journal of Experimental Social Psychology*, 46, 1114–1117. doi:10.1016/j.jesp.2010.05.020
- Padilla, A. M. (2006). Bicultural social development. *Hispanic Journal of Behavioral Sciences*, 28(4), 467–497.
- Portes, A., & Rumbaut, R. G. (2006). *Immigrant America: A portrait* (3rd ed.). Berkeley, CA: University of California Press.
- Schwartz, S. J., Pantin, H., Sullivan, S., Prado, G., & Szapocznik, J. (2006). Nativity and years in the receiving culture as markers of acculturation in ethnic enclaves. *Journal of Cross-Cultural Psychology*, 37(3), 345–353.
- Schwartz, S. J., Unger, J. B., Baezconde-Garbanati, L., Benet-Martínez, V., Meca, A., Zamboanga, B. L., ... Szapocznik, J. (2015). Longitudinal trajectories of bicultural identity integration in recently immigrated Hispanic adolescents: Links with mental health and family functioning. *International Journal of Psychology*, 50(6), 440–450.
- Schwartz, S. J., Unger, J. B., Baezconde-Garbanati, L., Zamboanga, B. L., Córdova, D., Lorenzo-Blanco, E. I., ... Szapocznik, J. (2016). Testing the parent–adolescent acculturation discrepancy hypothesis: A five-wave longitudinal study. *Journal of Research on Adolescence*, 26(3), 567–586.
- Schwartz, S. J., Unger, J. B., Zamboanga, B. L., & Szapocznik, J. (2010). Rethinking the concept of acculturation: Implications for theory and research. *The American Psychologist*, 65(4), 237–251. doi:10.1037/a0019330.
- Schwartz, S. J., Weisskirch, R. S., Zamboanga, B. L., Castillo, L. G., Ham, L. S., Huynh, Q. L., ... Cano, M. A. (2011). Dimensions of acculturation: Associations with health risk behaviors among college students from immigrant families. *Journal of Counseling Psychology*, 58(1), 27–41. doi:10.1037/a0021356

- Staerklé, C., Sidanius, J., Green, E. G. T., & Molina, L. E. (2010). Ethnic minority-majority asymmetry in national attitudes around the world: A multilevel analysis. *Political Psychology*, 31, 491–519. doi:10.1111/j.1467-9221.2010.00766.x
- Stepick, A., Grenier, G., Castro, M., & Dunn, M. (2003). *This land is our land: Immigrants and power in Miami*. Berkeley, CA: University of California Press.
- Szapocznik, J., & Kurtines, W. M. (1993). Family psychology and cultural diversity: Opportunities for theory, research, and application. *American Psychologist*, 48(4), 400–407. doi:10.1037/0003-066X.48.4.400
- Tran, A. G., Lee, R. M., & Burgess, D. J. (2010). Perceived discrimination and substance use in Hispanic/Latino, African-born Black, and Southeast Asian immigrants. *Cultural Diversity and Ethnic Minority Psychology*, 16(2), 226–236. doi:10.1037/a0016344
- Umaña-Taylor, A. J., Bhanot, R., & Shin, N. (2006). Ethnic identity formation during adolescence: The critical role of families. *Journal of Family Issues*, 27, 390–414. doi:10.1177/0192513X05282960
- Unger, J. B., Ritt-Olson, A., Soto, D. W., & Baezconde-Garbanati, L. (2009). Parent–child acculturation discrepancies as a risk factor for substance use among Hispanic adolescents in Southern California. *Journal of Immigrant and Minority Health*, 11(3), 149–157. doi:10.1007/s10903-007-9083-5
- Unger, J. B., Schwartz, S. J., Huh, J., Soto, D. W., & Baezconde-Garbanati, L. (2014). Acculturation and perceived discrimination: Predictors of substance use trajectories from adolescence to emerging adulthood among Hispanics. *Addictive Behaviors*, 39(9), 1293–1296. doi:10.1016/j.addbeh.2014.04.014
- Zamboanga, B. L., Schwartz, S. J., Jarvis, L. H., & Van Tyne, K. (2009). Acculturation and substance use among Hispanic early adolescents: Investigating the mediating roles of acculturative stress and self-esteem. *The Journal of Primary Prevention*, 30, 315–333.

Suggested Reading

- Ferguson, G. M., Iturbide, M. I., & Gordon, B. P. (2014). Tridimensional (3D) acculturation: Ethnic identity and psychological functioning of tricultural Jamaican immigrants. *International Perspectives in Psychology*, 3, 238–251. doi:10.1037/ipp0000019
- Huynh, Q.-L., Nguyen, A.-M. D., & Benet-Martínez, V. (2011). Bicultural identity integration. In S. J. Schwartz, K. Luyckx, & V. L. Vignoles (Eds.), *Handbook of identity theory and research* (pp. 827–844). New York, NY: Springer.
- Schwartz, S. J., Unger, J. B., Baezconde-Garbanati, L., Zamboanga, B. L., Córdova, D., Lorenzo-Blanco, E. I., ... Szapocznik, J. (2016). Testing the parent-adolescent acculturation discrepancy hypothesis: A five-wave longitudinal study. *Journal of Research on Adolescence*, 26(3), 567–586.
- Schwartz, S. J., Unger, J. B., Baezconde-Garbanati, L., Zamboanga, B. L., Lorenzo-Blanco, E. I., ... Szapocznik, J. (2015). Trajectories of cultural stressors and effects on mental health and substance use among Hispanic immigrant adolescents. *Journal of Adolescent Health*, 56, 433–439.
- Schwartz, S. J., Unger, J. B., Zamboanga, B. L., & Szapocznik, J. (2010). Rethinking the concept of acculturation: Implications for theory and research. *The American Psychologist*, 65(4), 237–251. doi:10.1037/a0019330.

Accurate Interpersonal Perception

Lauren J. Human

Department of Psychology, McGill University, Montreal, QC, Canada

Social relationships play an important role in individual functioning, shaping both psychological and physical health (e.g., Cohen, 2004). However, the more specific relationship processes that underlie these broader associations remain to be understood. One factor that may play an important role is the accuracy of interpersonal perceptions—how accurately we see and are seen by others. Specifically, being able to accurately perceive others' personality traits, such as kindness and sociability, and emotions, such as happiness and anxiety, may benefit our social interactions and relationships in ways that in turn promote better psychological and physical health. Further, being perceived accurately by others may also benefit our health and well-being. This entry considers these possibilities by selectively reviewing key research on mental and physical health-relevant correlates and consequences of accurate interpersonal perceptions, considering how accuracy relates to (a) relationship or social functioning, (b) psychological functioning, and (c) physiological functioning for both perceivers and targets.

Defining Accuracy

First, what does it mean for an interpersonal perception to be accurate? To determine whether perceptions of someone's personality or emotions are accurate, perceptions need to be compared to some type of accuracy validation measure or criterion. However, when forming impressions of real people, there is rarely one single objective criterion what for a person is truly like or what they are really feeling. As such, interpersonal perception research typically takes a “realistic accuracy” approach (Funder, 1995). Specifically, this approach utilizes a criterion, or combination of criteria, that represent a realistic approximation for what a person is like or what they are feeling, often relying upon self-reports, but also obtaining close other or behavioral indicators where possible. Thus, the degree of accuracy in a perceiver's impression of a target can be defined as the extent to which the impression maps on to a realistic indicator for what the target is like or what they are feeling. Of note, although interpersonal perceptions generally tend to contain some accuracy, there are individual differences in how accurately

people see and are seen by others (see Human & Biesanz, 2013). Thus, individuals may vary in how accurately they view and are viewed by others, and this variability may in turn have implications for social, psychological, and physical functioning.

A Brief Consideration of the Role of Bias

Although often accurate, interpersonal perceptions usually also involve some degree of bias, such as positive bias or assumed similarity. In turn, these biases may have their own implications for relationships and for psychological and physical health. Indeed, there is evidence that positivity bias and assumed similarity are associated with positive relationship processes, both in first impression contexts (e.g., Human, Sandstrom, Biesanz, & Dunn, 2013) and in romantic relationships (e.g., Luo & Snider, 2009). Given how important social relationships are to psychological and physical health (e.g., Cohen, 2004), such biases could therefore be very beneficial to individual functioning. As support, greater levels of positivity and assumed similarity in first impressions are associated with better psychological health, including higher levels of subjective well-being and self-esteem and lower levels of depression (Human & Biesanz, 2011). Thus, biased impressions of others may benefit psychological health, yet it remains to be seen whether such biases are in turn linked to physical health outcomes or related processes.

Importantly, accuracy and bias can be independent in interpersonal perceptions (see Gagné & Lydon, 2004 for review). For example, you may view a new acquaintance as more kind and intelligent than she actually is, but still be able to tell that she is more kind and less intelligent than another new acquaintance, thereby helping you to decide which person would be a better friend versus colleague. Thus, the argument that positive bias may benefit psychological and physical health does not imply that accuracy would be maladaptive. Instead, the potential consequences of accuracy may be independent of any consequences of bias.

Consequences of Accuracy: Potential Benefits

What are the consequences of accuracy for social, psychological, and physical functioning? This section will consider the evidence suggesting that accuracy may have benefits for each of these aspects of individual functioning, considering implications for both perceivers and targets.

Relationship or Social

There is growing evidence that accurate perceptions of others may have positive relationship or social consequences, independently of the benefits of positive bias or assumed similarity. In particular, among new classmates in large university courses, forming more accurate first impressions of personality was associated with greater liking initially and up to 3 months later and with spending more time together throughout the semester (Human et al., 2013). Thus, forming a more accurate first impression may facilitate relationship initiation. This may be because accurate first impressions could promote experiences such as familiarity, which fosters perceived knowing, increased social comfort during interactions, and, ultimately, greater liking (Reis, Maniaci, Caprariello, Eastwick, & Finkel, 2011). In turn, accuracy and corresponding experiences could set the stage for more positive and frequent future social interactions, promoting relationship development.

Furthermore, accuracy is argued to benefit close relationships by fostering communication and a shared reality (e.g., Ickes & Simpson, 1997). Indeed, greater accuracy in close relationships is associated with greater relationship satisfaction and positive outcomes (e.g., Luo & Snider, 2009; Neff & Karney, 2005), indicating that accuracy may promote relationship quality and maintenance. For example, more accurate personality perceptions among newlyweds promote a lower likelihood of divorce over time, at least for wives (Neff & Karney, 2005). Thus, accuracy does appear to carry positive social consequences, for both new and long-term relationships, although a better understanding of the mechanisms linking accuracy to positive social outcomes is needed.

Psychological Consequences

Given the generally positive links between accuracy and social outcomes, in both new and close relationships, it seems likely that accuracy could in turn be highly beneficial to the psychological health of the individuals involved in these social relationships—the perceivers and targets. But is there evidence to support this?

Perceivers

There are several reasons why perceiving others accurately could benefit psychological health. Specifically, the practical benefits of greater accuracy, such as improved communication, could enhance psychological well-being by enabling individuals to more effectively navigate their social worlds. In addition, the positive, relationship-enhancing consequences of accuracy could carry benefits to perceivers: if perceiving others accurately enhances relationship initiation and quality, then accuracy might in turn benefit perceiver psychological adjustment. Finally, forming accurate impressions of others may carry its own direct psychological benefits. For example, people tend to enjoy perceiving familiar stimuli (Reis et al., 2011), which accurate perceptions may facilitate. Thus, forming accurate perceptions of others may have social and psychological benefits that in turn promote mental health.

However, the evidence linking the formation of accurate perceptions to psychological health is mixed. On the one hand, there is evidence that forming more accurate perceptions of others' nonverbal behavior (e.g., reading emotion from facial expressions) is associated with better psychosocial functioning (see Hall, Andrzejewski, & Yopchick, 2009 for review). There is also evidence that forming more accurate personality perceptions is associated with better psychological functioning (Human & Biesanz, 2011). However, this appears to be due to forming more normative perceptions of others (e.g., viewing others as similar to the average personality profile), rather than achieving greater distinctive accuracy (an understanding of others' unique, differentiating traits). Thus, it is unclear whether accurate perceptions of specific others promote psychological adjustment or if a generalized understanding of other people is sufficient. Further, much of this work has been cross-sectional in nature, making it difficult to draw inferences about the direction of the associations between accuracy and psychological health.

Targets

Does being perceived more or less accurately relate to psychological health? Here, there is much stronger evidence that accuracy is associated with better psychological functioning. For example, individuals who are better nonverbal encoders (able to accurately express their

emotions nonverbally) tend to demonstrate better psychological adjustment (e.g., Friedman, Prince, Riggio, & DiMatteo, 1980). Further, greater psychological well-being is associated with having one's personality be more accurately perceived by new acquaintances and close others (see Human & Biesanz, 2013 for review). These findings are often interpreted as suggesting that well-being promotes being perceived more accurately, and there is prospective data to support this interpretation (Colvin, 1993). Nevertheless, it is also highly plausible that being perceived accurately in turn benefits well-being.

To elaborate, people who are viewed more accurately may experience boosts to well-being because of the indirect social and direct psychological benefits of being accurately perceived. Specifically, if being accurately perceived promotes being better liked in first impressions (Human & Biesanz, 2011; Human et al., 2013) and relationship quality in close relationships (Neff & Karney, 2005), then targets of accurate perceptions are likely to experience enhanced psychological well-being. Further, being accurately perceived could also directly benefit well-being by enhancing experiences of self-verification (being perceived in line with one's self-views). Indeed, self-verification can be a subjectively pleasing, sought-after experience, even when feedback is negative (see Kwang & Swann, 2010). Thus, the inter- and intrapersonal benefits of being perceived accurately may help to promote psychological health. Again, more research on this topic is needed, particularly research that can disentangle causality and examine mechanisms.

Biological Processes

Perceivers

To date, there has not been much direct examination of whether forming accurate perceptions of others is associated with physical health or health-relevant processes. However, one group has found that more accurate perceptions of a romantic partner's attitudes are associated with both greater relationship satisfaction and lower ambulatory blood pressure (e.g., Sanbonmatsu, Uchino, & Birmingham, 2011). Further, more indirect evidence comes from work on expressive suppression—the tendency to hide or refrain from expressing one's emotions to others. Specifically, interacting with a target who is engaging in suppression can result in enhanced physiological reactivity for perceivers, such as increased blood pressure responses (Butler et al., 2003). This negative effect of suppression on perceivers may be at least in part because suppression may hinder a perceiver's ability to accurately perceive the target, which could in turn be psychologically and physiologically distressing for perceivers. Over time, such negative physiological responses could accumulate to carry negative physical health consequences. Thus, perceivers who are not particularly adept at accurately perceiving others, or who tend to interact with targets who are difficult to perceive, may come to experience worse physical functioning.

Targets

There has also been relatively little work examining whether being perceived accurately relates to physical health, yet initial evidence suggests there may be links. For example, adolescents whose daily experiences are viewed more accurately by their parents demonstrate better inflammatory regulation (Human et al., 2014) in the form of glucocorticoid sensitivity—the extent to which cortisol is able to dampen the production of inflammatory proteins. In addition, there is evidence that receiving information that is self-verifying, even when negative, promotes

more positive physiological responses, such lower blood pressure reactivity (Ayduk, Gyurak, Akinola, & Mendes, 2013).

In addition, just as expressive suppression can elicit greater physiological reactivity in perceivers, there is also evidence that engaging in suppression can result in greater blood pressure reactivity for the target (Butler et al., 2003). Again, this may be driven in part by the stressful experience of not being able to accurately express oneself and in turn be accurately perceived. Thus, targets who have difficulty accurately expressing themselves to others, or who interact with perceivers who have difficulty accurately perceiving them, may experience negative physiological consequences. In contrast, accurately expressing oneself to others may promote more positive physiological profiles during social interactions, in turn promoting physical health over time. This is a critical area for future research, which would benefit from more experimental and longitudinal studies that examine additional relationship contexts as well as other biomarkers and clinical health outcomes.

Potential Downsides to Accuracy

Importantly, it is also possible that accurate interpersonal perceptions could carry some negative consequences. Specifically, greater accuracy in perceptions of close others' thoughts and feelings has been linked to worse relationship quality in some cases (e.g., Simpson, Orina, & Ickes, 2003). This is argued to occur when accuracy exposes individuals to relationship-threatening information (Ickes & Simpson, 1997). For example, accurate perceptions of a partner's relationship-threatening thoughts and feelings during a conflict discussion can lead to declines in feelings of closeness (Simpson et al., 2003). This could in turn have negative consequences for psychological and physical health. Indeed, more accurate perceptions of a romantic partner's relationship perceptions have been linked to greater depression (Overall & Hammond, 2012). Further, adolescents who more accurately perceive their parent's behaviors during interactions together, which may contain relationship-threatening information, report worse psychological functioning and exhibit pro-inflammatory profiles (Human et al., 2016). Although the direction of the associations is unclear in these studies, it seems plausible that perceiving others accurately may carry costs to psychological and physical health, at least when accuracy exposes individuals to negative relationship information.

In addition, it is possible that being perceived accurately could have negative implications in some cases. In particular, if one's relationship-threatening thoughts or behaviors are accurately perceived by a close other, this could be detrimental to the target as well. More generally, having one's negative traits and experiences be perceived by others may have negative consequences, especially when rejection risk is high (Kwang & Swann, 2010). For example, those with low self-esteem feel less valued by their dating partner when they disclose a personal failure to them (Cameron, Holmes, & Vorauer, 2009). Thus, in some cases, accurate self-expression and, in turn, being seen accurately may backfire, resulting in negative social, psychological, and physiological consequences for targets.

However, when people high in self-esteem disclose negative information, they experience positive social and psychological consequences (Cameron et al., 2009). Further, even individuals who may be most at risk to experiencing negative consequences by being viewed accurately (e.g., those with negative self-views) do not always experience such downsides. For example, although individuals who are lower in self-esteem feel less valued when they disclose a failure, their dating partner does not actually appear to perceive them less positively (Cameron et al., 2009). Further, individuals with more negative self-views have more positive implicit and behavioral responses to

negative than positive feedback (Ayduk et al., 2013). Overall, then, more accurate interpersonal perceptions may have some negative consequences for relationships, perceivers, and targets, but in general accuracy seems to be more beneficial than not. Perhaps the ideal scenario is to pair accurate perceptions with positively biased perceptions, which is both possible and can have synergistically positive associations with social processes (e.g., Luo & Snider, 2009).

Conclusion

Overall, interpersonal perceptions play a central role in social and psychological functioning, and there is preliminary evidence that such perceptions may in turn play a role in biological processes related to physical health. In general, but with some exceptions, accurate interpersonal perceptions appear to promote positive social outcomes, which in turn may benefit both perceiver and target psychological and physical health. Although to date there is little work directly examining the links between accurate perceptions and biological outcomes, initial work suggests accurate perceptions are linked to more positive physiological profiles, making this an important topic for future research. In particular, longitudinal and experimental studies that track the influence of accuracy on health-relevant biological processes (e.g., cardiovascular, endocrine, and immune functioning) and clinical health outcomes are needed. In sum, examining the role of accuracy in interpersonal perceptions may help shed light on how social interactions and relationships come to influence mental and physical health.

Author Biography

Lauren J. Human is an assistant professor in the Psychology Department at McGill University. Dr. Human's work examines the causes and consequences of accurate interpersonal perceptions, with a focus on the role of social, psychological, and biological functioning.

References

- Ayduk, O., Gyurak, A., Akinola, M., & Mendes, W. B. (2013). Consistency over flattery: Self-verification processes revealed in implicit and behavioral responses to feedback. *Social Psychological and Personality Science*, 4, 538–545. doi:10.1177/1948550612471827
- Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion*, 3, 48–67. doi:10.1037/1528-3542.3.1.48
- Cameron, J. J., Holmes, J. G., & Voraue, J. D. (2009). When self-disclosure goes awry: Negative consequences of revealing personal failures for lower self-esteem individuals. *Journal of Experimental Social Psychology*, 45(1), 217–222. doi:10.1016/j.jesp.2008.09.009
- Cohen, S. (2004). Social relationships and health. *American Psychologist*, 59, 676–684. doi:10.1037/0003-066X.59.8.676
- Colvin, C. R. (1993). Childhood antecedents of young-adult judgability. *Journal of Personality*, 61, 611–635. doi:10.1111/j.1467-6494.1993.tb00784.x
- Friedman, H. S., Prince, L. M., Riggio, R. E., & DiMatteo, M. R. (1980). Understanding and assessing nonverbal expressiveness: The affective communication test. *Journal of Personality and Social Psychology*, 39, 333–351. doi:10.1037/0022-3514.39.2.333
- Funder, D. C. (1995). On the accuracy of personality judgment: A realistic approach. *Psychological Review*, 102, 652–670. doi:10.1037/0033-295X.102.4.652

- Gagné, F. M., & Lydon, J. E. (2004). Bias and accuracy in close relationships: An integrative review. *Personality and Social Psychology Review*, 8, 322–338.
- Hall, J. A., Andrzejewski, S. A., & Yopchick, J. E. (2009). Psychosocial correlates of interpersonal sensitivity: A meta-analysis. *Journal of Nonverbal Behavior*, 33, 149–180. doi:10.1007/s10919-009-0070-5
- Human, L. J., & Biesanz, J. C. (2011). Through the looking glass clearly: Accuracy and assumed similarity in well-adjusted individuals' first impressions. *Journal of Personality and Social Psychology*, 100(2), 349–364.
- Human, L. J., & Biesanz, J. C. (2013). Targeting the good target: An integrative review of the characteristics and consequences of being accurately perceived. *Personality and Social Psychology Review*, 17, 248–272.
- Human, L. J., Chan, M., DeLongis, A., Roy, L., Miller, G. E., & Chen, E. (2014). Parental accuracy regarding adolescent daily experiences: Relationships with adolescent psychological adjustment and inflammatory regulation. *Psychosomatic Medicine*, 76, 603–610.
- Human, L. J., Chan, M., Ifthikhar, R., Williams, D., DeLongis, A., & Chen, E. (2016). Accuracy and positivity in adolescent perceptions of parent behavior: Links with adolescent psychological and inflammatory functioning. *Social Psychological and Personality Science*, 7, 796–805. doi:10.1177/1948550616660590
- Human, L. J., Sandstrom, G. M., Biesanz, J. C., & Dunn, E. W. (2013). Accurate first impressions leave a lasting impression: The long-term effects of accuracy on relationship development. *Social Psychological and Personality Science*, 4, 395–402.
- Ickes, W., & Simpson, J. A. (1997). Managing empathic accuracy in close relationships. In W. Ickes (Ed.), *Empathic accuracy* (pp. 218–250). New York, NY: Guilford Press.
- Kwang, T., & Swann, W. B., Jr. (2010). Do people embrace praise even when they feel unworthy? A review of critical tests of self-enhancement versus self-verification. *Personality and Social Psychology Review*, 14, 263–280. doi:10.1177/1088868310365876
- Luo, S., & Snider, A. (2009). Accuracy and biases in newlyweds' perceptions of each other: Not mutually exclusive but mutually beneficial. *Psychological Science*, 20, 1332–1339.
- Neff, L. A., & Karney, B. R. (2005). To know you is to love you: The implications of global adoration and specific accuracy for marital relationships. *Journal of Personality and Social Psychology*, 88, 480–497. doi:10.1037/0022-3514.88.3.480
- Overall, N. C., & Hammond, M. D. (2012). Biased and accurate: Depressive symptoms and daily perceptions within intimate relationships. *Personality and Social Psychology Bulletin*, 39, 636–650.
- Reis, H. T., Maniaci, M. R., Caprariello, P. A., Eastwick, P. W., & Finkel, E. J. (2011). Familiarity does indeed promote attraction in live interaction. *Journal of Personality and Social Psychology*, 101, 557–570. doi:10.1037/a0022885
- Sanbonmatsu, D. M., Uchino, B. N., & Birmingham, W. (2011). On the importance of knowing your partner's views: Attitude familiarity is associated with better interpersonal functioning and lower ambulatory blood pressure in daily life. *Annals of Behavioral Medicine*, 41, 131–137.
- Simpson, J. A., Orina, M. M., & Ickes, W. (2003). When accuracy hurts and when it helps: A test of the empathic accuracy model in marital interactions. *Journal of Personality and Social Psychology*, 85, 881–893.

Suggested Reading

- Funder, D. C. (2012). Accurate personality judgment. *Current Directions in Psychological Science*, 21, 177–182.
- Human, L. J., & Biesanz, J. C. (2013). Targeting the good target: An integrative review of the characteristics and consequences of being accurately perceived. *Personality and Social Psychology Review*, 17, 248–272.

- Human, L. J., Chan, M., DeLongis, A., Roy, L., Miller, G. E., & Chen, E. (2014). Parental accuracy regarding adolescent daily experiences: Relationships with adolescent psychological adjustment and inflammatory regulation. *Psychosomatic Medicine*, 76, 603–610.
- Neff, L. A., & Karney, B. R. (2005). To know you is to love you: The implications of global adoration and specific accuracy for marital relationships. *Journal of Personality and Social Psychology*, 88, 480–497. doi:10.1037/0022-3514.88.3.480
- Uchino, B. M., Sanbonmatsu, D. M., & Birmingham, W. (2013). Knowing your partner is not enough: Spousal importance moderates the link between attitude familiarity and ambulatory blood pressure. *Journal of Behavioral Medicine*, 36, 549–555. doi:10.1007/s10865-012-9437-x

Affective Forecasting

Elinor Flynn, Arpine Hovasapian, and Linda J. Levine

Department of Psychological Science, University of California Irvine, Irvine, CA, USA

People often face decisions that have major repercussions for their health and well-being. They must decide whether to undergo elective surgery or risk developing a hereditary disease, whether to take a vacation or put their money into savings, or whether to break up with their partner or stay put. To make such decisions, people do not merely tally objective costs and benefits. They also try to anticipate how happy or unhappy potential outcomes will make them. They select options that they expect will make them happy and avoid those that they expect will make them miserable. The greater the anticipated emotional impact of an outcome, the more effort people devote to attaining or avoiding it. Thus, predicting emotion, known as “affective forecasting,” plays a major role in decision making, and inaccurate predictions can lead to poor choices (Loewenstein, 2007). This entry reviews when and why people show biases in affective forecasting, over- or underestimating the emotional impact of future events, with an emphasis on forecasts related to health.

Affective Forecasting and Health

The accuracy with which people predict their future feelings has powerful implications for understanding a wide range of health issues including decisions about vaccination (Chapman & Coups, 2006), addiction (Loewenstein, 2005), genetic testing and cancer screenings (Peters, Laham, Pachter, & Winship, 2014), engaging in risky sexual behaviors (Ariely & Loewenstein, 2006), and end-of-life medical decision making (Ditto, Hawkins, & Pizzaro, 2005). In fact, people’s expectations about future feelings can even be the strongest predictor of their decisions about health. For example, university employees’ decisions to get a flu shot were predicted more strongly by their anticipated worry and regret should they later come down with the flu than by their perceived risk of actually getting the flu (Chapman & Coups, 2006).

Physicians also need to understand how predicted emotion influences patients’ decisions and that these predictions can be inaccurate. Moreover, physicians themselves may be biased

when predicting how their patients will respond emotionally to health-related outcomes. People can be subject to the same types of errors when forecasting others' emotional reactions as they are when forecasting their own (see Peters et al., 2014 for review). Policy makers evaluating the value and ethics of administering diagnostic tests for incurable conditions, such as Huntington's disease, can also benefit from an understanding of biases in affective forecasting. People's views about the value of such tests often depend on beliefs about the emotional impact of receiving positive test results. Thus, it is critically important for patients, healthcare providers, and policy makers to understand when and why predictions about future emotion are likely to be inaccurate and result in misguided or unsatisfying decisions.

Overestimating the Emotional Impact of Future Events

In pioneering work on affective forecasting, Timothy Wilson, Daniel Gilbert, and their colleagues have shown that whether people are predicting their reaction to the breakup of a relationship, achieving or being denied tenure, or the victory or loss of their favored sports team, they have a persistent tendency to overestimate the emotional impact of future events – an error referred to as the “impact bias.” People expect positive events to have a greater positive emotional impact than those events later turn out to have. They expect negative events to be more devastating than they later turn out to be (e.g., Gilbert, Pinel, Wilson, Blumberg, & Wheatley, 1998; Wilson & Gilbert, 2003). People have also been shown to overestimate the emotional impact of a wide range of medical conditions including paraplegia, hemophilia, kidney disease, and diabetes. People awaiting HIV test results overestimate how happy they will feel if the test comes back negative and how distressed they will feel if the test comes back positive (Peters et al., 2014).

Two key mechanisms have been proposed to explain overestimation in affective forecasting: focalism and adaptation neglect. Focalism refers to people's tendency to focus too much on salient features of a single emotion-eliciting event when they are predicting how they will feel. In doing so, they fail to consider that other more mundane concerns and events will also occupy their attention in the future and mitigate the intensity of their emotional response (Wilson, Wheatley, Meyers, Gilbert, & Axson, 2000). Schkade and Kahneman (1998) demonstrated this phenomenon in a study of college students' perceptions of how happy they would be if they lived in California versus the Midwest. Both Californians and Midwesterners predicted that people living in California would be more satisfied with life, when in fact the two groups rated their life satisfaction similarly. The discrepancy was due to the excessive focus of both groups on climate and the outdoors when making their predictions. When these features were prominent, predictors paid less attention to other factors such as academic opportunities, job prospects, and financial circumstances, which were among the variables rated most important to life satisfaction by individuals who were actually living in California and the Midwest.

Adaptation neglect also contributes to overestimation in affective forecasting (Gilbert et al., 1998; Wilson & Gilbert, 2003). When predicting the emotional impact of future events, particularly negative events, people tend to overlook the psychological resources and coping strategies they will draw on in the face of adversity to help them adjust to the new circumstances. Gilbert et al. (1998) found that across a wide range of situations, from failing to get tenure to receiving negative feedback about one's personality, people consistently overestimated the duration of negative emotion in part because they failed to anticipate the strength of their “psychological immune system” to combat distressed feelings.

Focalism and adaptation neglect can lead to inaccurate forecasts about the emotional impact of health outcomes. When healthy people predict what their general level of happiness would be if they had a serious illness, they often focus too much on their feelings specifically about that illness and underestimate the countervailing influence of other aspects of life, such as family and work, on their overall well-being. Healthy people also consistently overestimate the negative impact that becoming disabled would have on their well-being, a robust finding known as the “disability paradox” (Ubel, Loewenstein, & Jepson, 2005). Even after taking into account that disabled patients may adjust their interpretation of well-being and using momentary assessments of mood instead of relying on global self-reports, the well-being of disabled and chronically ill people is generally higher than healthy individuals expect it to be. Similarly, when people undergo medical testing, for instance, to determine whether they are carriers of genetic diseases, they tend to undergo a period of shock and distress after receiving a positive diagnosis, but later return to their baseline levels of well-being. Overestimating future distress can render patients risk averse and lead them to avoid diagnostics such as genetic testing and opt out of potentially beneficial treatments (Peters et al., 2014).

Overestimating future distress also makes it hard for people to predict their future preferences when making decisions about end-of-life medical treatment. For example, nursing home residents with varying levels of health and functioning were presented with health scenarios, such as being confined to bed or experiencing severe confusion (Winter, Moss, & Hoffman, 2009). The residents indicated their ideal number of future years of living with these conditions. Those who were currently in the best health indicated they would want to live significantly less time with these conditions than those in the worst health. Thus, healthier residents viewed declines in their health as more intolerable than did individuals who had experienced some of these health problems themselves. Healthier residents did not anticipate the extent to which people adapt to declining health and maintain the will to live. More generally, studies examining people’s desire for life-sustaining treatment have found considerable variation in their preferences over time, further calling into question people’s ability to accurately anticipate their future feelings and preferences (Ditto et al., 2005).

Investigators have explored how to promote more accurate forecasts. Focusing narrowly on the most uncomfortable or stressful elements of a condition can lead people to fail to consider the many aspects of their lives that will remain unaffected by their condition. Consequently, one strategy for reducing focalism and improving forecasts asks people to consider the wide range of activities and situations they will be engaged in before predicting their future level of emotional well-being (e.g., Wilson et al., 2000). Interestingly, although this strategy has proved successful in other situations, defocusing exercises have been shown to be ineffective, and even to backfire, in forecasts about disability. Asking people to imagine how becoming paraplegic or undergoing amputation below the knee would impact various aspects of their life, including spiritual life, romantic relationships, and social ties, actually resulted in *lower* subsequent estimates of well-being (Ubel et al., 2005). Thus, defocusing may not be a promising way to help people think about chronic disability.

Interventions aimed at reducing adaptation neglect have proven more successful than those aimed at reducing focalism for promoting accurate forecasts of emotional responses to illness and disability. When participants reflected on a prior life event that had inspired great happiness or great sadness and then considered whether those feelings had become stronger or weaker over time, they predicted that their quality of life, if they were to become paraplegic, would be significantly higher (Ubel et al., 2005). These findings suggest that for improving affective forecasts concerning health, interventions that enhance awareness of adaptation may be more fruitful than interventions that reduce focalism. Halpern & Arnold (2008) advised

that when consulting with a patient who has just received a difficult diagnosis, physicians ought to first attend to the patient's emotional distress and then encourage the person to reflect on coping strategies they used to get through other challenges in life. Urging patients to allow a significant amount of time to pass between an initial consideration of healthcare options and making a final decision may also combat adaptation neglect.

In summary, events often elicit short-lived emotional responses that are followed by relatively quick adaptation. As people's goals and expectations change, and as other events capture their attention, they think about the focal event less often. But people often fail to take these changes into account when predicting their emotional responses. They expect to think about positive and negative outcomes more than they actually do (focalism) and fail to anticipate how quickly they will adapt to negative outcomes (adaptation neglect). As a result, they tend to overestimate the impact that events will have on their overall emotional well-being (e.g., Gilbert et al., 1998; Wilson & Gilbert, 2003; Wilson et al., 2000).

Underestimating the Emotional Impact of Future Events

Although people often overestimate the emotional impact of future events, the opposite bias has also been demonstrated. Research on "empathy gaps" shows that when people are in a "cold," affectively neutral state, it can be hard for them to anticipate the intensity of future "hot" emotional and physiological states. As a result, they underestimate the power of those future feelings to drive their thinking, preferences, and behaviors (e.g., Loewenstein, 2007; Van Boven, Loewenstein, & Dunning, 2005). People have been shown to underestimate the intensity of their future urges to eat, smoke, drink alcohol, gamble in Las Vegas, and engage in risky or unethical sexual behavior (see Loewenstein, 2007 for review). For example, compared with men who were not aroused, men who were sexually aroused reported being more likely to be able to imagine having unprotected sex, being attracted to a 12-year-old girl, and slipping a woman a drug on a date (Ariely & Loewenstein, 2006).

People also underestimate the intensity of both emotional pain, such as embarrassment, and physical pain. Van Boven et al. (2005) found that people underestimated the influence embarrassment would have on their own and others' willingness to engage in public performances such as miming or dancing. Similarly, Read and Loewenstein (1999) evaluated participants' willingness to undergo a painful task in the laboratory (immersing a hand in ice water) for monetary payment. Participants who had immersed their hand in ice water a week prior asked for significantly less compensation than those who had immersed their hand just moments before. A group who had never done the task demanded the lowest payment of all. Although participants understood that the task was painful, those who had completed it a week ago, or had never completed it, had a hard time recalling or imagining the degree of pain and consequently underestimated the intensity of future pain.

Even after recognizing forecasting errors, people do not permanently adjust their preferences. Christensen-Szalanski (1984) asked pregnant women about their preferences regarding the use of anesthesia during childbirth at several time points: weeks before giving birth, while in labor, and afterward. Weeks before going into labor and during the early stages of labor, many women expressed a preference for natural childbirth without anesthesia. However, as the labor continued and their discomfort increased, the women's preferences shifted toward desiring medication. The women had experienced an empathy gap between their idea of how natural childbirth would feel and how it felt in reality. Most striking, however, were the women's evaluations 1 month after childbirth. By this point, their preferences had returned to their

pre-labor position, and they again favored childbirth without anesthesia, which suggests that they again underestimated the level of pain they had experienced during childbirth.

Interpersonal empathy gaps may arise between physician and patient when discussing treatment options. For instance, physicians in an affectively “cold” state may fail to appreciate the degree of pain a patient is experiencing and therefore under-prescribe pain medication. Empathy gaps may also help to explain the callous bedside manner of certain physicians, a source of complaint for many patients and their families. After many years of handling similar cases, physicians may have adapted to negative health outcomes and thus may have difficulty anticipating the emotional impact of the news they bear (Loewenstein, 2005).

Identifying People’s Forecasting Strengths and Weaknesses

Findings concerning the impact bias and empathy gaps reveal a clear tension in the research literature on affective forecasting. It is highly unlikely that people would show an impact bias, overestimating the emotional impact of future events, and simultaneously show an empathy gap, underestimating the impact of future emotion on their thinking, preferences, and behavior. For instance, if people predict that singing in public will be more embarrassing than it later turns out to be (impact bias), it is unlikely that they will underestimate their reluctance to sing (empathy gap). Moreover, people sometimes forecast emotion with striking accuracy (Levine, Lench, Kaplan, & Safer, 2012, 2013). To address these inconsistencies, researchers have examined the context in which an emotional experience occurs, the specific features of emotion people are asked to predict, and the procedures that researchers use to assess forecasting accuracy. This work sheds light on important moderators of the magnitude and direction of affective forecasting biases.

Lench, Safer, and Levine (2011) found that focalism can lead to either over- or underestimating the emotional impact of events depending on the context in which the event occurs. Students who were in a romantic relationship predicted how they would feel if they broke up with their partner. One group predicted how they would feel on February 7th and the other group predicted how they would feel on February 14th. Weeks later, those who had broken up with their partners reported their actual feelings on either February 7th or 14th. Students who rated their levels of distress on February 7th showed the expected impact bias—they had exaggerated how distressed they would feel. But students who rated their feelings on February 14 felt *worse* than they had anticipated, showing a reversal of the impact bias. When making their predictions, students underestimated how the connotations surrounding Valentine’s Day would conspire to focus their attention on their lack of a relationship and intensify their negative emotions. Thus, the context in which an event occurs can determine the direction of bias in affective forecasting.

The magnitude and direction of forecasting bias also depends on the feature of emotion people are trying to predict: intensity versus duration or frequency. The impact bias is believed to characterize predictions about all of these features of emotion, and most forecasting studies do not distinguish between them (Wilson & Gilbert, 2003). However, in a series of studies and a meta-analysis of forecasting research, Levine et al. (2012, 2013) showed that although people overestimated when making judgments that encompassed the *duration* of future emotion (how happy they would feel in general), they were strikingly accurate when judging the *intensity* of future emotion (how happy would they will feel about a specific event). For example, people overestimated their reaction to the outcome of the 2008 US presidential election when they were later asked to report how they were feeling in general without reference to the

election. This bias was eliminated, and participants were far more accurate, when they were asked, "How happy are you feeling about Barack Obama being elected President?" Indeed, they showed a slight but significant tendency to underestimate emotional intensity, revealing an empathy gap rather than impact bias. These findings suggest that mispredicting the duration or frequency of emotion, rather than its peak intensity, accounts for people's tendency to overestimate the emotional impact of events.

This distinction has important implications for decision making. Peak intensity provides an index of how good or how bad an experience will be, whether one has the resources to cope with it, and the amount of effort it is worth expending to achieve or avoid it (Fredrickson, 2000). Indeed, the intensity of emotion people expect to feel while an experience is occurring is often a primary determinant of whether they engage in an activity or avoid it (e.g., potentially painful medical procedures). Even when making life-altering decisions concerning career, marriage, and children, people consider the peak positive and negative feelings that different choices will bring about, as well as longer term effects on their emotional well-being. Thus, accuracy in predicting emotional intensity bodes well for decision making (Levine et al., 2012, 2013).

Further research is needed, but three factors may contribute to greater accuracy in predictions of emotional intensity compared to duration or frequency. First, as emotional intensity increases, attention narrows to focus on central or salient features of events at the expense of more peripheral features (for a review, see Levine & Edelstein, 2009). People also tend to focus on an event's salient features when predicting the intensity of their feelings. Thus, the features of an emotion-eliciting event that are central at the time when people are attempting to predict their future emotional reaction to an event are also likely to be salient when they later experience the event, promoting accurate forecasts. Second, people may draw on their appraisals of an event's importance when attempting to predict the intensity of their emotional response. Stability in people's appraisals of the importance of events for their goals would contribute to accuracy in predicting the intensity of emotion. Third, the peak intensity of past emotional episodes is remembered more accurately than their duration (e.g., Fredrickson & Kahneman, 1993), providing a better basis for prediction (Levine et al., 2012, 2013).

Finally, the degree of bias people display in forecasting emotion depends on how researchers conceptualize accuracy and on the procedures they use to assess accuracy. A meta-analysis of affective forecasting studies showed that although people are often inaccurate in their forecasts in an absolute sense, they are more accurate in a relative sense (Mathieu & Gosling, 2012). That is, people who predict they will feel the greatest emotion often end up feeling the greatest emotion, even if the absolute degree of emotion experienced ends up being less than expected.

Levine et al. (2012) found that procedures commonly used to assess forecasting accuracy can artificially increase the magnitude of the impact bias. In most forecasting studies, researchers ask participants to imagine that an event has occurred and rate how they will feel in general after a specified period of time. After that period of time has passed, researchers ask participants to report how they are feeling in general without reference to the focal event. Levine et al. found that in the context of having just been asked to imagine a specific future event, most participants interpret the request to predict how they will feel *in general* as asking how they will feel when they are later thinking about that specific event. When the intended meaning of the question was clarified, participants still overestimated the impact of events on their general emotional state, but the magnitude of bias was dramatically reduced. Thus, future research on affective forecasting should assess people's ability to predict specific features of their emotional experiences and ensure that they clearly understand the features they have been asked to predict.

In conclusion, life is much harder when one cannot foresee what lies ahead, and people's inability to accurately predict the emotional consequences of their choices can lead to poor decisions and unhappiness. The research literature contains conflicting findings concerning the magnitude and direction of forecasting biases. However, taking a more nuanced approach to investigating people's ability to predict specific features of emotion helps to resolve these seemingly contradictory findings and sheds light on people's forecasting strengths and weaknesses. Recent findings suggest that people are fairly accurate at predicting the intensity of emotion they will experience in the future when emotion-eliciting events come to mind and may even underestimate that intensity (Lench et al., 2011; Levine et al., 2012). But people tend to exaggerate how frequently they will think about emotional events, and they neglect to consider the strategies they will use to cope with their emotions. As a result, they often overestimate the frequency and duration of their emotional responses (Wilson & Gilbert, 2003, 2005). Given that people predict some features of emotion more accurately than others, further research is needed to identify which features people spontaneously forecast in order to make decisions in their daily lives. Understanding sources of accuracy and bias in affective forecasting can help healthcare professionals guide people as they confront decisions concerning predictive genetic testing, end-of-life medical provisions, treatments for addiction, and other health-related issues.

Author Biographies

Elinor Flynn is a doctoral student in the Psychology and Social Behavior Department at the University of California, Irvine. She received a BA with honors in English from Princeton University. Her research focuses on the factors that influence the development of children's emotion regulation, how emotions influence judgment and decision making, accuracy and bias in predicted emotion, and moral psychology.

Arpine Hovasapian is a doctoral candidate in the Psychology and Social Behavior Department at the University of California, Irvine, where she also received an MA in social ecology. Her research investigates the processes by which emotions are expressed, communicated, and regulated, with a focus on the interpersonal context. Specifically, she studies the nonverbal expression of pride and joy, how sharing life experiences with others influences emotions, and the effects of reappraisal on memory for pain.

Linda J. Levine is a professor of psychology and social behavior at the University of California, Irvine. She received an MEd from Harvard University and PhD from the University of Chicago, both in psychology. Her research focuses on the cognitive and motivational processes that evoke emotions; how emotions influence the features of events people attend to, find important, and later remember; biases in predicting and remembering emotions; and the development of children's ability to regulate emotions.

References

- Ariely, D., & Loewenstein, G. (2006). The heat of the moment: The effect of sexual arousal on sexual decision making. *Journal of Behavioral Decision Making*, 19, 87–98. doi:10.1002/bdm.501
- Chapman, G. B., & Coups, E. J. (2006). Emotions and preventive health behavior: Worry, regret, and influenza vaccination. *Health Psychology*, 25(1), 82–90. doi:10.1037/0278-6133.25.1.82
- Christensen-Szalanski, J. J. (1984). Discount functions and the measurement of patients' values: Women's decisions during childbirth. *Medical Decision Making*, 4, 47–58.

- Ditto, P. H., Hawkins, N. A., & Pizzaro, D. A. (2005). Imagining the end of life: On the psychology of advance medical decision making. *Motivation and Emotion*, 29, 481–502. doi:10.1007/s11031-006-9017-x
- Fredrickson, B. L. (2000). Extracting meaning from past affective experiences: The importance of peaks, ends, and specific emotions. *Cognition & Emotion*, 14, 577–606. doi:10.1080/026999300402808
- Fredrickson, B. L., & Kahneman, D. (1993). Duration neglect in retrospective evaluations of affective episodes. *Journal of Personality and Social Psychology*, 65, 45–55. doi:10.1037/0022-3514.65.1.45
- Gilbert, D. T., Pinel, E. C., Wilson, T. D., Blumberg, S. J., & Wheatley, T. P. (1998). Immune neglect: A source of durability bias in affective forecasting. *Journal of Personality and Social Psychology*, 75, 617–638. doi:10.1037/0022-3514.75.3.617
- Halpern, J., & Arnold, R. M. (2008). Affective forecasting: An unrecognized challenge in making serious health decisions. *Journal of General Internal Medicine*, 23, 1708–1712. doi:10.1007/s11606-008-0719-5
- Lench, H. C., Safer, M. A., & Levine, L. J. (2011). Focalism and the underestimation of future emotion: When it's worse than imagined. *Emotion*, 11, 278–285. doi:10.1037/a0022792
- Levine, L. J., & Edelman, R. S. (2009). Emotion and memory narrowing: A review and goal-relevance approach. *Cognition and Emotion*, 23, 833–875. doi:10.1080/02699930902738863
- Levine, L. J., Lench, H. C., Kaplan, R. L., & Safer, M. A. (2012). Accuracy and artifact: Reexamining the intensity bias in affective forecasting. *Journal of Personality and Social Psychology*, 103, 584–605. doi:10.1037/a0029544
- Levine, L. J., Lench, H. C., Kaplan, R. L., & Safer, M. A. (2013). Like Schrödinger's cat, the impact bias is both dead and alive: Reply to Wilson and Gilbert. *Journal of Personality and Social Psychology*, 105, 749–756. doi:10.1037/a0034340
- Loewenstein, G. (2005). Hot-cold empathy gaps and medical decision making. *Health Psychology*, 24, S49–S56. doi:10.1037/02786133.24.4.S49
- Loewenstein, G. (2007). Affect regulation and affective forecasting. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 180–203). New York, NY: Guilford Press.
- Mathieu, M. T., & Gosling, S. D. (2012). The accuracy or inaccuracy of affective forecasts depends on how accuracy is indexed: A meta-analysis of past studies. *Psychological Science*, 23, 161–162. doi:10.1177/0956797611427044
- Peters, S. A., Laham, S. M., Pachter, N., & Winship, I. M. (2014). The future in clinical genetics: Affective forecasting biases in patient and clinician decision making. *Clinical Genetics*, 85, 312–317.
- Read, D., & Loewenstein, G. (1999). Enduring pain for money: Decisions based on the perception and memory of pain. *Journal of Behavioral Decision Making*, 12(1), 1–17.
- Schkade, D. A., & Kahneman, D. (1998). Does living in California make people happy? A focusing illusion on judgments of life satisfaction. *Psychological Science*, 9, 340–346. doi:10.1111/1467-9280.00066
- Ubel, P. A., Loewenstein, G., & Jepson, C. (2005). Disability and sunshine: Can hedonic predictions be improved by drawing attention to focusing illusion or emotional adaptation? *Journal of Experimental Psychology: Applied*, 11, 111–123. doi:10.1037/1076-898X.11.2.111
- Van Boven, L., Loewenstein, G., & Dunning, D. (2005). The illusion of courage in social predictions: Underestimating the impact of fear of embarrassment on other people. *Organizational Behavior and Human Decision Processes*, 96, 130–141. doi:10.1016/j.obhdp.2004.12.001
- Wilson, T. D., & Gilbert, D. T. (2003). Affective forecasting. In M. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 35, pp. 345–411). New York, NY: Elsevier.
- Wilson, T. D., Wheatley, T., Meyers, J. M., Gilbert, D. T., & Axson, D. (2000). Focalism: A source of durability bias in affective forecasting. *Journal of Personality and Social Psychology*, 78(5), 821–836.
- Winter, L., Moss, M. S., & Hoffman, C. (2009). Affective forecasting and advance care planning. *Journal of Health Psychology*, 14, 447–456. doi:10.1177/1359105309102201

Suggested Reading

- Halpern, J., & Arnold, R. M. (2008). Affective forecasting: An unrecognized challenge in making serious health decisions. *Journal of General Internal Medicine*, 23, 1708–1712. doi:10.1007/s11606-008-0719-5
- Levine, L. J., Lench, H. C., Kaplan, R. L., & Safer, M. A. (2012). Accuracy and artifact: Reexamining the intensity bias in affective forecasting. *Journal of Personality and Social Psychology*, 103, 584–605. doi:10.1037/a0029544
- Loewenstein, G. (2005). Hot-cold empathy gaps and medical decision making. *Health Psychology*, 24, S49–S56. doi:10.1037/02786133.24.4.S49
- Peters, S. A., Laham, S. M., Pachter, N., & Winship, I. M. (2014). The future in clinical genetics: Affective forecasting biases in patient and clinician decision making. *Clinical Genetics*, 85, 312–317.
- Wilson, T. D., & Gilbert, D. T. (2003). Affective forecasting. In M. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 35, pp. 345–411). New York, NY: Elsevier.
- Wilson, T. D., & Gilbert, D. T. (2005). Affective forecasting: Knowing what to want. *Current Directions in Psychological Science*, 14(3), 131–134.

Attachment Processes and Health

Onawa P. LaBelle¹, Britney M. Wardecker¹, William J. Chopik², and Robin S. Edelstein¹

¹*Psychology Department, University of Michigan, Ann Arbor, MI, USA*

²*Psychology Department, Michigan State University, East Lansing, MI, USA*

Social relationships are a central component of human existence; they influence the way we think and act in nearly every situation. The *quality* of our relationships is also important for both mental and physical health. For example, people whose relationships are more stable and secure, and those with larger social networks, are generally happier and healthier. Yet not everyone approaches relationships in the same way. Some people are relatively self-reliant and independent, choosing to maintain distance from relationship partners; others continually seek support from their relationships and need relationship partners to be available at all times. How do these kinds of differences, also known as *individual differences in attachment*, influence physical health?

In this entry, we discuss how a person's *attachment orientation*, or characteristic approach toward close relationships, is associated with health behaviors and outcomes. Our main focus is individual differences in *adult* attachment and their implications for health, rather than early childhood attachment to a primary caregiver. We also focus on individual differences in close relationship processes, as opposed to close relationships more broadly (see Ge, Lembke, & Pietromonaco's entry in this volume for a review of intimate relationships and physical health outcomes). First, we provide an introduction to attachment theory and describe how social-personality researchers typically study attachment orientation in adults. Next, we discuss the state of current research linking adult attachment with health. We then turn our attention toward the mechanisms that may underlie these links to explain *how* and *why* individual differences in attachment might be related to physical health. We conclude by highlighting future theoretical and methodological directions that can help elucidate when and for whom attachment processes influence health.

What is Attachment?

Attachment theory was originally conceptualized to describe the emotional bond between an infant and his or her primary caregiver and the anxiety that occurs upon separation from that caregiver (Bowlby, 1969). Social-personality psychologists later noted the many similarities between a child's first relationship with caregivers and subsequent social relationships (e.g., with friends, small groups, coworkers, and especially romantic partners; Hazan & Shaver, 1987). Over the last several decades, these observations have contributed to the emergence of attachment theory as a dominant framework for understanding thoughts, feelings, and behaviors in close relationships across the lifespan.

Attachment researchers also highlight the importance of individual differences in the quality of close relationships across the lifespan, otherwise known as *attachment orientation*. These differences are generally conceptualized by a person's position on two relatively independent dimensions: attachment-related anxiety and avoidance. The *anxiety* dimension reflects "hyper-activation" of the attachment system and often manifests in an obsessive concern over the availability of attachment figures. People with higher levels of attachment anxiety tend to have more negative views about the self and others, worry about being abandoned, and become easily overwhelmed by interpersonal stressors (Mikulincer & Shaver, 2008). The *avoidance* dimension is characterized by chronic attempts to deactivate or inhibit the attachment system (Edelstein & Shaver, 2004). People with higher levels of avoidance have more negative views of others (but not necessarily the self), are uncomfortable with physical and emotional intimacy, and try to minimize the experience and expression of distress. People with low levels of both anxiety and avoidance are considered "secure" and are typically comfortable with interpersonal closeness and intimacy. Secure individuals are more likely to have relationships that are characterized by trust and commitment compared with their insecure (anxious and avoidant) counterparts. As described next, one's attachment orientation also plays a critical role in physical health throughout the lifespan.

State of the Current Research Linking Attachment Orientations with Physical Health

Individual differences in attachment have been consistently linked with physical health outcomes. Attachment-related anxiety is a particularly robust correlate of physical health conditions that involve the cardiovascular system, such as stroke and heart attack. For example, people with high levels of attachment anxiety may have lower levels of cardiac vagal tone (Diamond & Hicks, 2005), which has been linked with early mortality and other negative health outcomes. Associations between anxiety and cardiovascular function may be attributed, in part, to chronic exposure to high levels of stress, which can impact a variety of physiological processes. In this vein, anxious people also report more physical health symptoms, such as headaches and digestive issues (Feeney & Ryan, 1994); anxious individuals with a diagnosed illness, such as irritable bowel syndrome, also report more severe symptomology compared with less anxious people.

Attachment avoidance has been less consistently linked with physical health, but avoidant people may still be at greater risk for physical health problems. Characteristics of avoidant attachment—such as suppressing negative emotions—contribute to a number of health problems, especially those involving pain, such as migraines and rheumatoid arthritis. In addition,

like attachment anxiety, avoidance is associated with poor heart rate regulation. For instance, more avoidant individuals have decreased vagal tone (McWilliams & Bailey, 2010), which also puts them at higher risk for cardiovascular problems. However, much remains to be understood about the mechanisms that may underlie attachment-related differences in health, that is, *how* and *why* attachment may be associated with physical health outcomes. We turn to these potential mechanisms in the following sections.

Mechanisms Linking Individual Differences in Attachment with Physical Health Outcomes

How might individual differences in attachment influence physical health? In the following sections, we focus on four of the most commonly investigated mechanisms that have strong implications for health and well-being: social support, health behavior, emotion regulation, and physiological response systems.

Social Support

Attachment orientations have been linked with social support, which is a strong predictor of health. For instance, people with stronger social support networks consistently report fewer health problems, including fewer cardiovascular conditions, lower rates of infectious disease, and even lower mortality rates from cancer than those without such support. Importantly, attachment orientations influence people's perceptions about the *availability* of social support: anxious people often feel a lack of support from close others, despite evidence to the contrary; avoidant people are less likely to acknowledge that they need support at all (Ognibene & Collins, 1998). Securely attached people view others as reliable sources of support and tend to make accurate appraisals about the amount of social support that is available to them (Ognibene & Collins, 1998). Differences in beliefs about the availability of support could be an important link between attachment orientation and physical health outcomes.

Attachment-related differences in physiological responses to social interactions in everyday life can also affect physical health. For instance, anxious adults exhibit increases in blood pressure while engaging with friends, indicating a state of hyperarousal not typically seen among less anxious individuals (Gallo & Matthews, 2006). Avoidant people similarly show a marked increase in blood pressure during relationship conflict compared with those who are less avoidant (Gallo & Matthews, 2006), which may reflect attempts to stifle emotions or avoid intimate conversation. Although these individual differences in regulatory systems may seem minor at first glance, the cumulative effects of stress can have a major impact on health across the lifespan (Maunder & Hunter, 2001; Robles, Brooks, Kane, & Schetter, 2013). Yet, despite the importance of social interactions for health and well-being, surprisingly little research has been conducted to examine whether differences in social support or social interactions underlie attachment-related differences in physical health outcomes.

Health Behavior

Individual differences in attachment are associated with a range of behaviors that directly impact health, including diet and eating habits, smoking, risk taking, and sexual behavior. For example, people with higher levels of attachment anxiety report more distress about their

weight than less anxious individuals in the same weight range, indicating a preoccupation with body image. Yet anxious individuals report exercising *less* than their nonanxious counterparts (Ahrens, Ciechanowski, & Katon, 2012). Moreover, women with anorexia nervosa (restricting and binge-purge subtypes) or bulimia nervosa report significantly higher levels of anxiety and avoidance than women without a diagnosed eating disorder (Illing, Tasca, Balfour, & Bissada, 2010). Attachment anxiety is also associated with greater symptom severity and poorer treatment outcomes among women with an eating disorder, regardless of the subtype (Illing et al., 2010). These associations suggest that there may be some overlap in the mechanisms that contribute to attachment insecurity and disordered eating. Specifically, restrictive eating behavior may be a way to create distance from the self, a characteristic of avoidant attachment; overeating or bingeing, in contrast, may be an expression of unregulated emotion, which is related to anxious attachment (Zachrisson & Skårderud, 2010).

Avoidance is also associated with risky health behaviors, including smoking and not wearing a seat belt (Ahrens et al., 2012; Zachrisson & Skårderud, 2010), both of which can contribute to poor health outcomes. Failure to wear a seat belt increases the likelihood of suffering a serious injury in a car accident; long-term smoking sharply increases the probability of health issues, including breast, lung, and throat cancer (Silverberg & Ratner, 2015). Perhaps not surprisingly, both risk behaviors reflect some degree of compulsive self-sufficiency or self-reliance, a key characteristic of avoidant attachment. Smoking is often used to reduce anxiety; not wearing a seat belt could reflect the rejection of others' concerns about safety or of societal norms more generally.

Both attachment avoidance and anxiety have also been linked with risky sexual behavior, which could contribute to long-term health outcomes by increasing rates of sexually transmitted infections (STIs). As an example, anxious women are less likely to know details about their partners' sexual history and are more likely to have consensual but unwanted sexual encounters (Ahrens et al., 2012; Maunder & Hunter, 2001). These differences might reflect anxious women's need for acceptance and approval, which could make it difficult for them to say "no" to a partner or to engage in conversations about safer sex practices. Among young adults, attachment avoidance has been linked with endorsement of casual sex (Gentzler & Kerns, 2004) and increased likelihood of engaging in sexual intercourse with a stranger, which could increase risk for unplanned pregnancy and contracting STIs.

Finally, there are attachment-related differences in the extent to which people report health symptoms and follow instructions given by a physician. Although anxious people are more likely to report health issues than those who are secure or avoidant (Ahrens et al., 2012), they may become frustrated if medical problems persist and may even sabotage their own treatment (Feeney & Ryan, 1994). In contrast, avoidant people who experience chronic pain are less likely to seek help in general and may be less likely to comply with treatment (Mikail, Henderson, & Tasca, 1994). Secure people with chronic pain disorders are more likely to see a doctor, comply with a prescribed course of treatment, and seek social support when dealing with pain (Mikail et al., 1994). Thus, attachment-related differences in health behavior may be an important reason for attachment-related health disparities. Health interventions that align with a person's attachment orientation may, therefore, be more effective than general interventions. Perhaps anxious people would respond more favorably to group interventions (e.g., Weight Watchers or group therapy). Anxious individuals' need for approval from group members could increase their motivation to achieve health goals. Avoidant individuals, in contrast, might be more receptive to interventions that do not include others (e.g., self-education or informational videos).

It is important to note, however, that many studies of attachment-related differences in physical health behaviors utilize relatively small samples that are homogenous with respect to

participant age and other important demographics (e.g., social class). More research is needed with larger and more diverse samples to determine whether extant findings are generalizable to the population as a whole.

Emotion Regulation

When experiencing a strong emotion, such as anger, fear, or excitement, physiological alterations occur in multiple systems of the body (e.g., cardiac functioning, blood pressure, stress levels). Depending on how often and the degree to which an emotion is experienced, fluctuations in regulatory systems can have a direct impact on health. Moreover, insecurely attached people are more likely to use emotion regulation strategies that either exaggerate or inhibit emotions, which can have the consequence of *increasing* negative emotions, stress, and physiological reactivity.

For instance, anxious individuals are more likely to utilize strategies centered on managing negative emotions, such as rumination, and often envision worst-case scenarios when attempting to control emotions (Mikulincer & Shaver, 2008). Doubts about a partner's availability can trigger repetitive worry about abandonment that further exacerbate painful feelings and thoughts. Avoidant individuals learn early in life that expressing emotions or seeking support from others can lead to rejection. As a result, people with avoidant attachment orientations try to limit their distress by suppressing their emotions and by not seeking support in order to avoid rejection or unwanted closeness (Edelstein & Shaver, 2004). In contrast, people with a secure attachment orientation generally report less negative emotional reactions to distressing situations and are more likely to use support-seeking strategies to regulate their emotions, which, as described earlier, can have important health benefits.

The association between insecure attachment and poor emotion regulation skills may reflect the influence of stress on neural processes. Stress is linked with low cell density in the hippocampus, an area of the brain that assists in emotion regulation (Mikulincer & Shaver, 2008). Higher levels of stress may reduce the density of cells in the hippocampus and impact one's ability to effectively process and handle emotions. Similar to disorders that have accompanying emotion regulation difficulties (e.g., depression and PTSD), both anxious and avoidant attachment are associated with lower cell density in this region of the brain (Quirin, Gillath, Pruessner, & Eggert, 2010). However, it is not yet clear whether emotion regulation and stress lead to lower cell density, or whether lower cell density in this region of the brain leads to poor emotion regulation and stress (see section on biological mechanisms for further discussion of attachment and stress). Thus, more research is needed to determine the directionality of these associations.

In addition to reduced hippocampal cell density, attachment anxiety is associated with increased activity in the anterior temporal pole, a brain region associated with sadness and emotional memory retrieval (Quirin et al., 2010). Overstimulation of these regions, which can happen during relationship conflict, can make it difficult for people to recover memories. The association between anxious attachment and emotional memory retrieval may explain why anxious individuals find it difficult to recall positive memories during conflicts (Dolan, Lane, Chua, & Fletcher, 2000); this difficulty may, in turn, increase sadness and stress.

Physiological Response Systems

Biological processes may provide another important link between individual differences in attachment and health outcomes. When an environment is perceived as threatening or uncontrollable, the hypothalamic–pituitary–adrenal (HPA) axis is activated, and its product, the

steroid hormone cortisol, is released into the bloodstream. Although short-term stress responses are generally adaptive, more chronic HPA activation can be problematic for health. People with maladaptive stress response patterns are at greater risk of cardiovascular disease, stroke, and lower well-being. Importantly, there are attachment-related differences in the basic functioning of the HPA axis. For example, insecure individuals report more stress and display an abnormally high cortisol response during disagreements with their romantic partner compared with secure adults (Kafetsios & Sideridis, 2006). Attachment avoidance has also been associated with lower levels of estradiol (Edelstein, Kean, & Chopik, 2012), another steroid hormone that can help to attenuate physiological stress responses.

Individual differences in attachment also influence the body's ability to effectively defend against infection, disease, and illness. To illustrate, attachment anxiety is linked with multiple indicators of a disrupted immune response during relationship conflicts. This may be a result of anxiously attached individuals' generally poor conflict management skills and their tendency to be more psychologically aggressive during conflict (Turner & Langhinrichsen-Rohling, 2011). Turner and Langhinrichsen-Rohling (2011) suggest that anxious individuals' aggressive tendencies might result from attempts to coerce their partner to meet their needs or keep their partner from withdrawing during conflicts. However, psychological aggression in an attempt to keep one's partner close and prevent abandonment can make relationship conflicts even worse. Not surprisingly, more negative conflicts are associated with greater stress, and this stress can often be seen at the biological level. Indeed, psychological aggression during conflict is linked with decreased immune cell function and larger increases in interleukin-6 (IL-6, a common measure of immune functioning) up to 18 hr after a conflict (Péloquin, Lafontaine, & Brassard, 2011). Increased IL-6 levels, in turn, are linked with a number of poor health outcomes, including cardiovascular disease, diabetes, and even some forms of cancer (Kiecolt-Glaser et al., 2005). Similar associations have been uncovered with other measures of immune functioning; for example, people with higher levels of attachment anxiety have fewer helper and killer T cells, which aid in the destruction of cells in the body that are infected with cancer or viruses or that have been damaged in some way.

Trans-epidermal water loss is an indicator of wound healing (skin barrier recovery) and provides yet another measurement of an individual's immune system functioning. In one study, couples experienced a relatively painless tape-stripping procedure before participating in a conflict discussion (Robles et al., 2013). The amount of trans-epidermal water loss was measured for 2 hr after skin disruption. Among women, higher levels of attachment anxiety predicted *faster* wound healing, but among men, higher anxiety predicted *slower* wound healing. Robles et al. (2013) posit that providing and receiving support may be more threatening for anxious men than for anxious women due to gender differences in social norms and socialization pressures, which could decrease the efficiency of anxious men's immune system functioning.

Avoidant attachment has been less consistently linked with immune function. In one study, avoidant individuals produced more IL-6 in response to relationship conflict and had a greater pro-inflammatory cytokine response (Gouin et al., 2009), suggesting a limited ability to fight off infection and inflammation. Although some cytokines are anti-inflammatory and are good for maintaining overall health, pro-inflammatory responses accelerate disease progression and are associated with poorer health outcomes (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). However, in another study, avoidant attachment was not associated with immune functioning (Kiecolt-Glaser et al., 2010).

It is important to note that measures of stress, cortisol reactivity, and immune function should be considered in the broader context of biomarkers that, if dysregulated, *could* have implications for poor health outcomes. As an example, it is not yet clear whether the association

between attachment insecurity and cardiovascular disease can be explained by a heightened stress response, unhealthy behaviors such as smoking, a lower likelihood of healthcare utilization, or a combination of all of these factors (and likely others). It does seem clear, however, that biological processes can help to explain links between attachment and health. It will be important for future research to assess both psychological and physiological processes to better understand the influence of adult attachment orientation on health outcomes. Such integrative research is an ambitious endeavor, but it is essential for theory development.

Future Directions

Situating Attachment Theory and Health Within a Dyadic Framework

To date, most studies of attachment and health have examined relations among preventative behaviors, stress-related emotions and behavior, and physiological and neuroendocrine responses to interpersonal stress at the individual level. Recently, however, researchers have attempted to situate the effects of attachment orientations on health within a relational, or *dyadic*, framework. A dyadic framework is critical because many of the proposed mechanisms linking close relationships with health (e.g., support, perceived partner responsiveness, relationship satisfaction) are hypothesized to occur between relationship partners (Jaremka et al., 2013). Generally, one romantic partner's positive psychological characteristics and health behaviors are thought to benefit his/her partner's physical well-being.

Pietromonaco, Uchino, and Dunkel Schetter (2013) argue that individual differences in attachment interact with relationship processes to influence physical health outcomes. According to their theoretical model, individual differences in attachment influence interpersonal behavior in stressful situations, such as major life transitions (e.g., pregnancy and parenthood) and acute relationship conflicts. For instance, in a stressful situation, avoidant adults may be less likely to seek out support from a romantic partner and more likely to try to suppress their emotions. As a result, avoidant adults may feel particularly stressed; their stress response system may become overly active, and/or they may engage in unhealthy behavior. Greater stress responses and deleterious health behaviors, in turn, can increase the likelihood for poor health outcomes and disease. Importantly, this model also accounts for the influence of a partner's dispositions and behavior on an individual's health. For example, an avoidant person may be less likely to provide support to a loved one who is particularly stressed; she/he may even undermine a partner's efforts to fix a problem. These kinds of interactions could increase feelings of stress, and the associated physiological stress responses, in turn increasing the likelihood of poor health outcomes and disease.

Although there is growing empirical support for Pietromonaco et al.'s (2013) model, important questions remain to be addressed by future research. For instance, the psychological and physiological variables assumed to mediate associations between attachment orientations and health outcomes are not often tested. Little is known about how both partners' attachment orientation might predict relationship processes (e.g., responsiveness, satisfaction, commitment) and health behavior that lead to poorer physical health outcomes over time. Parts of this model have been tested, but it has yet to be evaluated in full. Even less is known about pathways linking attachment orientations to dyadic interactions, and ultimately to health, among middle-aged and older adults. Most studies of attachment involve younger adults and couples; the challenges that middle-aged and older couples encounter are often unique to their stage in life and differ from that of a younger population. Perhaps the consequences of

these processes *accumulate* over time, only arising after people have been in relationships for many years (as would be the case for middle-age and older adults). Other age-graded responsibilities (e.g., children, work, retirement, caregiving) may also exert additional stress that is not well accounted for by the theoretical model.

Future research on attachment and health outcomes should implement dyadic and longitudinal research designs and focus on more objective measures of health. For instance, people with anxious attachment styles report more health symptoms, but it is unclear whether they are simply more likely to report poorer health, or if they objectively experience more health symptoms. Another fruitful avenue for future research is examining the developmental antecedents of the attachment–health link. In addition to focusing on self-reported health among insecure individuals, it is important to look at early childhood attachment experiences as they relate to the diagnoses of specific diseases in adulthood.

Examining Prospective Associations Between Attachment and Physical Health

Much of our discussion thus far has focused on links between individual differences in attachment and health in cross-sectional and isolated, short-lived settings (i.e., during a single interaction task or a laboratory stressor). However, many of the mechanisms that are hypothesized to link attachment to health, such as allostatic load (i.e., the long-term effects of chronic stress exposure), reflect phenomena that unfold over time. Indeed, attachment processes and experiences at one point in time may be related to physical health at a later point in time. For instance, memories of higher quality care in childhood are related to better self-rated health, fewer chronic illnesses, and lower depression over a 4-year period (for adults in their 60s) and over a 10-year period (for adults in their 40s; Chopik & Edelstein, 2015). Attachment insecurity in childhood also predicts the likelihood of reporting physical health symptoms 30 years later. Adults who were anxiously attached in childhood are seven times more likely to report inflammation-related diseases (i.e., coronary heart disease, asthma, diabetes, stroke, and hypertension) and three times more likely to report nonspecific symptoms (i.e., migraines, chest pains, fainting spells, indigestion, and backache) compared with adults who had a secure attachment style in childhood (Puig, Englund, Simpson, & Collins, 2013).

Although prospective data are crucial to understanding these linkages, longitudinal studies that examine attachment, health, and possible mediators between the two are relatively rare. Longitudinal data are particularly important because they can help parse out which factors are most strongly related to health and well-being and provide further evidence of the enduring effects of attachment on health. Yet even prospective studies are not without flaws. For example, multiple mechanisms are rarely tested within the same study to assess the relative contributions of each—an important consideration given the overlap among many explanatory variables. To illustrate, secure attachment is associated with healthier behavior and greater feelings of social support, two variables that may be (a) intercorrelated (e.g., people with greater social support may also exercise and/or diet more) and (b) predict health independently of each other. But which is more likely to explain the link between attachment and health—health behavior, social support, or both? Examining prospective links while examining multiple mechanisms is often the best approach to disentangling exactly what is driving a specific association.

Another limitation of these studies is the tendency to focus on the “book ends” of life—either late adolescence/early adulthood or older adulthood, so the processes associated with health in middle adulthood are less clear. Additionally, most studies are correlational, so relatively little is known about the causal direction of any associations. Future research could make greater use of longitudinal data to provide stronger tests of the various mechanisms

hypothesized to link attachment to physical health and to better understand the causal direction of attachment–health associations.

Conclusion

Close relationships have important implications for physical health and well-being, and higher quality social relationships are consistently related to better physical health. In this entry, we presented evidence that individual differences in how people approach relationships can have important health consequences. Overall, both attachment anxiety and avoidance are associated with a greater risk for the development of disease and chronic illness. Anxiously attached people generally report more physical health symptoms and seem particularly vulnerable to conditions that involve the cardiovascular system. Attachment avoidance has been less directly related to physical health, although there is limited evidence of a link between attachment avoidance and physical conditions involving pain. But *how* does one's attachment orientation or feelings about close relationships influence their physical health?

Researchers are just beginning to examine the processes that might help to explain why attachment is associated with health outcomes. In this entry, we discussed several factors that impact health and well-being: perceptions of social support, health behaviors, emotion regulation, and physiological and neuroendocrine response systems. Examining these mechanisms (and others) can provide greater insight into both relationship *and* health-related processes and how they interact in different contexts.

Going forward, research on attachment-related differences in health should begin to address several key limitations. First, future research should implement dyadic designs that include both couple members to elucidate how people's attachment styles influence their own and their partner's health. Second, future research should adopt longitudinal designs to examine how attachment–health associations unfold over time. Longitudinal studies are necessary to understand the directional nature of attachment–health findings and to capture the developmental course of health from early childhood to late adulthood. Addressing these limitations and investigating the multiple pathways linking attachment orientations to health can help people live not only longer but also happier lives as well.

Author Biographies

Onawa P. LaBelle is a PhD candidate in psychology at the University of Michigan. Ms. LaBelle is interested in close relationships processes, positive emotions, and physical and mental health outcomes. Her current work examines the benefits of gratitude for insecurely attached individuals in at-risk populations (e.g., recovery from alcoholism, sexual minorities).

Britney M. Wardecker is a PhD candidate in psychology at the University of Michigan. Britney's research interests broadly focus on close relationships, hormones, and stress and health. More specifically, she examines how people respond to stressful situations and how hormones are tied to people's experiences in close and intimate relationships, such as romantic and parent–child relationships.

William J. Chopik, PhD, is an assistant professor at Michigan State University. Dr. Chopik researches how relationships—and the people in them—change over time. He focuses on how factors both inside (biological, hormonal) and outside (social roles, geography) of people influence their approach to social relationships.

Robin S. Edelstein, PhD, is an associate professor at the University of Michigan. Her research focuses on individual differences in emotional experience, regulation, and reactivity. She is particularly interested in how emotional processes unfold in interpersonal contexts and the implications of emotional processes for close relationships, behavior, and physiology.

References

- Ahrens, K., Ciechanowski, P., & Katon, W. (2012). Associations between adult attachment style and health risk behaviors in an adult female primary care population. *Journal of Psychosomatic Research*, 72, 364–370. doi:10.1016/j.jpsychores.2012.02.002
- Bowlby, J. (1969). *Attachment and loss: Attachment* (Vol. 1). New York, NY: Basic Books.
- Chopik, W. J., & Edelstein, R. S. (2019). Maternal support in childhood is associated with changes in physical and mental health in middle-age and older adulthood. *Health Psychology*, 38, 84–93.
- Diamond, L. M., & Hicks, A. M. (2005). Attachment style, current relationship security, and negative emotions: The mediating role of physiological regulation. *Journal of Social and Personal Relationships*, 22, 499–518. doi:10.1177/0265407505054520
- Dolan, R. J., Lane, R., Chua, P., & Fletcher, P. (2000). Dissociable temporal lobe activations during emotional episodic memory retrieval. *NeuroImage*, 11, 203–209. doi:10.1006/nimg.2000.0538
- Edelstein, R. S., Kean, E. L., & Chopik, W. J. (2012). Women with an avoidant attachment style show attenuated estradiol responses to emotionally intimate stimuli. *Hormones and Behavior*, 61, 167–175.
- Edelstein, R. S., & Shaver, P. R. (2004). Avoidant attachment: Exploration of an oxymoron. In D. J. Mashek & A. P. Aron (Eds.), *Handbook of closeness and intimacy* (pp. 397–412). Mahwah, NJ: Lawrence Erlbaum Associates.
- Feeney, J., & Ryan, M. (1994). Attachment style and affect regulation: Relationships with health behavior and family experiences of illness in a student sample. *Health Psychology*, 13, 334–354. doi:10.1037/0278-6133.13.4.334
- Gallo, L., & Matthews, K. (2006). Adolescents' attachment orientation influences ambulatory blood pressure responses to everyday social interactions. *Psychosomatic Medicine*, 68, 253–261. doi:10.1097/01.psy.0000204633.33599.81
- Gentzler, A. L., & Kerns, K. A. (2004). Associations between insecure attachment and sexual experiences. *Personal Relationships*, 11, 249–265.
- Gouin, J. P., Glaser, R., Loving, T. J., Malarkey, W. B., Stowell, J., Houts, C., & Kiecolt-Glaser, J. K. (2009). Attachment avoidance predicts inflammatory responses to marital conflict. *Brain, Behavior, and Immunity*, 23, 898–904. doi:10.1016/j.bbi.2008.09.016
- Hazan, C., & Shaver, P. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology*, 52, 511–524. doi:10.1037/002-3514.52.3.511
- Illing, V., Tasca, G. A., Balfour, L., & Bissada, H. (2010). Attachment insecurity predicts eating disorder symptoms and treatment outcomes in a clinical sample of women. *The Journal of Nervous and Mental Disease*, 198, 653–659. doi:10.1097/NMD.0b013e3181ef34b2
- Jaremka, L. M., Glaser, R., Loving, T. J., Malarkey, W. B., Stowell, J. R., & Kiecolt-Glaser, J. K. (2013). Attachment anxiety is linked to alterations in cortisol production and cellular immunity. *Psychological Science*, 24, 272–279. doi:10.1177/0956797612452571
- Kafetsios, K., & Sideridis, G. D. (2006). Attachment, social support and well-being in young and older adults. *Journal of Health Psychology*, 11, 863–875.
- Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience & Biobehavioral Reviews*, 35, 33–38.
- Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., & Glaser, R. (2005). Hostile marital interactions, proinflammatory cytokine production, and wound healing. *Archives of General Psychiatry*, 62, 1377–1384. doi:10.1001/archpsyc.62.12.1377

- Maunder, R. G., & Hunter, J. J. (2001). Attachment and psychosomatic medicine: Developmental contributions to stress and disease. *Psychosomatic Medicine*, 63, 556–567.
- McWilliams, L. A., & Bailey, S. J. (2010). Associations between adult attachment ratings and health conditions: Evidence from the National Comorbidity Survey Replication. *Health Psychology*, 29, 446–453. doi:10.1037/a0020061
- Mikail, S. F., Henderson, P. R., & Tasca, G. A. (1994). An interpersonally based model of chronic pain: An application of attachment theory. *Clinical Psychology Review*, 14, 1–16. doi:10.1016/0272-7358(94)90045-0
- Mikulincer, M., & Shaver, P. R. (2008). Adult attachment and affect regulation. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 503–531). New York, NY: Guilford Press.
- Ognibene, T., & Collins, N. (1998). Adult attachment styles, perceived social support and coping strategies. *Journal of Social and Personal Relationships*, 15, 323–345. doi:10.1177/0265407598153002
- Péloquin, K., Lafontaine, M.-F., & Brassard, A. (2011). A dyadic approach to the study of romantic attachment, dyadic empathy, and psychological partner aggression. *Journal of Social and Personal Relationships*, 28, 915–942. doi:10.1177/0265407510397988
- Pietromonaco, P. R., Uchino, B., & Dunkel Schetter, C. (2013). Close relationship processes and health: Implications of attachment theory for health and disease. *Health Psychology*, 32, 499–513. doi:10.1037/a0029349
- Puig, J., Englund, M. M., Simpson, J. A., & Collins, W. A. (2013). Predicting adult physical illness from infant attachment: A prospective longitudinal study. *Health Psychology*, 32, 409–417. doi:10.1037/a0028889
- Quirin, M., Gillath, O., Pruessner, J. C., & Eggert, L. D. (2010). Adult attachment insecurity and hippocampal cell density. *Social Cognitive and Affective Neuroscience*, 5, 39–47. doi:10.1093/scan/nsp042
- Robles, T. F., Brooks, K. P., Kane, H. S., & Schetter, C. D. (2013). Attachment, skin deep? Relationships between adult attachment and skin barrier recovery. *International Journal of Psychophysiology*, 88, 241–252. doi:10.1016/j.ijpsycho.2012.04.007
- Silverberg, J. I., & Ratner, D. (2015). Associations of non-melanoma skin cancer and melanoma, extracutaneous cancers and smoking in adults: A US population-based study. *Journal of European Academy of Dermatology and Venerology*, 29, 1389–1397. doi:10.1111/jdv.12883
- Turner, L. A., & Langhinrichsen-Rohling, J. (2011). Attachment, relationship beliefs, and partner-specific assertiveness and psychological aggression among college students. *Partner Abuse*, 2, 387–403. doi:10.1891/1946-6560.2.4.387
- Zachrisson, H. D., & Skårderud, F. (2010). Feelings of insecurity: Review of attachment and eating disorders. *European Eating Disorders Review*, 18, 97–106. doi:10.1002/erv.999

Suggested Reading

- Feeney, J. A. (2000). Implications of attachment style for patterns of health and illness. *Child: Care, Health and Development*, 26, 277–288. doi:10.1046/j.1365-2214.2000.00146.x
- Loving, T. J., & Slatcher, R. B. (2013). Romantic relationships and health. In J. A. Simpson, L. Campbell, J. A. Simpson, & L. Campbell (Eds.), *The Oxford handbook of close relationships* (pp. 617–637). New York, NY: Oxford University Press.
- McWilliams, L. A., & Bailey, S. J. (2010). Associations between adult attachment ratings and health conditions: Evidence from the National Comorbidity Survey Replication. *Health Psychology*, 29, 446–453. doi:10.1037/a0020061
- Stanton, S. E., & Campbell, L. (2014). Psychological and physiological predictors of health in romantic relationships: An attachment perspective. *Journal of Personality*, 82, 528–538. doi:10.1111/jopy.12056

Bereavement

Mary-Frances O'Connor

Psychology Department, University of Arizona, Tucson, AZ, USA

Bereavement is the period after the death of a loved one. It is distinguished in bereavement research from grief and mourning. Grief is the emotional, cognitive, and physiological experiences that occur during bereavement and therefore has wide individual differences. Mourning is the term given to the outward behaviors in which people engage in during bereavement.

Why does grief happen? One of the primary theories used to explain the grief reaction is attachment theory (Bowlby, 1969). Attachment theory proposes that individuals seek proximity to each other after a period of bonding (in particular, bonding of an infant to a caregiver and pair-bonded monogamous adults). The proximity-seeking functions to keep the infant safe, and in later childhood and adulthood, serve as a safe haven to seek during distress and as a secure base from which to confidently explore the world. The absence of the attachment figure is therefore distressing, and concerted effort is made to reunite with the loved one. This effort at reunion is very useful when the separation is temporary, but in the case of death, reunion is not possible. Nonetheless, the desire to reunite with the deceased loved one, even though the bereaved person knows that it is not possible, may originate with the strong motivation to seek proximity that is present in attachment bonds established during infant bonding or pair bonding.

In adulthood, working models representing the attachment figure are often utilized in lieu of physical proximity. These working models, or schemas, allow for longer periods of separation, while still maintaining a sense of security. Schemas of the attachment figure often incorporate aspects of the self as well (e.g., a pair-bonded adult relationship has a schema not only for the other [husband] but also for the self [wife]). Therefore, intense grief after the death of a loved one usually occurs in cases where the deceased was an attachment figure and the death affects the bereaved person's own identity.

Early theories about the stages of grief (initially adapted from theories of terminal illness) suggested that bereaved individuals passed through denial, bargaining, anger, depression, and acceptance (Kubler-Ross, 1969). This older theory was influential because it shed light on grief as a possible phenomenon for empirical study. However, more recent empirical work has shown that bereaved individuals do not necessarily pass through these stages sequentially nor

does everyone experience each stage. For example, acceptance arises early in bereavement and continues to increase as the reality of the death is incorporated into the bereaved person's current working models of the world. A more recent theory suggests that bereaved individuals oscillate between focusing on the loss and focusing on the restoration of the new chapter of life after the death (Stroebe & Schut, 1999). The capacity to oscillate between these two distressing aspects of bereavement seems to predict adaptation, with more time spent focusing on restoration as adaptation continues. Adaptation in grief, or the integration of the fact of the death event and its consequences, can be seen when the bereaved is able to once again experience joy, derive meaning in life, and pursue important interests, such as work, caring for family members, or social activities.

Empirical work from a seminal prospective study of bereavement has shown that several trajectories of adaptation can be distinguished (Bonanno et al., 2002). The importance of this work is that it incorporated individual's functioning prior to the illness or death of the loved one. This enabled the authors to differentiate individuals based on characteristics observed prior to bereavement and following the death event at several time points. Patterns of adaptation include (a) individuals depressed prior to the loss who remain depressed following the loss (chronic depression), (b) individuals who become depressed only after the loss and remain depressed (chronic grief), and (c) individuals who become depressed only after the loss then improved (depressed improved). Notably, the majority of individuals fall into the category of (d) resilient. Despite the distress of acute grief, the resilient group does not show depression at any point during the study. It is worth noting that this research was done prior to the conceptualization of complicated grief symptomatology (discussed below). Consequently, depressive symptoms served as the primary variables of interest.

Factors that help and hinder adjustment following the loss of a loved one have been identified through empirical research, although there is not always agreement between studies on all factors. Social support is clearly a great resource during bereavement and aids in adjustment. Insecure attachment style, and dependency on the deceased, also predicts poorer adjustment than secure attachment, and so does the kinship relationship of the deceased. The loss of a child predicts the poorest adjustment, followed by the death of a spouse, compared with other relationship categories. Other factors (religiosity, sex, age, unexpectedness of the death) vary by research study as to whether they assist in adjustment, and this may be because they interact in complex ways with other factors. Secondary losses, such as changes in financial status and housing, often follow the death of a loved one. Coping with these types of additional stressors hinders adjustment to the loss.

Complicated grief is a term given to the disorder that arises when acute grief does not become integrated into the working models of the bereaved person. There are a number of different names that have been used for this disorder, reflecting the development and refinement of measuring of this phenomenon by the clinical science community. These names include traumatic grief, prolonged grief, and pathological grief and are associated with slightly different symptom sets. The DSM-5 has included this disorder in the section on disorders that require further research, and it is termed persistent complex bereavement disorder (PCBD).

PCBD includes criteria based on an individual having experienced the death of a loved one followed by one or more of the following symptoms on more days than not for at least 12 months following the loss (or 6 months for children): persistent yearning/longing for the deceased, intense sorrow, preoccupation with the deceased, and/or the circumstances of death (American Psychiatric Association, 2013). They must also endorse six or more symptoms that indicate clinically significant reactive distress to death (e.g., marked difficulty accepting the death and excessive avoidance of reminders of the loss) and social or identity disruption

(e.g., a desire to die in order to be with the deceased and feeling alone or detached from other individuals since the death). DSM-5 requires that the grief reaction must be beyond cultural, religious, and/or age-appropriate norms to avoid pathologizing a normal response to a common, yet very distressing, experience.

Less than 10% of the general bereaved population develop complicated grief, although that number increases significantly to approximately one fifth of those who lose a child or a spouse (Kersting, Brähler, Glaesmer, & Wagner, 2011). This highlights the resilient nature of most individuals who have this nearly universal experience of bereavement. Recent research has developed psychotherapeutic treatments specifically to address complicated grief, with efficacy demonstrated through several randomized controlled trials. These include complicated grief therapy, which combines a variety of psychotherapeutic approaches, including principles of cognitive behavior therapy (especially exposure to the aspects of the death that are avoided), interpersonal therapy, and motivational interviewing (especially focusing on creating a future that is meaningful and positive). Other trials have utilized cognitive behavioral therapy in individual, group, and Internet settings. Across several studies, exposure appears to be a critical ingredient, over and above the effect of cognitive restructuring. Thus far, mixed results have been reported for the pharmacological treatment of complicated grief (e.g., antidepressants), but benzodiazepines are not an effective treatment strategy.

Although grief is a universally experienced emotion, the expression of grief varies widely across history, culture, religion, and geography. Recent concerns have emerged that the “medicalization” of grief has developed along with a mismatch between the intense experience and slow natural adaptation following the loss and contemporary Western culture. Current Western culture values productivity, happiness, and progress. The grieving person only sporadically exhibits these qualities during bereavement, and there is often a denial and censorship by society regarding grief and loss. On the other hand, research shows that for those who experience complicated grief, the recognition that their experience is not normative and can be treated comes as a relief.

Physiological changes accompany bereavement, most likely due to the intensely stressful nature of the experience, and the intense emotions that it often engenders. The physiological systems that have been affected in empirical studies include the sympathetic and parasympathetic autonomic nervous system and the endocrine and immune systems. These changes include increases in heart rate, cortisol, and circulating levels of inflammation and decreases in heart rate variability and specific immune cell types. These changes are most pronounced in the first 6 months after the death of a loved one and then typically return to previous levels (or put differently to the level of matched nonbereaved individuals). The physical symptoms most often noted by bereaved individuals include difficulty sleeping, reduced appetite, and pangs of grief often accompanied by crying.

Presumably because of these changes in the stress response systems of the body, the “widowhood effect” also occurs during bereavement. This is the term for the phenomenon whereby the death of a loved one increases the morbidity and mortality of the survivor. The widowhood effect has been empirically demonstrated since the 1960s, and improved statistical modeling that accounts for a host of covariates has continued to document it. Broadly speaking, the increased risk for men after the death of a spouse is approximately twice the risk of their married counterparts, and the increased risk is approximately 1.5 times for women. The term “broken-heart phenomenon” has also been used, but the widowhood effect is preferred because the effect is on all-cause mortality, and not just cardiovascular health. In addition, the term “broken-heart phenomenon” has also become synonymous with takotsubo cardiomyopathy, which often, but not exclusively, occurs in the wake of a loved one’s death.

Future research on bereavement should continue to focus on dismantling clinical interventions that treat complicated grief, in order to determine the effective components of treatment. Additional interventions that would reduce morbidity and mortality during the period of greatest risk, such as cardioprotective medications, should be studied. Finally, the similarities and differences in grief between different types of loss experiences (death, divorce, job loss, loss of health, etc.) may clarify the key dimensions of this universal experience.

Author Biographies

Dr. Mary-Frances O'Connor, PhD, is an assistant professor at the Department of Psychology at the University of Arizona, following a PhD in clinical psychology and a postdoctoral fellowship at the Cousins Center for Psychoneuroimmunology at UCLA. Her research focuses on the physiological correlates of emotional responses to bereavement, including neurobiological, immune, and autonomic parameters that vary between individual grief responses. She has investigated the failure to adapt following the death of a loved one, termed complicated grief.

References

- American Psychiatric Association. (2013). DSM 5. *American Journal of Psychiatry*. <http://doi.org/10.1176/appi.books.9780890425596.744053>
- Bonanno, G. A., Wortman, C. B., Lehman, D. R., Tweed, R. G., Haring, M., Sonnega, J., ... Nesse, R. M. (2002). Resilience to loss and chronic grief: A prospective study from preloss to 18-months postloss. *Journal of Personality and Social Psychology*, 83, 1150–1164. <http://doi.org/10.1037/0022-3514.83.5.1150>
- Bowlby, J. (1969). *Attachment and loss*. New York, NY: Basic Books.
- Kersting, A., Brähler, E., Glaesmer, H., & Wagner, B. (2011). Prevalence of complicated grief in a representative population-based sample. *Journal of Affective Disorders*, 131(1–3), 339–343. <http://doi.org/10.1016/j.jad.2010.11.032>
- Kubler-Ross, E. (1969). *On death and dying*. New York, NY: Macmillan Company.
- Stroebe, M., & Schut, H. (1999). The dual process model of coping bereavement: Rationale and description. *Death Studies*, 23(3), 197–224. <http://doi.org/10.1080/074811899201046>

Suggested Reading

- Bonanno, G. A. (2009). *The other side of sadness*. New York, NY: Basic Books.
- Bowlby, J. (1969). *Attachment and loss*. New York, NY: Basic Books.
- Bowlby, J. (1980). *Loss: Sadness and depression*. New York, NY: Basic Books.
- William Worden, J. (1982). *Grief counseling and grief therapy: A handbook for the mental health practitioner*. New York, NY: Springer.

Conflicting Health Information

Delesha M. Carpenter¹ and Paul K. J. Han²

¹*Eshelman School of Pharmacy, University of North Carolina at Chapel Hill,
Chapel Hill, NC, USA*

²*Center for Outcomes Research and Evaluation, Maine Medical Center, Portland, ME, USA*

The explosive growth of the Internet, social media, and traditional news outlets has provided people with unprecedented access to health information (Fox, 2011). This expanded access has had both positive and negative effects. It has enabled laypersons to become more empowered consumers of healthcare. At the same time, however, it has altered the health information seeking behaviors of individuals and members of their social network (Fox, Duggan, & Purcell, 2013) and exposed the public to an ever-increasing supply of low-quality and often contradictory health information (Eysenbach, Powell, Kuss, & Sa, 2002). For example, a national survey found that 71.5% of the general public endorsed the view that “there are so many recommendations about preventing cancer, it’s hard to know which ones to follow” (Niederdeppe & Levy, 2007).

Conflicting health information burdens both laypersons and experts alike with the task of evaluating what information is credible. It also has other potentially deleterious effects. As researchers, healthcare providers, and government organizations have affirmed, conflicting health information can cause confusion about what health-related behaviors are appropriate to engage in and therefore expose individuals to harms resulting from indecision or misguided decisions (Wansink, 2006). Conflicting health information can also lead people to distrust health professionals, discount valid health information, and forego potentially beneficial medical interventions (Kienhues, Stadler, & Bromme, 2011; Theroux & Taylor, 2003). Furthermore, it can negatively impact medication adherence (Carpenter et al., 2010; Carpenter, Elstad, Blalock, & DeVellis, 2014) and individuals’ decisions to engage in even healthy behaviors for which there is clear consensus—for example, consuming fruits and vegetables (Nagler & Hornik, 2012).

Yet despite emerging evidence and consensus on the growth and negative effects of conflicting health information, surprisingly little is known about the magnitude and mechanisms of these effects. The construct of “conflicting information” has neither been defined conceptually nor measured systematically. The dearth of rigorous studies on the prevalence and effects

of conflicting health information is attributable to several factors, including the lack of a standardized definition, the absence of validated and reliable measures of the phenomenon, and a lack of theoretical models to explain its causes and effects.

In this entry we address these current deficiencies by summarizing insights from the available literature, with the aim of (a) proposing a working definition and conceptual typology of conflicting health information; (b) distinguishing conflicting health information from related terms, such as misinformation, ambiguous information, and confusing information; and (c) briefly discussing potential theoretical models that could serve as a starting point for efforts to develop theoretical frameworks to guide future research and to enhance our understanding of the antecedents, processing, and effects of conflicting health information.

Working Definition

Previous empirical research on conflicting health information offers few insights about how this phenomenon should be conceptualized or operationalized. Some studies have identified a primary source of information, such as a prescribing physician, and then examined whether other sources present information that conflicts with this primary source (Carpenter et al., 2014). Other studies have identified contradictory health messages and asked respondents to indicate how frequently they were exposed to those messages (Nagler & Hornik, 2012). In all of these studies, however, the meaning of conflicting health information has been implicitly assumed rather than explicitly defined. A coherent working definition is thus a primary need for further research on conflicting health information.

We propose defining conflicting health information in terms of health-related propositions. By “propositions” we mean statements or assertions about a health-related issue. Propositions may originate from either a single source or multiple information sources and may either be actively sought by an individual (e.g., through an Internet search) or passively encountered (e.g., through an overheard conversation).

“Conflicting health information” can then be operationally defined as two or more health-related propositions that are logically inconsistent with one another. The Oxford Dictionary (n.d.) defines the term “conflicting” as “at variance,” “incompatible,” or “contradictory.” We take the defining feature of “conflicting” to be that the propositions are discrepant such that a person could not simultaneously act upon or believe both propositions at once. For example, if two propositions differed on one point, such as the recommended age to initiate mammography screening (age 40 or 50), a woman could not initiate screening at both age 40 and 50. Similarly, if a person found a proposition online that “vaccines cause autism” and the person’s physician told him/her that “vaccines do not cause autism,” the person could not simultaneously believe both propositions.

Conceptual Typology

Proceeding from this operational definition, conflicting health information can be further classified according to four fundamental dimensions: the substantive issue of conflict, the number of conflicting sources, the degree of evidence heterogeneity, and temporal inconsistency. The first dimension of conflicting health information, the *issue of conflict*, is the specific health topic for which conflicting propositions exist. Issue is an important dimension because people’s reactions to conflicting information may vary based on the topic. For example, people may

have more negative reactions when conflicting information applies to cancer prognosis as opposed to cancer screening. The second dimension is *multiplicity* or the number of different sources of conflicting information. Conflicting information may be present in a single source, such as a cancer screening guideline that includes both of these propositions (Moyer, 2012):

The U.S. Preventive Services Task Force recommends against prostate-specific antigen (PSA)-based screening for prostate cancer. The potential benefit does not outweigh the expected harms.

If your patient does raise the issue of PSA screening, or if you believe his individual circumstances warrant consideration of PSA screening, be sure to discuss it with him thoroughly, so he can make an informed decision. The USPSTF strongly recommends that you do not order a PSA test without such a discussion. The decision to start or continue PSA screening should reflect your patient's understanding of the possible benefits and expected harms and should respect his preferences.

On the other hand, conflicting propositions may also exist across multiple sources, such as a PSA screening guideline released by a national task force versus a differing guideline published on a prestigious cancer center's website. Multiplicity is potentially important because people may react differently if they encounter conflicting information from a single source versus multiple sources, and people's negative reactions may increase or plateau as the number of conflicting sources increases.

The third dimension, *evidence heterogeneity*, is the extent to which conflicting information originates from similar versus differing evidentiary types. This attribute is important because individuals may process conflicting information differently when it comes from homogenous sources, such as various health professionals, as opposed to heterogeneous sources, such as health professionals versus family members. The fourth dimension, *temporal inconsistency*, is the extent to which conflicting propositions about an issue exist at the same point as opposed to different points in time. Conflicting information that is temporally inconsistent (i.e., asynchronous or separated over time) may produce different behavioral effects than conflicting information that is temporally consistent (i.e., synchronous or occurring at the same point in time). Temporally inconsistent (asynchronous) conflicting information, furthermore, may have varying effects depending on its frequency—that is, the extent to which a person is exposed to the conflicting information. For example, if conflicting cancer screening guidelines emerge every 5 years, this may have less of a negative effect on a person's screening behavior than if conflicting screening guidelines emerge every year.

This proposed definition and conceptual typology of conflicting information has potential utility as a framework to guide future research on this phenomenon. The magnitude of the effects of conflicting information may depend on its various properties (issue of conflict, multiplicity, evidence heterogeneity, temporal inconsistency). The mechanisms by which conflicting health information affects health judgments, decisions, and behaviors may also depend on these properties. All of these possibilities remain to be explored in future research studies, and the typology can help organize these efforts. Furthermore, although our proposed conceptual typology focuses on properties of conflicting information itself, the factor that exerts the greatest influence on people's responses may not be the objective existence of conflict in information but people's perceptions of such conflict.

Related Phenomena

Our working definition can be used to help differentiate conflicting information from related phenomena, including misinformation, ambiguous information, and confusing information.

Misinformation is defined as “false or inaccurate information, especially that which is deliberately intended to deceive” (Oxford Dictionary, n.d.). The American Dietetic Association (ADA) released a position statement that defined food and nutrition misinformation as “erroneous, incomplete, or misleading science without any scientific basis at all” (Wansink, 2006). The ADA further stated that conflicting food and nutrition information “can be disseminated naively or with malicious or self-serving intent” (Wansink, 2006). Thus, misinformation is conceptually distinct from conflicting information because it (a) focuses on the accuracy of a proposition and (b) makes attributions as to the motive for information dissemination. Our proposed definition of conflicting information examines the degree of conflict rather than the accuracy of two propositions and makes no attributions about how or why the information was disseminated.

The term *ambiguity* signifies another related but distinct phenomenon. Ambiguity itself has multiple meanings; the conventional dictionary definition is “open to more than one interpretation” or “having a double meaning” (Oxford Dictionary, n.d.). In behavioral decision theory, however, the term “ambiguity” refers to a specific feature of risk information pertaining to a lack of reliability, credibility, or adequacy and giving rise to epistemic uncertainty (Ellsberg, 1961). In this conception, ambiguity can arise from conflicting information and other limitations including missing or insufficient data or other limitations in data reliability. Conflicting information is conceptually distinct from ambiguous information because a single proposition may be ambiguous, but conflicting information cannot exist for a single proposition; conflicting information only exists when there are multiple propositions. However, it is likely that individuals are more likely to interpret ambiguous health propositions as conflicting than unambiguous propositions.

The term *confuse* is defined as making “something more complex or less easy to understand” (Oxford Dictionary, n.d.). Thus, confusing information could be defined as information that is difficult to understand. Similar to ambiguous information, a single proposition may be considered confusing; by definition, however, two propositions are needed for conflicting information to exist. Like ambiguous information, it is also likely that propositions that are confusing are more likely to be interpreted as conflicting. Thus, although one may assign the attributes of being confusing or ambiguous to health propositions, those propositions would not be considered conflicting unless they involved two or more health propositions that directly contradicted one another.

Theoretical Models

Currently, we lack theoretical models and frameworks to explain the antecedents and mechanisms of effect for conflicting information in the health domain. Theories from various disciplines can shed light on these problems, but thus far have not been applied to understanding the phenomenon of conflicting *health* information. We now briefly review theoretical perspectives that may serve as useful starting points for this effort.

Behavioral decision theorists since Ellsberg (1961) have conceptualized conflicting information as one source of ambiguity in risk information. Ambiguity, in turn, leads to a set of responses that include pessimistic appraisals of the benefits and risks of alternative choice options and avoidance of decision making—a phenomenon known as “ambiguity aversion.” This phenomenon has been demonstrated in multiple decision-making domains, both in and outside of healthcare. For example, higher levels of perceived ambiguity have been strongly associated with lower beliefs that cancer is preventable (Han et al., 2007; Han, Moser, &

Klein, 2006) and lower intentions toward future uptake of screening mammography (Han et al., 2007). Conflicting information as a specific source of ambiguity, furthermore, seems to evoke greater avoidance of decision making than ambiguity arising from information that is merely missing or unreliable; for this reason, some researchers have distinguished “conflict aversion” from ambiguity aversion (Smithson, 1999). This finding shows how ambiguity theory, as conceptualized in behavioral decision research, provides a useful framework for understanding the phenomenon of conflicting health information. Our conceptual typology may complement this theoretical framework by decomposing conflict into discrete constituent dimensions (i.e., issue of conflict, multiplicity, evidence heterogeneity, and temporal inconsistency). It is possible that these different dimensions exert varying influence on ambiguity aversion as classically characterized.

Theories of decision making, such as fuzzy-trace theory (Reyna & Brainerd, 1995), also have great potential relevance and value for the study of conflicting health information. Fuzzy-trace theory posits that people form two types of cognitive representations of information: a verbatim representation and a gist representation. The verbatim representation reflects the precise, often quantitative, aspect of the information received (e.g., 10% of patients who take medication X experience side effect Y), whereas the gist representation captures the essential “bottom-line” meaning of the information, as understood by the receiver, in qualitative terms (e.g., medication X can cause side effect Y). Fuzzy-trace theory is salient for the study of conflicting information because it suggests that gist representations of health information may lead patients to perceive information as conflicting even when, in fact, it is not. Moreover, it suggests that other factors, such as an individual’s level of health literacy, could interfere with the processing of health information and could lead to inaccurate gist representations. Additional work is needed to identify the person-level factors that may influence how individuals process and react to conflicting information. For example, perceptions of the salience of conflicting information and trust of information sources may influence how individuals process and react to conflicting information.

Another potentially useful theoretical perspective comes from artificial intelligence research that has produced mathematical formulas for how conflicting information is incorporated into existing knowledge systems (Benferhat, Kaci, Le Berre, & Williams, 2004; Cantwell, 1998; Lin, 1996). These formulas can be weighted by the trustworthiness of sources. The main construct of interest in this area is belief revision that focuses on how new information is incorporated into a knowledge base and the way in which conflicting information from different sources is identified and integrated into a given knowledge base. However, these theories focus on knowledge integration in computer systems and treat actors as rational. Yet human beings often do not act rationally with regard to their health, and their belief revision processes in the face of conflicting information will likely operate differently than what has been described in the artificial intelligence domain. Computational theories of conflicting information, despite their well-developed nature and relevance, would require significant adaptation in order to be applied to a health context.

These and other theories have clear relevance to understanding the phenomenon of conflicting health information, but they accommodate this phenomenon only indirectly. The critical need moving forward is for an integrative theoretical model that provides a more direct, coherent account of the causes and effects of conflicting information. Truly advancing our understanding of conflicting information requires synthesizing diverse insights about conflicting information—from multiple fields of inquiry—into a unified theory or model. Although existing theories provide useful lenses for understanding the causes and effects of conflicting information, exactly how to integrate these theories is unclear and represents a fundamental

research need. In addition to theory development, valid and reliable measures of conflicting health information are needed. Measure development in this area should focus on assessing each of the four dimensions of conflicting information described previously. Additional measurement work should focus on assessing subjective perceptions of various aspects of conflicting health information (e.g., trustworthiness of conflicting information sources) that may influence individuals' decisions and health behaviors.

Research on the nature, extent, causes, and effects of conflicting health information can ultimately serve as the foundation for the development of interventions to help individuals, including patients and clinicians, better manage conflicting health information when making decisions about their health. The working definition and conceptual typology of conflicting health information presented in this entry are offered as a first step toward a comprehensive program of research on this increasingly important problem.

Author Biographies

Delesha M. Carpenter is an assistant professor in the Division of Pharmaceutical Outcomes and Policy at the University of North Carolina Eshelman School of Pharmacy. She is a behavioral researcher with expertise in understanding how patient–provider communication affects behaviors and outcomes for individuals living with chronic disease. Her research explores how receiving conflicting information from various sources affects patient medication adherence. She is currently exploring ways to accurately measure conflicting medication information.

Paul K. J. Han is director of the Center for Outcomes Research and Evaluation at Maine Medical Center. He is a behavioral and health services researcher and a general internist and palliative medicine physician. His main research interests are in risk communication and shared decision making, and his work focuses on the communication and management of uncertainty in healthcare.

References

- Benferhat, S., Kaci, S., Le Berre, D., & Williams, M. A. (2004). Weakening conflicting information for iterated revision and knowledge integration. *Artificial Intelligence*, 153(1–2), 339–371. doi:10.1016/j.artint.2003.08.003
- Cantwell, J. (1998). Resolving conflicting information. *Journal of Logic, Language and Information*, 7(2), 191–220. doi:10.1023/A:1008296216319
- Carpenter, D. M., DeVellis, R. F., Hogan, S. L., Fisher, E. B., DeVellis, B. M., & Jordan, J. M. (2010). The effect of conflicting medication information and physician support on medication adherence for chronically ill patients. *Patient Education and Counseling*, 81(2), 7. doi:10.1016/j.pec.2009.11.006
- Carpenter, D. M., Elstad, E. A., Blalock, S. J., & DeVellis, R. F. (2014). Conflicting medication information: Prevalence, sources, and relationship to medication adherence. *Journal of Health Communication*, 19(1), 67–81. doi:10.1080/10810730.2013.798380
- Ellsberg, D. (1961). Risk, ambiguity, and the Savage axioms. *The Quarterly Journal of Economics*, 643–669. doi:10.2307/1884324
- Eysenbach, G., Powell, J., Kuss, O., & Sa, E.-R. (2002). Empirical studies assessing the quality of health information for consumers on the World Wide Web. *Journal of the American Medical Association*, 287(20), 2691–2700. doi:10.1001/jama.287.20.2691
- Fox, S. (2011). *The social life of health information 2011*. Washington, DC: Pew Internet & American Life Project.

- Fox, S., Duggan, M., & Purcell, K. (2013). *Family caregivers are wired for health*. Washington, DC: Pew Research Center's Internet & American Life Project.
- Han, P. K., Kobrin, S. C., Klein, W. M., Davis, W. W., Stefanek, M., & Taplin, S. H. (2007). Perceived ambiguity about screening mammography recommendations: Association with future mammography uptake and perceptions. *Cancer Epidemiology, Biomarkers & Prevention*, 16(3), 458–466. doi:10.1158/1055-9965.EPI-06-0533
- Han, P. K. J., Moser, R. P., & Klein, W. M. P. (2006). Perceived ambiguity about cancer prevention recommendations: Relationship to perceptions of cancer preventability, risk, and worry. *Journal of Health Communication*, 11(1), 51–69. doi:10.1080/10810730600637541
- Kienhues, D., Stadtler, M., & Bromme, R. (2011). Dealing with conflicting or consistent medical information on the web: When expert information breeds laypersons' doubts about experts. *Learning and Instruction*, 21(2), 193–204. doi:10.1016/j.learninstruc.2010.02.004
- Lin, J. (1996). Integration of weighted knowledge bases. *Artificial Intelligence*, 83(2), 363–378. doi:10.1016/0004-3702(95)00019-4
- Moyer, V. A. (2012). Screening for prostate cancer: US Preventive Services Task Force recommendation statement. *Annals of Internal Medicine*, 157(2), 120–134. doi:10.7326/0003-4819-157-2-201207170-00459
- Nagler, R. H., & Hornik, R. C. (2012). Measuring media exposure to contradictory health information: A comparative analysis of four potential measures. *Communication Methods and Measures*, 6(1), 56–75. doi:10.1080/19312458.2011.651348
- Niederdeppe, J., & Levy, A. G. (2007). Fatalistic beliefs about cancer prevention and three prevention behaviors. *Cancer Epidemiology, Biomarkers & Prevention*, 16(5), 998–1003. doi:10.1158/1055-9965.EPI-06-0608
- Oxford Dictionary. (n.d.). Retrieved from http://www.oxforddictionaries.com/us/definition/american_english
- Reyna, V. F., & Brainerd, C. J. (1995). Fuzzy-trace theory: An interim synthesis. *Learning and Individual Differences*, 7(1), 1–75. doi:10.1016/1041-6080(95)90031-4
- Smithson, M. (1999). Conflict aversion: Preference for ambiguity vs conflict in sources and evidence. *Organizational Behavior and Human Decision Processes*, 79(3), 179–198. doi:10.1006/obhd.1999.2844
- Theroux, R., & Taylor, K. (2003). Women's decision making about the use of hormonal and nonhormonal remedies for the menopausal transition. *Journal of Obstetric, Gynecologic & Neonatal Nursing*, 32(6), 712–723. doi:10.1177/0884217503257339
- Wansink, B. (2006). Position of the American Dietetic Association: Food and nutrition misinformation. *Journal of the American Dietetic Association*, 106(4), 601–607. doi:10.1016/j.jada.2006.02.019

Suggested Reading

- Carpenter, D. M., DeVellis, R. F., Hogan, S. L., Fisher, E. B., DeVellis, B. M., & Jordan, J. M. (2010). The effect of conflicting medication information and physician support on medication adherence for chronically ill patients. *Patient Education and Counseling*, 81(2), 7. doi:10.1016/j.pec.2009.11.006
- Carpenter, D. M., Elstad, E. A., Blalock, S. J., & DeVellis, R. F. (2014). Conflicting medication information: Prevalence, sources, and relationship to medication adherence. *Journal of Health Communication*, 19(1), 67–81. doi:10.1080/10810730.2013.798380
- Han, P. K. J., Moser, R. P., & Klein, W. M. P. (2006). Perceived ambiguity about cancer prevention recommendations: Relationship to perceptions of cancer preventability, risk, and worry. *Journal of Health Communication*, 11(1), 51–69. doi:10.1080/10810730600637541

Coping Strategies

Ellen Stephenson and Anita DeLongis

Department of Psychology, University of British Columbia, Vancouver, BC, Canada

Much of current theory and research on coping stems from a transactional model of stress and coping (Folkman, Lazarus, Gruen, & DeLongis, 1986), in which coping is defined as thoughts and behaviors used to manage the internal and external demands of a stressful situation. Earlier theories took a more hierarchical approach and conceptualized coping as referring exclusively to adaptive responses that were contrasted with maladaptive responses (e.g., defending, fragmenting; Haan, 1969; Menninger, 1963; Vaillant, 1977). Transactional models expanded the conceptualization of coping to include a broader set of cognitive and behavioral responses to stress. How a given situation is appraised determines whether the individual experiences it as stressful. Situations that are appraised as threatening or taxing available coping resources are experienced as stressful and necessitate a coping response. These coping responses are not viewed as inherently adaptive or maladaptive. Instead, the answer to whether a given coping strategy is effective is expected to vary by person and situation (DeLongis & Holtzman, 2005).

Coping Strategies

Several ways of categorizing coping strategies have been proposed (see Skinner, Edge, Altman, & Sherwood, 2003). Coping responses are frequently categorized in terms of their focus or function (O'Brien & DeLongis, 1996). Problem-focused coping describes direct efforts to solve the problem at hand, whereas emotion-focused coping involves attempts to lessen the emotional distress associated with a stressful situation (Lazarus & Folkman, 1984). Relationship-focused coping describes a third coping function and includes efforts to manage and maintain important social relationships during periods of stress (e.g., O'Brien, DeLongis, Pomaki, Puterman, & Zwicker, 2009). Another key method for describing coping strategies is in terms of approach or avoidance (Roth & Cohen, 1986). Within this framework, coping strategies are differentiated based on whether cognitive and emotional activity is oriented either toward or away from threat. Stanton and her colleagues have argued that approach-

oriented emotion-focused coping must be distinguished from avoidance-oriented emotion-focused coping. Emotional approach coping involves actively processing and expressing emotions rather avoiding them (Austenfeld & Stanton, 2004). Similar to the approach/avoidance framework, coping can also be described in terms of strategies involving engagement or disengagement. Engagement responses are oriented toward either the source of stress or toward one's emotions and thoughts, whereas disengagement describes responses that are oriented away from the stressor or one's thoughts or emotions (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). These examples are just some of the ways coping strategies can be categorized. Within these broader categories, many more specific coping strategies have also been described (e.g., planning, denial, support seeking, empathic responding, positive reappraisal, emotional expression).

Coping as a Process

Coping is a dynamic process that can involve the use of various coping strategies at different phases of a stressor. For laboratory stressors that are typically acute in nature due to the constraints of the experimental setting, the coping process can be relatively brief. However, most life stressors are either chronic or stress-event sequences. Given this, coping most often involves a cascade of cognitive and behavioral responses to a sequence of stressful events. For example, coping with cancer might start with the response to a perception of symptoms, followed by coping with the diagnosis and then treatment, which are related but separate stressors. There are many additional stressors typically experienced by cancer patients, including changes in family relationships, and these may all involve very different coping responses.

Coping strategies can operate synergistically, with one form of coping facilitating or hindering the use or effectiveness of other strategies. For example, emotion-focused coping may be necessary to manage emotions before an individual can effectively engage in problem-focused coping. A patient receiving a negative diagnosis may need to first engage in emotion-focused coping to reduce the perceived threat. This strategy may in turn facilitate understanding important treatment information and effectively allow engagement in problem-focused coping efforts. Another example involves relationship-focused coping facilitating problem-focused coping. Caregiving spouses of patients with Alzheimer's disease reported that by engaging in perspective taking and empathy for their spouse, they were able to generate better solutions to problems posed by the disease (DeLongis & O'Brien, 1990).

Coping was traditionally conceptualized as a response to stressful events that occurred in the past or that were occurring in the present. However, another way of reducing stress and regulating emotion is to avoid stressful situations or prevent them from occurring (Gross & Thompson, 2007). Individuals can also use proactive coping strategies to build up resources to help them cope with stressors that they might encounter in the future (Aspinwall & Taylor, 1997). Future-oriented coping strategies are sometimes further categorized depending on the certainty and temporal proximity of future events. For example, Schwarzer and Knoll (2003) distinguish between *anticipatory coping* (efforts to deal with a critical event that is certain or fairly certain to occur in the near future), *preventive coping* (efforts to deal with an uncertain threat potential in the distant future), and *proactive coping* (efforts to prepare for upcoming challenges that are potentially self-promoting). Incorporating both the antecedents and consequences of stressful events provides a fuller perspective on the coping process.

Choosing an Effective Coping Strategy

Although it is tempting to look for the “perfect” coping strategy, no one strategy is ideally suited to all contexts. Whether a particular coping strategy is “effective” depends on how we define successful coping. For example, problem-focused coping is likely to be effective in solving the problem or eliminating the stressful situation. There are some situations, such as the death of a loved one, where the source of the stress cannot be eliminated. In these situations, where successful coping may be defined in terms of reducing psychological distress, emotion-focused strategies are needed. If maintaining relationships with close others is an important outcome defining successful coping, then relationship-focused coping efforts may be necessary. Indeed, most stressful situations likely need all three forms of coping for successful resolution of the stressor. However, sometimes a stressor presents competing demands that conflict or favor different coping responses. For example, coping strategies that maximize individual well-being may come at a cost to the people involved.

The time frame used to evaluate the effects of a particular coping strategy can influence whether it is adaptive or maladaptive. That is, the same strategy can have different short-term and long-term effects. Avoidance can be useful in reducing distress in the short term, but if the stressor persists, this strategy can hinder long-term adjustment. For example, although processing emotions can be adaptive in the shorter term, continuing to engage in emotion processing over the longer term may become ruminative and therefore less beneficial in terms of adjustment (Austenfeld & Stanton, 2004). Other strategies may increase distress in the short term but promote adaptation in the long term. For example, husbands’ use of empathic responding is associated with increased marital tension on the same day as family stress occurs, but reduced tension the following day (O’Brien et al., 2009). This suggests that we need to consider both short- and long-term outcomes in order to fully understand the coping process. What is effective in the short term may be ineffective in the long term, and vice versa.

Which coping strategy is used and whether it is effective is determined by a combination of situational and personality factors. Some situations tend to be appraised in a particular way (e.g., threat vs. challenge appraisal; threat to agency vs. threat to communion), and this appraisal is associated with which coping strategies are used (Franks & Roesch, 2006; Pow, Lee-Baggley, & DeLongis, 2016). However, successful adaption often involves tailoring the coping response to the demands of the situation (Lee-Baggley, Preece, & DeLongis, 2005) and knowing when to persist with a given strategy or shift to other coping strategies (Chen & Miller, 2012). For example, in situations where perceived control is high, problem-focused coping tends to be the preferred strategy—but if actual control is low, other coping strategies are more effective (Zakowski, Hall, Klein, & Baum, 2001). Some people show greater flexibility in their ability to match their coping response to the situational context, and others tend to employ certain coping strategies or are more skilled at using specific coping strategies (Cheng, Lau, & Chan, 2014; Lee-Baggley et al., 2005).

Coping Within a Social Context

There is growing recognition that coping is a social process (e.g., Revenson, Kayser, & Bodenmann, 2005). Although interpersonal conflicts are one of the most common sources of daily stress (Bolger, Stadler, Paprocki, & DeLongis, 2010), social relationships also play an important role throughout the coping process (DeLongis & Holtzman, 2005). Findings from several studies

have highlighted the role of social support in times of stress (e.g., DeLongis, Holtzman, Puterman, & Lam, 2010; Uchino, Bowen, Carlisle, & Birmingham, 2012). Even if no support is provided from another person, perceiving that social support is available can be an important coping resource that helps reduce distress and facilitate coping (e.g., Pow, King, Stephenson, & DeLongis, 2016). Although some researchers distinguish between individual and dyadic forms of coping (e.g., Badr & Acitelli, 2017), social influences can have an even more pervasive effect throughout the coping process. The social context can affect how stressors are appraised, which coping strategies are used, and whether they are effective (DeLongis & Holtzman, 2005). Individuals can cope in ways that provide a good or bad fit with the coping strategies used by others involved in the situation. For example, although avoidance may be associated with poorer adjustment overall, couples in which one partner was high in avoidance and the other was low in avoidance reported better dyadic adjustment (Badr, 2004). This suggests that spouses can cope in ways that buffer against the negative effects of what might otherwise be maladaptive coping strategies. Although existing research examining the fit between coping strategies of involved others has focused primarily on dyads, this perspective could be applied to wider social networks such as provider–patient–spouse triads or larger family systems.

The social context surrounding the coping process can be extended to include cultural influences. The effectiveness of a given coping strategy must also be considered within its cultural context. Although some aspects of the coping process are considered culturally universal, there are also cultural differences in the degree to which individuals are exposed to certain types of stressors, how these events are appraised, which coping strategies are used, and whether these coping responses are considered effective (Chun, Moos, & Cronkite, 2006).

Summary

Although some coping strategies are more consistently associated with successful adaption than others, there is no one coping strategy that is universally adaptive or maladaptive. Contextual factors play an important role in determining the coping strategies used and the outcomes of the coping process. The nature of the stressful situation, the personality of the individual, and social context can all influence whether or not a particular coping strategy is employed and whether or not it is effective.

Author Biographies

Ellen Stephenson is a PhD student in the Department of Psychology at the University of British Columbia. Her research focuses on understanding how couples and families come together to cope with stress. She has examined ways in which close relationships can facilitate successful coping and individual adjustment (e.g., reduced pain and functional impairment) and the consequence stress and coping can have for relationship outcomes (e.g., divorce and marital satisfaction).

Anita DeLongis is a professor in the Department of Psychology at the University of British Columbia. Her research in health psychology focuses on stress, coping, and social support. Her current work spans a number of areas including rheumatoid arthritis and chronic pain, spinal cord injury, stress in stepfamilies, stress among emergency medical personnel, health behaviors such as condom use, and psychological reactions to global pandemics.

References

- Aspinwall, L. G., & Taylor, S. E. (1997). A stitch in time: Self-regulation and proactive coping. *Psychological Bulletin*, 121(3), 417–436. doi:10.1037/0033-2909.121.3.417
- Austenfeld, J. L., & Stanton, A. L. (2004). Coping through emotional approach: A new look at emotion, coping, and health-related outcomes. *Journal of Personality*, 72, 1335–1363. doi:10.1111/j.1467-6494.2004.00299.x
- Badr, H. (2004). Coping in marital dyads: A contextual perspective on the role of gender and health. *Personal Relationships*, 11, 197–211. doi:10.1111/j.1475-6811.2004.00078.x
- Badr, H., & Acitelli, L. K. (2017). Re-thinking dyadic coping in the context of chronic illness. *Current Opinion in Psychology*, 13, 44–48. doi:10.1016/j.copsyc.2016.03.001
- Bolger, N., Stadler, G., Paprocki, C., & DeLongis, A. (2010). Grounding social psychology in behavior in daily life: The case of conflict and distress in couples. In C. R. Agnew, D. E. Carlston, W. G. Graziano, & J. R. Kelly (Eds.), *Then a miracle occurs: Focusing on behavior in social psychological theory and research* (pp. 368–390). New York, NY: Oxford University Press.
- Chen, E., & Miller, G. E. (2012). “Shift-and-persist” strategies: Why low socioeconomic status isn’t always bad for health. *Perspectives on Psychological Science*, 7, 135–158. doi:10.1177/1745691612436694
- Cheng, C., Lau, H. B., & Chan, M. S. (2014). Coping flexibility and psychological adjustment to stressful life changes: A meta-analytic review. *Psychological Bulletin*, 140, 1582–1607. doi:10.1037/a0037913
- Chun, C.-A., Moos, R. H., & Cronkite, R. C. (2006). Culture: A fundamental context for the stress and coping paradigm. In P. T. P. Wong & L. C. J. Wong (Eds.), *International and cultural psychology series. Handbook of multicultural perspectives on stress and coping* (pp. 29–53). New York, NY: Springer. doi:10.1007/0-387-26238-5_2
- Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin*, 127, 87–127. doi:10.1037//0033-2909.127.1.87
- DeLongis, A., & Holtzman, S. (2005). Coping in context: The role of stress, social support, and personality in coping. *Journal of Personality*, 73, 1633–1656. doi:10.1111/j.1467-6494.2005.00361.x
- DeLongis, A., Holtzman, S., Puterman, E., & Lam, M. (2010). Spousal support and dyadic coping in times of stress. In K. Sullivan & J. Davila (Eds.), *Support processes in intimate relationships* (pp. 153–174). New York, NY: Oxford University Press.
- DeLongis, A., & O’Brien, T. B. (1990). An interpersonal framework for stress and coping: An application to the families of Alzheimer’s patients. In M. A. P. Stephens, J. H. Crowther, S. E. Hobfoll, & D. L. Tennenbaum (Eds.), *Stress and coping in later life families* (pp. 221–239). Washington, DC: Hemisphere Publishers.
- Folkman, S., Lazarus, R. S., Gruen, R. J., & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of Personality and Social Psychology*, 50, 571–579. doi:10.1037/0022-3514.50.3.571
- Franks, H. M., & Roesch, S. C. (2006). Appraisals and coping in people living with cancer: A meta-analysis. *Psycho-Oncology*, 15, 1027–1037. doi:10.1002/pon.1043
- Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York, NY: Guilford Press.
- Haan, N. (1969). A tripartite model of ego functioning values and clinical and research applications. *The Journal of Nervous and Mental Disease*, 148(1), 14–30. doi:10.1097/00005053-196901000-00003
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer.
- Lee-Baggeley, D., Preece, M., & DeLongis, A. (2005). Coping with interpersonal stress: Role of big five traits. *Journal of Personality*, 73(5), 1141–1180. doi:10.1111/j.1467-6494.2005.00345.x
- Menninger, K. (1963). *The vital balance: The life process in mental health and illness*. New York, NY: Viking Penguin.

- O'Brien, T. B., & DeLongis, A. (1996). The interactional context of problem-, emotion-, and relationship-focused coping: The role of the big five personality factors. *Journal of Personality*, 64, 775–813.
- O'Brien, T. B., DeLongis, A., Pomaki, G., Puterman, E., & Zwicker, A. (2009). Couples coping with stress: The role of empathic responding. *European Psychologist*, 14, 18–28. doi:10.1027/1016-9040.14.1.18
- Pow, J., King, D. B., Stephenson, E., & DeLongis, A. (2016). Does social support buffer the effects of occupational stress on sleep quality among paramedics? A daily diary study. *Journal of Occupational Health Psychology*. doi:10.1037/a0040107
- Pow, J., Lee-Baggley, D., & DeLongis, A. (2016). Threats to communion and agency mediate associations between stressor type and daily coping. *Anxiety, Stress, and Coping*, 29(6), 660–672. doi:10.1080/10615806.2015.1126258
- Revenson, T. A., Kayser, K., & Bodenmann, G. (2005). *Couples coping with stress: Emerging perspectives on dyadic coping*. Washington, DC: American Psychological Association.
- Roth, S., & Cohen, L. J. (1986). Approach, avoidance, and coping with stress. *The American Psychologist*, 41, 813–819. doi:10.1037/0003-066X.41.7.813
- Schwarzer, R., & Knoll, N. (2003). Positive coping: Mastering demands and searching for meaning. In S. J. Lopez & C. R. Snyder (Eds.), *Handbook of positive psychological assessment* (pp. 393–409). Washington, DC: American Psychological Association. doi:10.1037/10612-025
- Skinner, E. A., Edge, K., Altman, J., & Sherwood, H. (2003). Searching for the structure of coping: A review and critique of category systems for classifying ways of coping. *Psychological Bulletin*, 129, 216–269. doi:10.1037/0033-2909.129.2.216
- Uchino, B. N., Bowen, K., Carlisle, M., & Birmingham, W. (2012). Psychological pathways linking social support to health outcomes: A visit with the “ghosts” of research past, present, and future. *Social Science & Medicine*, 74, 949–957. doi:10.1016/j.socscimed.2011.11.023
- Vaillant, G. E. (1977). *Adaptation to life*. Boston, MA: Little Brown.
- Zakowski, S. G., Hall, M. H., Klein, L. C., & Baum, A. (2001). Appraised control, coping, and stress in a community sample: A test of the goodness-of-fit hypothesis. *Annals of Behavioral Medicine*, 23, 158–165.

Suggested Reading

- DeLongis, A., & Holtzman, S. (2005). Coping in context: The role of stress, social support, and personality in coping. *Journal of Personality*, 73(6), 1633–1656.
- Folkman, S., & Moskowitz, J. T. (2004). Coping: Pitfalls and promise. *Annual Review of Psychology*, 55, 745–774.
- Skinner, E. A., Edge, K., Altman, J., & Sherwood, H. (2003). Searching for the structure of coping: A review and critique of category systems for classifying ways of coping. *Psychological Bulletin*, 129(2), 216.
- Taylor, S. E., & Stanton, A. L. (2007). Coping resources, coping processes, and mental health. *Annual Review of Clinical Psychology*, 3, 377–401.

Coping with Chronic Illness

Aliza A. Panjwani¹ and Tracey A. Revenson²

¹*Graduate School and University Center, City University of New York, New York, NY, USA*

²*Department of Psychology, Hunter College, New York, NY, USA*

Up to the mid-twentieth century, the leading causes of sickness and death were acute and infectious diseases, such as malaria, tuberculosis, and influenza, that resulted from contaminated water and food and poor hygiene. Until public health advances brought these conditions under control, there was little physicians could do. Now, particularly in developed countries, few people die from acute infections. Does that mean that there is less illness? To the contrary, approximately 117 million American adults are living with a chronic condition, the most prevalent being cardiovascular and pulmonary diseases, cancer, and diabetes. This number is expected to grow as the nation's life expectancy increases and baby boomers age. By 2030, the number of adults over the age of 65 will double, accounting for 20% of the population. As such, the number of people living with chronic illnesses is expected to rise to over 160 million by 2040.

What Is a Chronic Illness?

Alana was diagnosed with multiple sclerosis (MS) in her mid-20s while she was in social work school. Symptoms halted within a few years. She married, had children, and developed a career nearer to her heart – landscape design. In her late 40s, symptoms reemerged, and within five years she was having trouble walking, forcing her to give up her landscape business and move to a one-level house. She continues to be involved in her temple and sees her extended family often but wonders what the next decade will bring.

Chronic diseases are enduring health conditions that cause disruptions in daily activities and have no cure. Some are degenerative in nature, marked by increasing disability and pain (e.g., MS, rheumatoid arthritis), while others can be managed with treatment (e.g., hypertension, diabetes, HIV/AIDS). According to the commonsense framework of illness representations (Leventhal, Brissette, & Leventhal, 2003), which describes the ways ordinary people think

about disease, chronic and acute illnesses can be distinguished along four dimensions: cause, timeline, illness identity, and outcomes. The *cause* of many chronic diseases is a result of lifestyle and health behaviors, such as diet and exercise; in contrast, acute diseases are often caused by bacteria or injuries. Acute diseases are considered short term, whereas chronic illnesses have a longer *timeline* with a slow onset and long-term trajectory. Consequently, the *identity* of a chronic illness is less concrete: the causes, trajectory, and symptomology are often unpredictable or unobservable. Take the case of hypertension—people may not recognize its symptoms, although clinically elevated blood pressure is dangerous for one's health. Finally, the *outcomes* of chronic illness, as compared with infectious diseases, are not clear-cut. Even with treatment, most chronic diseases remain incurable. Many are progressive, often involving chronic or intermittent pain, increasing physical disability, and lifelong illness management.

MS, the disease Alana was diagnosed with, provides a prototypical illustration of a chronic disease. MS is a chronic neurological condition, in which the myelin sheath, a protective covering surrounding nerve fibers in the central nervous system, deteriorates. Patients with MS alternate through periods of symptom relapse and remission. They experience a range of physical (pain, fatigue, difficulty walking, sexual impairment, paralysis) and psychological (increased depression, uncertainty, anxiety, cognitive impairments) symptoms that are difficult to predict and often worsen over time. As the cause and cure of MS remain unknown, current treatment is mostly geared toward management of symptoms and decelerating illness progression.

The Stresses of Chronic Illness

As chronic diseases progress, patients experience changes related to social relationships, work, and daily life. Many psychological frameworks have described challenges individuals with chronic illnesses encounter. Illness creates disruptions in many areas that impinge upon a patient's life, in areas of work or finances, social relationships, sex life, and personal development. For example, when women with rheumatoid arthritis, a disabling autoimmune disease, strongly identified with roles at work or as parents, illness intrusions affected their psychological well-being to a greater degree. This finding suggests that disease-related disruptions call into question aspects of one's identity.

Moos and Holahan (2007) describe an *adaptive tasks* framework that separates the impact of illness into two broad categories: illness-related tasks and general life-related tasks. Illness-related adaptive tasks include managing symptoms and treatment and developing relationships with healthcare professionals. General adaptive tasks include maintaining an emotional balance, a positive self-image, and relationships with loved ones as well as preparing for an uncertain future.

What Is Adaptation to Illness?

Stanton and Revenson (2011) describe adaptation to illness in a number of ways. First, adaptation is multifaceted and dynamic. Second, people can feel both negative and positive emotions throughout their illness trajectory. Third, the process of adjustment is heterogeneous; not everyone experiences identical challenges, and each individual brings different personal and psychological resources "to the table." Another explanation for this heterogeneity is that adaptation depends on context. Context includes the medical aspects of the illness (Has the person lost an essential functional ability, such as walking or remembering things? Is the treatment

making one sick and to what degree?), aspects of the social environment (Are there people that can provide support?), and availability of resources (Does the patient have health insurance? Does she live in a home environment that allows for independence?).

Cognitive Adaptation to Chronic Illness

Taylor (1983) proposed a theory of cognitive adaptation that continues to ground research on psychosocial adjustment to illness today. Based largely on interviews with a sample of women with breast cancer, Taylor delineated three essential processes that promote adaptation to illness: *making meaning of the illness, regaining mastery, and restoring self-esteem*.

Meaning making

Encountering a life-altering stressor, such as an illness, creates inconsistencies in our beliefs about the world, self, and personal goals. At this time, people initiate cognitive processes, such as “meaning making,” to align those recent experiences with existing belief systems. The search for meaning often entails efforts to answer existential questions, like “Why me?” In Taylor’s (1983) study, most of the breast cancer survivors were able to provide some explanation as to why their cancer occurred. Having (any) explanation was more strongly related to adjustment than *not* having one; the type of attribution made (self or environment) did not matter as much. Park (2010) echoed this early finding in a review of over 75 studies: successful meaning making had a positive impact on adjustment to stressful life events, including living with or caring for someone with a chronic illness. Meaning making leads to increases in personal resources, such as optimism, self-efficacy, and self-esteem. Incidentally, these three personal resources have been shown to promote adjustment among people with various chronic illnesses like diabetes, cancer, and arthritis.

Mastery

Mastery refers to an individual’s perception of control over life events and capability to achieve desired outcomes. In attempts to control the illness, the possibility of recurrence, or the side effects of treatment, the breast cancer patients in Taylor’s (1983) study exerted mental (e.g., positive thinking) and behavioral (e.g., dietary changes) coping efforts, which built a sense of mastery. Mastery protects individuals against depressive symptoms and increases quality of life in studies of many illnesses, such as diabetes, lung and cardiac disease, arthritis, cancer, and HIV. An increased sense of personal control mitigated the negative effects of stress on mental health, possibly through the use of more adaptive coping strategies. A randomized controlled trial, designed to improve symptom management in patients diagnosed with late-stage cancers, found that an increased sense of mastery was related to lower levels of fatigue and pain, particularly when participants felt confident in their ability to use the coping techniques taught in the intervention. People who have high mastery are more likely to feel capable of utilizing active coping strategies, such as reframing life challenges in a more positive light or using problem-solving skills.

Restoring self-esteem

Chronic illness can diminish self-esteem. Some people report feeling betrayed by their bodies. Surgery and treatment often cause permanent changes in physical appearance and self-concept. Thus, it is critical to restore self-esteem. Taylor (1983) found that people cope with threatening events by comparing themselves with less fortunate others to maintain a positive outlook. These *downward comparisons*, in turn, result in increased self-esteem. In Taylor’s

study of breast cancer patients, most women were able to make these downward comparisons by finding a target for comparison that was worse off than they were. For example, women who had a lumpectomy (removal of the tumor only, conserving the breast) compared themselves with those who had a mastectomy (invasive surgery to remove the entire breast). Those who had a mastectomy compared themselves with patients whose cancer metastasized (spread to vital organs). It was always possible to find someone in a situation more severe than one's own.

Theories of Coping

Coping has been widely studied in social, personality, developmental, clinical, and health psychology (see entries on Coping Strategies and Coping Styles in this volume) and is essential to understanding adjustment to chronic illness.

The Stress and Coping Paradigm

Perhaps the most well-known theory of coping is Lazarus and Folkman's (1984) stress and coping paradigm, in which coping is defined as "constantly changing cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person" (p. 141). There is a lot to consider in this definition. First, like the process of adaptation, coping is dynamic. People can utilize different coping strategies depending on context or phase of illness. For example, a newly diagnosed cancer patient may use different coping strategies than when he or she is at a more advanced stage of illness. Second, coping involves cognitions (thoughts, beliefs) and behaviors (actions). A person with multiple sclerosis, like Alana, may engage in active attempts to seek social support or change her environment (behavior) as well as efforts to reframe the illness (cognition). Third, stressors can be external (changing one's work schedule because of chemotherapy) or internal (worry about progression of illness). Fourth, though coping and adaptation are related, they are not the same concept. That is, we cannot make a priori determinations about whether a specific coping strategy is effective; rather, the success of a coping strategy depends on context.

The stress and coping paradigm proposes that the type of coping strategies used, or the decision to "cope" at all, is based on *psychological appraisals* of the stressor. Individuals first evaluate whether or not an event qualifies as a stressor in a process called *primary appraisal*: What does this stressor mean for my health? Is it a threat (future stressor), loss (past stressor), or challenge? Then, in a process known as *secondary appraisal*, they decide whether they have the resources to cope with the stressor: Can I control it? Have I experienced this before and what did I do then? Based on these appraisals, individuals choose one or more coping strategies in response to the stressor. After attempts to cope with a stressor, individuals reappraise whether coping efforts have been successful, and the appraisal-coping cycle begins anew.

In the original stress and coping theory, coping efforts were classified as either problem or emotion focused. Problem-focused coping refers to efforts to change the situation by resolving or minimizing the stressor (e.g., planning, seeking support). Emotion-focused coping refers to managing the emotional distress caused by the stressor, particularly if the stressor is uncontrollable. Emotion-focused coping involves expressing and processing emotions, minimizing the stressor, self-soothing (e.g., drinking, shopping), and seeking emotional support. However, many coping efforts contain aspects of both problem- and emotion-focused coping

(e.g., asking for social support), so this distinction has become less important. A more complex definition, *emotional approach coping*, involves processing and expressing emotions (Stanton, 2011).

Approach Versus Avoidance

A second approach classifies coping strategies as *approach* or *avoidance* (Carver, 2007). These categories refer to an individual's motivation to either confront or avoid a stressor. Approach-oriented strategies are active (i.e., seeking social support, problem solving), whereas avoidance-oriented strategies are geared toward escape (i.e., denial, abusing drugs and alcohol).

A Combination Approach

Moos and Holahan (2003) combined the focus of coping (approach vs. avoidance) with the type of coping (behavioral vs. cognitive). Behavioral coping refers to strategies used to change situations, whereas cognitive coping involves changing thoughts or perceptions of stressors, resulting in four categories of coping strategies: *behavioral approach* (i.e., seeking social support), *cognitive approach* (i.e., positively reframing the situation), *behavioral avoidance* (i.e., using drugs and alcohol), and *cognitive avoidance* (i.e., engaging in denial or minimization).

It is worthwhile to note that these three theories overlap. For example, the strategy of positive reappraisal could be considered an emotion-focused coping strategy, an approach-oriented coping strategy, or a cognitive approach coping strategy.

Coping and Adaptation: What Works?

Though no single coping strategy is effective for all people all the time, research suggests that avoidance-oriented coping is associated with poorer illness adjustment than approach-oriented coping. Much of this evidence comes from cross-sectional studies, and, therefore, causality cannot be established. However, a number of systematic reviews indicate that specific coping strategies affect mental health in different ways. In many studies, avoidant strategies have been associated with increased anxiety and depression among people with MS (like Alana), malignant melanoma (type of cancer with a poor prognosis), heart failure, and HIV/AIDS.

Does approach-oriented coping confer salutary (positive) benefits on adjustment? Problem-solving coping has been associated with decreased physical symptoms, negative affect, depressive symptoms, and fatigue—that is, better mental and physical health. Similarly, seeking social support also has been associated with positive adjustment to illness, although the magnitude of this relationship is often smaller than the one between avoidance-oriented coping (i.e., denial) and negative adjustment. Thus, approach-oriented coping might increase adjustment through the reduction of negative emotional states such as depression and fatigue.

The positive effects of approach-oriented coping depend on contextual factors, such as stage of illness. For example, among HIV/AIDS patients, behavioral approach-oriented coping, such as seeking information about treatment, was more effective at earlier points in the disease trajectory. Similarly, the association between approach-oriented coping and adjustment among people with late-stage cancers is not as strong when compared with those with early-stage cancers. One reason for this may be that later stages of illness present different challenges (i.e., making end-of-life arrangements) than earlier stages (i.e., deciding between treatment options).

Posttraumatic Growth as Adjustment

Studies examining adaptation to illness mostly focus on indicators of negative adjustment, such as depressive symptoms and anxiety. In this context, “good” adjustment refers to fewer symptoms or lower anxiety. In comparison, fewer studies look at positive outcomes, although that has changed in recent years.

Posttraumatic growth (PTG) refers to a positive change experienced as a result of adversity, which reflects higher levels of psychological functioning. People who report PTG find themselves transformed as a result of illness, often by building a reservoir of personal strength, acquiring a better appreciation of life, or strengthening relationships with family and friends.

Research has found a relationship between coping with illness and PTG. Approach-oriented strategies (e.g., acceptance, positive reappraisal) are associated with PTG, whereas avoidant strategies (e.g., escape, self-blame) are not. Although studies examining the construct of PTG have been primarily conducted with cancer populations, approach-oriented coping (positive reappraisal, seeking emotional support, and acceptance) has been associated with greater PTG among individuals with a number of diseases, including MS, rheumatoid arthritis, heart disease, and HIV/AIDS. Consequently, one should consider both positive and negative aspects of adaptation to illness.

Dyadic Coping

Most studies on coping with chronic diseases have focused on individual coping efforts. Recently, an interest has developed in how couples cope with a chronic illness, as a unit (Revenson & DeLongis, 2011). Patient and partner psychological distress are related; as one partner’s distress increases, so does the other partner’s. A systematic review of studies of cancer patients found that women experienced more distress than men—whether they were the cancer patients or partners (Hagedoorn, Sanderman, Bolks, Tuinstra, & Coyne, 2008). This is likely reflective of the gender difference in depression found in the general population, with women reporting and experiencing greater depression (whether they have a physical illness or not). Overall, the meta-analysis results highlight the need for researchers to examine the impact of illness on the patient and partners, as well as the mutual influence of patients and partners on each other (Revenson & DeLongis, 2011).

Dyadic coping (Bodenmann, 2005) refers to the idea that when an adverse event occurs, couples evaluate the event as a shared stressor, combining their resources to cope with the problem together. Dyadic coping draws on the stress and coping model (Lazarus & Folkman, 1984) described earlier.

Relationship-focused coping, a form of dyadic coping, involves cognitive and behavioral efforts to resolve stressors while keeping close relationships intact. Two types of relationship-coping strategies are often studied: *protective buffering* and *active engagement*. Protective buffering refers to suppressing one’s own worries in order to minimize the other partner’s distress or to avoid relationship conflict. For example, a husband undergoing cancer treatment may hide his fears of dying in order to keep his wife from worrying. A number of studies have shown that protective buffering is harmful to the person doing the protecting. Active engagement, however, involves including one’s partner in dialogue about stressful circumstances to generate solutions that are acceptable to both parties. Active engagement may benefit patients by allowing them to reclaim a sense of personal control over the events surrounding their illness; it may benefit partners by reducing feelings of helplessness.

Consider Alana, the woman diagnosed with MS we referred to earlier. She may involve her husband in various decision-making discussions about treatment options, the children's needs, and her career plans. This engagement, in turn, may prove better not just for patients like Alana but also for a couple's overall relationship satisfaction. Protective buffering may not necessarily yield the same benefits. Women often refrain from asking their ill husbands for emotional support to protect them from experiencing additional distress, even at the cost of the women's own emotional well-being.

The *systemic transactional model* (STM) of coping (Bodenmann, 2005) provides a framework for dyadic research that has been utilized in many studies. When faced with a shared stressor, partners respond both individually and as a collective unit. At the individual level (see the stress and coping paradigm above), a person's cognitive appraisal of a dyadic stressor is shaped by his or her own needs and concerns. Based on these appraisals, a stress communication process is triggered, whereby each person communicates his or her own stress to the partner in hopes of receiving emotional and/or practical support. At the dyadic level, the couple copes to manage aspects of the illness together. In the STM, adjustment includes both individual-level outcomes (e.g., distress) and dyad-level outcomes (e.g., relationship quality).

The STM distinguishes among different types of dyadic coping. Positive dyadic coping includes shared problem solving, information seeking, emotional expression, practical support, mutual commitment, and task delegation. Negative dyadic coping involves ignoring requests for support as well as avoiding the problem and the partner. Joint problem solving (a form of positive dyadic coping) was associated with decreased cancer-related distress and increased marital adjustment among women with metastatic breast cancer and their husbands; mutual avoidance (a form of negative dyadic coping) was related to poorer adjustment. A longitudinal study of couples facing breast cancer corroborated these findings and also suggested that certain types of dyadic coping may benefit one partner and not the other. Specifically, patients may experience increased distress when they are delegated tasks by their partners, whereas *partners* might benefit when patients delegate tasks to them. One reason for this may be that being delegated tasks by one's partner reduces the caregiver's feelings of helplessness, not necessarily the patient's. Although the study of dyadic coping is complex, there is solid evidence that a chronic illness is a challenge, shared by both patients and their intimate partners, and that studying dyadic processes within this context is a warranted endeavor.

Family Coping

According to *family systems theory*, the family is a unitary system, composed of related individual parts that influence each other. A family member's diagnosis is likely to create both individual and shared stressors, calling for readjustment on the part of the entire family, not just the person who is ill or the spouse. As the scope of this topic is vast, we focus only on parents and siblings coping with a child's illness.

Studies on the psychological impact of childhood illness have documented increased post-traumatic stress, depressive symptoms, and anxiety among parents. Moreover, parents reported the greatest uncertainty, anxiety, depressive symptoms, and post-traumatic stress immediately after their child's cancer diagnosis. Some parents continued to experience these sequelae 5 or more years after the diagnosis. Mothers often report more stressors than fathers, as they are likely to provide more care to the ill child.

Particular parental coping strategies are more effective at specific stages of illness. Avoidance-oriented coping strategies are most functional at early stages of the child's illness when parents reported feeling overwhelmed by the diagnosis and the uncertainty it brings. When children are in treatment, however, avoidant coping was related to increased psychological distress, and approach-oriented coping was related to decreased distress. A number of studies have shown that elevated parental distress poses psychological implications for the child. For example, parental distress is related to depressive symptoms and may interfere with parents' management of their child's treatment regimen.

Sibling Coping and Adaptation to Illness

There is mixed evidence on psychological adjustment of siblings of chronically ill children. A few studies found little difference in functioning between siblings of ill and healthy children in the same family. Other studies established that siblings of ill children have greater symptoms of depression and anxiety, engage in fewer peer interactions, exhibit increased behavioral problems, and use more avoidant coping. These findings suggest that siblings of ill children may retreat "inward" and be less likely to actively seek support when needed.

Female siblings are more vulnerable to psychological distress, which may be due in part to the notion that they take on increased household responsibilities because their mother is caring for the ill child. Older siblings are at higher risk than younger siblings for psychological distress, especially when the ill child had a life threatening. Because of their cognitive ability to understand consequences of a chronic condition and its toll on the parents and the family, older siblings may be more likely to suppress feelings. Conversely, parents may be more likely to give older siblings more responsibility, thinking them better able to care for themselves than younger siblings.

Future Directions in Coping and Adaptation to Illness

Coping with chronic illness is a complex psychological process for individuals, dyads, and families. What we *do* know is that coping is shaped by many factors, including psychological factors (e.g., mastery) and illness stage (e.g., time of diagnosis, treatment phase). However, many unanswered questions remain. We know little about how coping processes unfold over time as many studies employ cross-sectional designs, studying individuals or families at one point in time. Often, only one illness is studied. Are there differences in coping and adaptation processes that are consistent across illnesses? The adaptive tasks model suggests that there are similar challenges across illnesses, but we have little data to support this. Future work would benefit from including immediate family members, examining factors that promote and hinder family adaptation to chronic illness, and using longitudinal methodologies. This information is critical for the development of cost- and time-efficient interventions geared toward improving the psychological well-being of those living with a chronic illness and their loved ones, starting at diagnosis and continuing beyond.

Author Biographies

Aliza A. Panjwani, MA, is a doctoral student at the Graduate Center, City University of New York. Her research interests lie in better understanding how individuals with chronic

illness, as well as caregivers, cope with the stresses associated with disease, how situational factors interact with individual differences to influence adaptation, and how personal resources promote resilience.

Tracey A Revenson, PhD, is professor of psychology at Hunter College and the Graduate Center, City University of New York. Her research focuses on factors that affect psychosocial adjustment among individuals, couples, and families facing serious physical illness. In addition to many chapters and articles, she has co-authored 10 volumes, including *Caregiving in the Illness Context* (2015). In 2013 she received the Nathan Perry Award for Career Contributions to Health Psychology from APA.

References

- Bodenmann, G. (2005). Dyadic coping and its significance for marital functioning. In T. A. Revenson, K. Kayser, & G. Bodenmann (Eds.), *Couples coping with stress: Emerging perspectives on dyadic coping* (pp. 33–50). Washington, DC: American Psychological Association.
- Carver, C. S. (2007). Stress, coping, and health. In H. S. Friedman & R. Cohen Silver (Eds.), *Foundations of health psychology* (pp. 117–144). Oxford, UK: Oxford University Press.
- Hagedoorn, M., Sanderman, R., Bolks, H. N., Tuinstra, J., & Coyne, J. C. (2008). Distress in couples coping with cancer: A meta-analysis and critical review of role and gender effects. *Psychological Bulletin*, 134, 1–30. doi:10.1037/0033-2909.134.1.1
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York, NY: Springer Publishing Company.
- Leventhal, H., Brissette, I., & Leventhal, E. A. (2003). The common-sense model of self-regulation of health and illness. In L. D. Cameron & H. Leventhal (Eds.), *The self-regulation of health and illness behavior* (pp. 42–65). New York, NY: Routledge.
- Moos, R. H., & Holahan, C. J. (2003). Dispositional and contextual perspectives on coping: Toward an integrative framework. *Journal of Clinical Psychology*, 59(12), 1387–1403. doi:10.1002/jclp.10229
- Moos, R. H., & Holahan, C. J. (2007). Adaptive tasks and methods of coping with illness and disability. In E. Martz & H. Livneh (Eds.), *Coping with chronic illness and disability* (pp. 107–128). New York, NY: Springer.
- Park, C. L. (2010). Making sense of the meaning literature: An integrative review of meaning making and its effects on adjustment to stressful life events. *Psychological Bulletin*, 136(2), 257–301. doi:10.1037/a0018301
- Revenson, T. A., & DeLongis, A. (2011). Couples coping with chronic illness. In S. Folkman (Ed.), *Oxford handbook of stress, health, and coping* (pp. 101–123). New York, NY: Oxford University Press.
- Stanton, A. L., & Revenson, T. A. (2011). Adjustment to chronic disease: Progress and promise in research. In H. S. Friedman (Ed.), *Oxford handbook of health psychology* (pp. 241–268). New York, NY: Oxford University Press.
- Taylor, S. E. (1983). Adjustment to threatening events: A theory of cognitive adaptation. *American Psychologist*, 38(11), 1161–1173. doi:10.1037/0003-066X.38.11.1161
- Stanton, A. L. (2011). Regulating emotions during stressful experiences: The adaptive utility of coping through emotional approach. In S. Folkman (Ed.), *The Oxford handbook of stress, health, and coping* (pp. 369–386). New York, NY: Oxford University Press.

Suggested Reading

- Cole, R. E., & Reiss, D. (Eds.) (1993). *How do families cope with chronic illness?* Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

- Folkman, S. (2011). *The Oxford handbook of stress, health, and coping*. New York, NY: Oxford University Press.
- Moss-Morris, R. (2013). Adjusting to chronic illness: Time for a unified theory. *British Journal of Health Psychology*, 18(4), 681–686. doi:10.1111/bjhp.12072
- Stanton, A., Revenson, T. A., & Tennen, H. (2007). Health psychology: Psychological adjustment to chronic disease. *Annual Review of Psychology*, 58, 13.1–13.28. doi:10.1146/annurev.psych.58.110405.085615

Counterfactual Thought

Rachel Smallman¹, Amy Summerville², Ryan J. Walker²,
and Brittney Becker¹

¹*Psychology Department, Texas A&M University System, College Station, TX, USA*

²*Psychology Department, Miami University, Oxford, OH, USA*

Counterfactual thoughts contrast reality to an imagined alternative: “what might have been” had some action or circumstance been other than it really was. For instance, an individual might think “if only I had stopped smoking sooner” or “at least we caught it early” when receiving a cancer diagnosis. Counterfactuals are a pervasive part of our mental landscape. Their evaluative nature facilitates causal reasoning. For instance, the counterfactual “if only I hadn’t been speeding, I wouldn’t have gotten into the accident” identifies speed as the cause of the accident, rather than weather conditions or a distracted driver. Counterfactuals therefore influence the formation of future intentions and subsequent behavior (e.g., intentions to obey speed limits and actually obeying speed limits in the future; for a review see Epstude & Roese, 2008; Roese, 1997; Roese & Epstude, 2017).

Counterfactuals can identify alternatives that are either better than reality (*upward counterfactuals*) or worse than reality (*downward counterfactuals*). Upward counterfactuals are generally related to self-improvement motivations (Roese, 1997), as they identify ways that an outcome might have been improved and thus suggest strategies to improve future outcomes (e.g., “if only I’d stretched first, I wouldn’t have gotten injured” suggests that stretching will reduce future injury). Upward counterfactuals facilitate the generation of intentions (Roese, 1994; Smallman & Roese, 2009) but are associated with negative affect and underpin the emotional experience of regret (Gilovich & Medvec, 1995). In contrast, downward counterfactuals are related to self-enhancement motivations and generally improve affect (Roese, 1997). For instance, the thought “at least I don’t need surgery” might make one feel better about having a minor injury. Exceptions to this pattern occur when the outcomes are ongoing rather than final. In these cases, individuals may focus more on similarities rather than differences between reality and the imagined alternative (Markman & McMullen, 2003). The team that is losing by one point at halftime, the student who almost gets an A on the first quiz, and the dieter who is almost at his goal weight may all feel hopeful and motivated rather than dejected if they perceive

ongoing opportunity to reach these goals, whereas the team with a narrow lead or the driver who narrowly missed an accident may feel apprehensive rather than overjoyed if they perceive continued threat. All counterfactuals can be structured either as *additive* counterfactuals, focused on the addition of actions that might have occurred, but didn't (e.g., "If only I had exercised more ..."), or as *subtractive* counterfactuals, focused on the deletion of actions that did occur, but should not have (e.g., "If only I hadn't consumed so much alcohol ...").

Two broad factors increase the likelihood of counterfactual generation: the valence (positive or negative) of an outcome and the ease of simulating alternatives. Counterfactuals are more likely following negative events, failure, or unmet goals (Roese, 1997). In general, counterfactuals are most likely to be generated when an alternative is easy to imagine and would clearly have led to another outcome (Kahneman & Miller, 1986). Unusual events are more likely to elicit counterfactuals: individuals are more likely to generate counterfactuals about a driver's route when he got into an accident if the driver took a detour rather than his typical commute (Kahneman & Miller, 1986). Finally, events that more nearly occurred are more likely to prompt counterfactuals: a student who got an 89% is more likely to generate counterfactuals about the exam than a student who got an 87%, because the former grade is closer to having been an A (Roese, 1997).

Given their strong links to emotional experience and decision making, counterfactuals play a broad and varied role in health. At times, counterfactuals can have deleterious effects: upward counterfactuals and the related emotion of regret are associated with various negative mental health outcomes, including depression, anxiety, and problems coping with traumatic events. Additionally, although counterfactual thought is a common and important cognitive process in healthy populations, particular health conditions are associated with deficits in counterfactual thought. Counterfactuals can also have mental health benefits, including promoting a sense of meaning in life. Furthermore, because of their important role in causal reasoning and future planning, counterfactuals can benefit health decisions, changing behavior by either strengthening behavioral intentions or increasing motivation and persistence.

Mental Health and Well-Being

Both excess and deficient counterfactual thought are associated with a broad range of negative outcomes. Mental disorders that are characterized by emotional distress or challenges in coping generally involve excessive or extreme responses to upward counterfactuals. In contrast, other disorders such as schizophrenia and neurological conditions including Parkinson's disease show deficits in counterfactual thought. However, both upward and downward counterfactuals can facilitate coping and enhance well-being.

Excessive Counterfactual Thought

Thoughts of "what might have been" are associated with emotional distress, even among healthy populations. Depression and anxiety are particularly associated with increased counterfactual generation. Although depressed and nondepressed individuals generate the same number of counterfactuals when given explicit instructions, Quelhas and colleagues found that depressed individuals generate more counterfactuals spontaneously, that is, when asked to describe their thoughts without specific instructions to think about "what might have been." Likewise, participants high in social anxiety are more likely to generate upward counterfactuals about social events compared with those low in social anxiety. The relationships between

counterfactuals, anxiety, and depression are not due simply to the presence of ruminative thinking in depression. In a nationally representative phone survey conducted in 2009, Roese and colleagues found that high levels of regret predicted depression and anxiety better than excessive rumination and the interaction between regret and repetitive thoughts was highly predictive of general distress (Roese et al., 2009).

Other research has focused on the role of chronic deficits in personal control in counterfactual thought. In the 1990s, Markman and colleagues found that perceived control mediated the association between depression and increased generation of controllable counterfactuals. Thus, these authors suggest that increased counterfactual generation is a response to greater chronic motivations for control among depressed participants.

In addition to the greater prevalence of counterfactual thought, depression is also associated with more negative responses to upward counterfactuals. For example, severely depressed participants generate counterfactuals that are less controllable and less reasonable, compared with mildly or moderately depressed participants. These less reasonable and controllable counterfactuals are less useful for managing ongoing behavior. Thus, although counterfactual thoughts can be functional, people with depression may generate less functional or potentially dysfunctional counterfactual thoughts.

Beyond its connections to depression and anxiety, excessive counterfactual thought directly contributes to insomnia. A series of recent studies by Schmidt and colleagues have shown that generating counterfactuals about the day's negative events at bedtime increases difficulty in falling asleep. This effect emerges even when individuals are randomly assigned to generate counterfactuals, suggesting a causal effect of these thoughts on insomnia, and when controlling for rumination and anxiety in correlational studies.

In short, excess counterfactual thought, particularly when not tailored to improving future outcomes, is associated with a range of emotional disturbances.

Counterfactuals and Coping

Just as increased upward counterfactuals are associated with emotional dysfunction and dysregulation, they also are associated with impaired coping with traumatic events. Given that counterfactuals are elicited by negative events (Roese, 1997) and have emotional consequences, counterfactual thoughts are particularly important in contexts of trauma. Pre-occupation with alternative outcomes (counterfactual thinking) is a central component of the ruminations of trauma victims. Increased counterfactual thinking has been linked to distress and anxiety for individuals who had lost a spouse or child in an automobile accident or a child to SIDs, been a victim of assault, or experienced a miscarriage.

One particularly influential aspect of counterfactuals is the tendency for trauma survivors to generate counterfactuals focused on how their own actions might have allowed them to avoid the event, fostering self-blame. Research by Branscombe and colleagues found that generating counterfactuals focused on how a victim of sexual assault might have avoided the incident increases victim blaming both by observers and victims. Among rape victims, those who generated more self-focused upward counterfactuals had lower well-being. Importantly, this relationship was mediated by self-blame: counterfactuals increased self-blame but not blame for the rapist or social factors, which led to deficits in coping. However, even in contexts in which self-blame is not focal, trauma is associated with increased upward counterfactual thought. Victims of terror attacks in Israel who were diagnosed with PTSD generated more upward counterfactuals, even though there was no particularly obvious self-blame for the incident (Gilbar, Plivazky, & Gil, 2010).

However, downward counterfactuals are more likely when individuals have a goal of regulating and improving affect following distressing events (Roese, 1997). Furthermore, downward counterfactuals may facilitate coping and psychological well-being by helping individuals to feel better about an outcome or by increasing meaning in life. Individuals who thought counterfactually about an important life event (e.g., how they might not have met a close friend) or how they might not have been born found these events or their life as a whole more meaningful than individuals who thought factually about the same event. In some cases, therefore, counterfactuals offer emotional benefits, particularly in the case of downward counterfactuals.

Deficits in Counterfactual Thought

Although excessive upward counterfactual thought is associated with emotional difficulties, other disorders show impoverished counterfactual thinking, which can cause difficulties with problem-focused cognition and reduced negative affect following negative outcomes.

In the mental health context, counterfactual impairment is best documented in schizophrenia. Research by Roese and colleagues shows that schizophrenia patients are less able to activate counterfactual thoughts relative to healthy control participants. When asked to generate counterfactual thoughts about a recent negative experience, schizophrenia patients described fewer counterfactuals compared with control participants. Additionally, research using the Counterfactual Inference Test (CIT), which presents scenarios that typically elicit a counterfactual response, found that schizophrenia patients had more difficulty drawing counterfactually driven inferences about social events compared with control participants. Importantly, diminished counterfactual thinking partially mediated the impaired social functioning experienced by the schizophrenia patients, thus highlighting the role that counterfactual thinking plays in regulating behavior and successfully negotiating the psychosocial world.

Furthermore, even when individuals with schizophrenia were provided with counterfactual thoughts, these counterfactuals did not strengthen relevant behavioral intentions. That is, after reading a negative event (e.g., missed a doctor's appointment) and the associated counterfactual (could have written it on my calendar), healthy control participants showed the expected pattern: a negative event paired with an upward counterfactual increased intentions to perform the behavior. In contrast, schizophrenia patients did not show an increase in the likelihood of performing that action in the future. Given that the deficits in counterfactual thinking did not appear to be the result of general cognitive impairment, researchers believe that impaired frontal lobe activity in particular may explain diminished counterfactual thinking in schizophrenia patients.

Diminished counterfactual thinking is also associated with Parkinson's disease (McNamara, Durso, Brown, & Lynch, 2003). Parkinson's patients generated fewer spontaneous counterfactuals in a thought-listing task and performed worse on the CIT compared with age-matched healthy control participants. Again, diminished counterfactual thinking significantly correlated with measures of frontal lobe functioning, indicating that an inability to generate counterfactuals might be related to frontal lobe dysfunction.

Together, this research suggests that frontal lobe impairment may partly explain why counterfactual thinking is diminished in schizophrenia and Parkinson's patients. More broadly, the possibility that counterfactual thinking is processed in the frontal lobe is supported both by neuropsychological theory and research on patients with brain lesions or damage (Camille et al., 2004; Gomez-Beldarrain, Garcia-Monco, Astigarraga, Gonzalez, & Grafman, 2005). In general, the frontal lobes are involved in social functioning, decision making, planning-related

cognitions, and the integration of emotion with decision making processes, functions also served by counterfactual thinking. Patients with prefrontal cortex lesions showed impaired spontaneous counterfactual thinking but were capable of cued counterfactual thinking and indirect counterfactual inferences. Thus, it appears that damage to the prefrontal cortex is particularly detrimental to self-generated counterfactual thoughts (Gomez-Beldarrain et al., 2005). Additional research has focused on the orbitofrontal cortex and its role in counterfactual thinking and the counterfactually induced emotion of regret, with studies showing that lesions in this area are associated with reductions in regret following a gambling task (Camille et al., 2004).

Accordingly, certain health problems may be associated with impairments in counterfactual thinking and related processes. Given that counterfactual thinking can be helpful in changing behavior, improving future outcomes, and increasing motivation and persistence, additional steps may be needed to elicit these functional aspects of counterfactual thinking that are not typically required in a healthy population.

Health Decisions and Health-Relevant Behaviors

Counterfactuals can hinder sound judgment, as they can alter evaluations in “irrational” ways (e.g., missing a flight by 5 or 50 min is objectively equivalent, but they feel different because of counterfactual thought). In addition to their emotional and mental health implications, counterfactuals can bias decision making such as blame and responsibility judgments during jury or medical treatment decisions. However, a more recent approach emphasizes the functional aspect of counterfactual thinking in regulating behavior via monitoring, managing, and coordinating behavior (Epstude & Roese, 2008; Roese, 1994; Roese & Epstude, 2017).

However, not all counterfactuals serve this preparative function. Upward counterfactuals that focus on taking action (e.g., if only I had gotten tested sooner) and counterfactuals following negative outcomes that involve future opportunities for action are most likely to be functional. Put simply, counterfactuals are most helpful when dealing with the actions one can take to address a problem that might occur again in the future (for a review, see Epstude & Roese, 2008; Roese & Epstude, 2017).

Upward counterfactuals facilitate changes in behavior in multiple ways. First, by providing insight and information about how a negative outcome might have been avoided, counterfactuals can strengthen intentions to change the behavior, increasing the likelihood of changing future behavior (Roese, 1994; Smallman & Roese, 2009). Second, counterfactuals that elicit negative affect can increase motivation and persistence on a task (Markman, McMullen, & Elizaga, 2008).

Counterfactuals and Behavior

According to the functional perspective, a primary function of counterfactual thought is problem solving. When individuals fail to reach a goal or encounter a problem, counterfactuals highlight behaviors that might correct those problems and facilitate goal achievement. For example, a person with a weight-loss goal might go grocery shopping while hungry, resulting in a cart full of junk food. Counterfactual thoughts such as “if only I had eaten before I went shopping” or “if only I had a shopping list” provide behaviors that could help avoid a recurrence of the situation and restart progress toward the weight-loss goal. In this way, counterfactuals help individuals evaluate a problem and offer new actions that will facilitate future improvement.

Originally, counterfactual-driven behavior change focused on academic (e.g., exam performance; Nasco & Marsh, 1999) and problem-solving (e.g., anagrams; Markman et al., 2008; Roese, 1994) behaviors. For example, after receiving exam grades, students who generated upward counterfactuals about their exam performance increased their grades on a subsequent exam (Nasco & Marsh, 1999). Upward counterfactuals increased students' feelings of control over the situation and made them more likely to perform the behaviors specified in the counterfactual, which in turn improved subsequent exam performance. Counterfactuals about anagram performance improved subsequent performance due to both the strategic thoughts (i.e., the useful tips to improve their anagram score) and also because of changes in motivation and subsequent effort (Roese, 1994; see also Markman et al., 2008).

Behavior change can occur for health-relevant behaviors as well. A series of studies examined whether counterfactual thinking could be an effective antismoking strategy (Page & Colby, 2003). In these studies, participants read a negative smoking scenario (describing a smoking-induced abnormal lung growth) then generated counterfactuals and rated affective responses about the scenario and indicated their willingness to participate in a smoking-related behavioral test (a lung capacity test) either immediately or after a 2- or 7-day delay. Although upward counterfactuals did not increase their willingness to sign up for the lung capacity test, additive counterfactuals did. Importantly, this increase occurred both immediately and after a 2- or 7-day delay, although the effect weakened over time. Although this finding differs from findings that upward counterfactuals increase behavior change, it supports the second tenet of the functional theory of counterfactual thinking, which predicts that additive counterfactuals should also increase behavior change.

Although so far we have focused on positive behavioral change, counterfactuals may also increase the likelihood of engaging in negative behaviors. Recently, Effron and colleagues have shown that counterfactuals that focus on the "sinful road not taken" can provide justification for unhealthy food choices. That is, considering less virtuous alternatives to one's past behavior (i.e., counterfactual sins) gives individuals the freedom to indulge (e.g., "I could have had a burger at lunch, but I didn't, so I can have ice cream tonight"). Additionally, since counterfactuals increase goal pursuit, there is no guarantee that they will increase behaviors related only to healthy goals; instead, they might help one pursue hedonic goals and increase the likelihood of enacting relevant unhealthy behaviors. For example, an individual who believes that his insistence on using a condom disrupted a previous sexual experience may think, "If only I hadn't stopped to talk about using a condom, things might have gone better," and thus be more likely to engage in unprotected sex in the future. Finally, even for healthy or adaptive goals, counterfactuals may help individuals identify unhealthy behaviors to achieve that goal. For instance, an individual with a healthy weight-loss goal might adopt a starvation "cleanse" diet, or a student hoping to improve class performance might abuse stimulants.

In short, counterfactuals can change future behavior. However, there are multiple pathways to behavior change. The first involves strengthening relevant behavioral intentions, which in turn increases the likelihood of performing the desired behavior in the future. The second involves increasing motivation and persistence toward future behavior.

Counterfactuals and Intentions

One way behavior change might occur is by strengthening intentions that are directly related to the behavioral outcome. That is, after a negative outcome (getting a particularly bad sunburn), a counterfactual thought ("if only I had worn sunscreen") might provide insight

regarding the usefulness of a particular action (wearing sunscreen), which prompts the relevant behavioral intention (“in the future I will wear sunscreen”), and consequently influences performance of that particular behavior (wearing sunscreen).

Upward counterfactuals strengthen relevant behavioral intentions in a variety of contexts. Students who generated upward counterfactuals about a recent negative academic performance (e.g., thinking about how they could have studied their notes after failing an exam) had higher intentions to perform success-facilitating behaviors in the future (e.g., saying they would be more likely to study their notes before the next exam; Roese, 1994). Similarly, pilots who generated upward and self-focused counterfactuals about their own near-accident flying incident had stronger intentions to perform behaviors that would have helped them avoid the incident (Morris & Moore, 2000).

Recent research by Smallman and colleagues (e.g., Smallman & Roese, 2009) using a reaction-time paradigm found that upward counterfactuals were more likely to activate and strengthen relevant behavioral intentions across a wide range of negative events, including academic, social, and health behaviors. However, it is important to note that not all counterfactuals are equally likely to strengthen relevant intentions. In particular, counterfactuals that are more detailed or include specific behaviors are more likely to strengthen intentions compared with counterfactuals that are more general or include traits. For example, after getting into a drunk-driving accident, the counterfactual “if only I had called a cab” is more likely to strengthen intentions compared with the counterfactual “if only I had acted more responsibly.”

Within health-relevant intentions, Epstude and Jonas (2015) examined the consequences of counterfactual thinking and regret in HIV-positive men. Results showed both harmful and helpful consequences. Counterfactuals and regret about their HIV status had a negative impact on well-being. Yet these counterfactuals also identified potential safe-sex behaviors, strengthening their safe-sex intentions for the future. This finding is particularly interesting because it focuses on an irreversible event (i.e., contracting the HIV virus). Typically, counterfactuals are most functional in the context of repeatable events, which allow for the application of the counterfactually driven insight to a future situation. These findings broaden that perspective, such that individuals might learn from non-repeatable events, as long as a future situation arises where the application of that knowledge is appropriate (e.g., future sexual encounters).

Similarly, research on binge drinking has found that counterfactual thinking can decrease intentions to binge drink (Baek, Shen, & Reid, 2013). In this study, participants generated counterfactuals in response to a binge drinking public service announcement. Although additive counterfactuals (actions that could have been taken) are typically more functional, this research demonstrated that subtractive counterfactuals (actions that should not have been taken) can also be functional depending on the message framing. That is, when researchers used a gain-framed appeal (beneficial outcomes of responsible drinking), additive counterfactuals lowered binge drinking intentions. But when a loss-framed appeal (harmful outcomes of irresponsible drinking) was used, subtractive counterfactuals lowered binge drinking intentions. Accordingly, message framing may be an additional factor to consider when trying to elicit functional counterfactual effects.

These findings describe one possible mechanism, the strengthening of behavioral intentions, through which counterfactual thinking can facilitate behavior change. Given the importance of intentions in other behavior-prediction theories (e.g., theory of planned behavior) and their prevalence in studying health-relevant behaviors, counterfactuals might serve as a cognitive tool to activate or strengthen a relevant behavioral intention.

Counterfactuals and Motivation

A second way that counterfactual thinking can influence performance is by increasing motivation and persistence. The negative affect produced by a counterfactual can increase motivation and persistence toward satisfying achievement goals. Unlike changes in intentions, motivational influences may result from both upward and downward counterfactuals (Markman & McMullen, 2003; Markman et al., 2008). First, increased motivation may occur when upward counterfactuals are considered in an evaluative manner, in which an individual contrasts the better alternative to the lesser reality. For example, a student who gets a C on an exam might feel bad after comparing it to the A he could have gotten, which motivates him to work harder. Additionally, increased motivation may occur when downward counterfactuals are considered in a reflective manner, in which an individual focuses mainly on the worse alternative (imagining it is true), without comparing it to the better reality. For example, a student who just barely gets a C on an exam might focus solely on how she nearly failed the test, motivating her to work harder.

Mechanisms that increase motivation and persistence toward goal attainment should be particularly relevant in the health domain. While health practitioners can provide information and tools for improving health outcomes, patients must have the motivation and persistence to work toward these goals. For example, researchers examined whether counterfactual thinking can increase a person's motivation to read and process food label information (Aboulnasr & Sivaraman, 2010). Results showed that counterfactual thinking increased participants' motivation to process this information systematically. Participants who generated counterfactuals examined both health claims and nutrition information more carefully and were more likely to use this information when evaluating the products. Importantly, these motivational effects may last up to seven days after counterfactual generation, indicating that the counterfactual-driven motivational boost may extend beyond the immediate time frame.

Importantly, these two pathways (strengthening relevant intentions and increasing motivation) may work together to change behavior and increase the likelihood of reaching one's goals. For example, a negative event (e.g., driving while intoxicated) might prompt counterfactual thoughts (e.g., "if only I had called a cab"). These thoughts provide useful insights about how an outcome might have been avoided, which can strengthen intentions to enact these behaviors in a future similar situation. However, these thoughts may also heighten negative affect (i.e., the person feels even worse about the situation), which in turn increases motivation and persistence related to these behaviors. Like a steering wheel and a motor, intentions and motivation may work together to change behavior and facilitate goal attainment.

Conclusion

Counterfactual thoughts, identifying alternatives to reality, play a wide and varied role in health. Both excessive counterfactual thought and deficits in counterfactual thought are associated with impaired mental health, with the former being associated with emotional dysfunction and the latter with frontal lobe damage characteristic of schizophrenia and Parkinson's disease. However, counterfactuals can also be beneficial: downward counterfactuals about worse outcomes can promote well-being and meaning in life, and upward counterfactuals can facilitate healthy behavior by facilitating the formation of behavioral intentions and increasing motivation. Whether counterfactuals are beneficial or harmful to health depends on a number

of factors, including the structure of the counterfactual thought, the specific behaviors identified, and potentially the broader context of these thoughts, such as whether a particular outcome is repeatable or final. Given their importance to health and their frequency in daily life, counterfactuals thus require thoughtful and nuanced consideration in health psychology.

Author Biographies

Rachel Smallman is an assistant professor of social psychology at Texas A&M University. Her research focuses on social cognition, with specific interests in counterfactual thinking and categorization. In particular, she is interested in how these thoughts of “what might have been” can both help and hinder future decisions, behavioral intentions, and future behavior. Additionally, she studies the relationship between counterfactual thinking and health-related outcomes.

Amy Summerville is an associate professor in the Department of Psychology at Miami University. Her work focuses on counterfactual thought and the experience of regret, particularly the origins of regret and the factors that determine its change over time. Her recent work has examined effects of the social context on counterfactuals and regret, including motivations for the expression of regret.

Ryan J. Walker is a doctoral student in the Department of Psychology at Miami University. His work focuses on counterfactual thought, with a particular focus on how these thoughts impact subsequent behavioral intentions and emotions. In addition, he is generally interested in individual differences and how they influence decisions.

Brittney Becker has her PhD from Texas A&M University in the area of social and personality psychology. Her primary research interests include counterfactual thinking, preference categorization, evaluative categorization, and how these processes affect judgment and decision making.

References

- Aboulnasr, K., & Sivaraman, A. (2010). Food for thought: The effect of counterfactual thinking on the use of nutrition information. *Journal of Consumer Behaviour*, 9, 191–205. doi:10.1002/cb.311
- Baek, T. H., Shen, L., & Reid, L. N. (2013). Effects of message framing in anti-binge drinking PSAs: The moderating role of counterfactual thinking. *Journal of Health Communication*, 18, 442–458. doi:10.1080/10810730.2012.743621
- Camille, N., Coricelli, G., Sallet, J., Pradat-Diehl, P., Duhamel, J. R., & Sirigu, A. (2004). The involvement of the orbitofrontal cortex in the experience of regret. *Science*, 304, 1167–1170. doi:10.1126/science.1094550
- Epstude, K., & Jonas, K. J. (2015). Regret and counterfactual thinking in the face of inevitability: The case of HIV-positive men. *Social Psychological and Personality Science*, 6, 157–163. doi:10.1177/1948550614546048
- Epstude, K., & Roese, N. J. (2008). The functional theory of counterfactual thinking. *Personality and Social Psychology Review*, 12, 168–192. doi:10.1177/1088868308316091
- Gilbar, O., Plivazky, N., & Gil, S. (2010). Counterfactual thinking, coping strategies, and coping resources as predictors of PTSD diagnosed in physically injured victims of terror attacks. *Journal of Loss and Trauma*, 15, 304–324. doi:10.1080/15325020903382350
- Gilovich, T., & Medvec, V. H. (1995). The experience of regret: What, when, and why. *Psychological Review*, 102, 379–395. doi:10.1037/0033-295X.102.2.379

- Gomez-Beldarrain, M., Garcia-Monco, J. C., Astigarraga, E., Gonzalez, A., & Grafman, J. (2005). Only spontaneous counterfactual thinking is impaired in patients with prefrontal cortex lesions. *Cognitive Brain Research*, 24, 723–726. doi:10.1016/j.cogbrainres.2005.03.013
- Kahneman, D., & Miller, D. T. (1986). Norm theory: Comparing reality to its alternatives. *Psychological Review*, 93, 136–153. doi:10.1037/0033-295X.93.2.136
- Markman, K. D., & McMullen, M. N. (2003). A reflection and evaluation model of comparative thinking. *Personality and Social Psychology Review*, 7, 244–267. doi:10.1207/S15327957PSPR0703_04
- Markman, K. D., McMullen, M. N., & Elizaga, R. A. (2008). Counterfactual thinking, persistence, and performance: A test of the reflection and evaluation model. *Journal of Experimental Social Psychology*, 44, 421–428. doi:10.1016/j.jesp.2007.01.001
- McNamara, P., Durso, R., Brown, A., & Lynch, A. (2003). Counterfactual cognitive deficit in persons with Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, 74, 1065–1070. doi:10.1136/jnnp.74.8.1065
- Morris, M. W., & Moore, P. C. (2000). The lessons we (don't) learn: Counterfactual thinking and organizational accountability after a close call. *Administrative Science Quarterly*, 45, 737–765. doi:10.2307/2667018
- Nasco, S. A., & Marsh, K. L. (1999). Gaining control through counterfactual thinking. *Personality and Social Psychology Bulletin*, 25, 556–568. doi:10.1177/0146167299025005002
- Page, C. M., & Colby, P. M. (2003). If only I hadn't smoked: The impact of counterfactual thinking on a smoking-related behavior. *Psychology & Marketing*, 20, 955–976. doi:10.1002/mar.10104
- Roose, N. J. (1994). The functional basis of counterfactual thinking. *Journal of Personality and Social Psychology*, 66, 805–818. doi:10.1037/0022-3514.66.5.805
- Roose, N. J. (1997). Counterfactual thinking. *Psychological Bulletin*, 121(1), 133–148. doi:10.1037/0033-2909.121.1.133
- Roose, N. J., & Epstude, K. (2017). The functional theory of counterfactual thinking: New evidence, new challenges, new insights. *Advances in Experimental Social Psychology*. doi:10.1016/bs.aesp.2017.02.001
- Roose, N. J., Epstude, K., Fessel, F., Morrison, M., Smallman, R., Summerville, A., ... Segerstrom, S. (2009). Repetitive regret, depression, and anxiety: Findings from a nationally representative survey. *Journal of Social and Clinical Psychology*, 28, 671–688. doi:10.1521/jscp.2009.28.6.671
- Smallman, R., & Roose, N. J. (2009). Counterfactual thinking facilitates behavioral intentions. *Journal of Experimental Social Psychology*, 45, 845–852. doi:10.1016/j.jesp.2009.03.002

Suggested Reading

- Camille, N., Coricelli, G., Sallet, J., Pradat-Diehl, P., Duhamel, J. R., & Sirigu, A. (2004). The involvement of the orbitofrontal cortex in the experience of regret. *Science*, 304, 1167–1170. doi:10.1126/science.1094550
- El Leithy, S., Brown, G. P., & Robbins, I. (2006). Counterfactual thinking and posttraumatic stress reactions. *Journal of Abnormal Psychology*, 115, 629–635. doi:10.1037/0021-843X.115.3.629
- Epstude, K., & Roose, N. J. (2008). The functional theory of counterfactual thinking. *Personality and Social Psychology Review*, 12, 168–192. doi:10.1177/1088868308316091
- Markman, K. D., McMullen, M. N., & Elizaga, R. A. (2008). Counterfactual thinking, persistence, and performance: A test of the reflection and evaluation model. *Journal of Experimental Social Psychology*, 44, 421–428. doi:10.1016/j.jesp.2007.01.001
- Roose, N. J., & Epstude, K. (2017). The Functional theory of counterfactual thinking: New evidence, new challenges, new insights. *Advances in Experimental Social Psychology*. doi:10.1016/bs.aesp.2017.02.001

Couple-Relationships and Cancer Adaptation

Hoda Badr and Zeba Ahmad

Icahn School of Medicine at Mount Sinai, New York, NY, USA

The past three decades have seen major increases in cancer survival through improvements in early detection and treatment. There are now over 14.5 million individuals alive today with a history of cancer, and this segment of the population continues to grow rapidly (Office of Cancer Survivorship, 2015). Today, two-thirds of cancer patients can expect to live 5 years or more after diagnosis. It is therefore important to begin to think about their quality of life (QOL) during and after cancer, starting from the point of diagnosis (Office of Cancer Survivorship, 2015).

QOL is a multidimensional construct encompassing physical, psychological, and social well-being. In cancer, social relationships are the most studied factor contributing to QOL (see Manne & Badr, 2008). For survivors who are married or in a committed relationship, their relationship with their partner (spouse or significant other) is often their primary coping resource, and studies have shown that survivors frequently rely on their partners to provide caregiving and emotional support (e.g., Keefe et al., 2005).

In the United States, approximately 4.6 million people care for a partner or family member with cancer at home (National Alliance for Caregiving, 2009). As the primary setting for care has shifted from hospital to home, family members have found themselves replacing skilled healthcare workers in the delivery of care. In addition to undertaking complex care tasks and providing emotional support, partners must cope with the grief and loss associated with their loved one's life-threatening illness. Most assume their role with little or no formal preparation and training and have low self-efficacy for caregiving. This is problematic because lower levels of caregiver self-efficacy are associated with increased psychological distress (Groß, Dahl, Moum, & Fosså, 2005). Caregivers also frequently encounter communication difficulties with patients, which can adversely affect the coordination of care as well as the quality of their relationship and QOL. Given the above, this chapter will review the QOL challenges facing cancer patients and their partners, discuss the impact of cancer on couple relationships, and posit directions for future research.

QOL Challenges Faced by Cancer Patients and Their Partners

Physical Well-Being

Although the goal of most cancer treatments is to improve or maintain physical functioning and overall QOL, patients often experience physical symptoms that persist long after treatment ends. Cancer patients have reported an average of 11–13 concurrent symptoms including pain, fatigue, labored breathing, nausea, shortness of breath, lack of appetite, loss of body mass, dry mouth, and difficulty sleeping (Portenoy et al., 1994). These symptoms may be a result of the disease or treatment and can adversely affect health-related QOL (Portenoy et al., 1994).

The physical and emotional toll of caring for a partner with cancer may also negatively impact the caregiver. In a study examining sick leave usage, partners of lung cancer patients had the most sick leave episodes, which may be attributed to higher physical and emotional burdens of care (Sjövall et al., 2010). As cancer progresses, added caregiver stress may result in sleep disturbance, fatigue, and unhealthy behaviors. Studies have shown that caregivers' health behaviors may also be negatively impacted following a loved one's cancer diagnosis (Beesley et al., 2011). One reason for this is may be that caregivers prioritize the care of the patient over their own self-care.

Psychological Well-Being

An estimated 35% of people diagnosed with cancer experience clinically significant levels of psychological distress (Halford, Chambers, & Clutton, 2010). Risk for depression is higher in this population than in the general population. Being diagnosed at a younger age and being newly diagnosed are associated with elevated risk for distress (Hagedoorn, Sanderman, Bolks, Tuinstra, & Coyne, 2008).

The psychological distress associated with cancer has negative health and behavioral consequences, including nonadherence to medical regimens, increased emergency and inpatient service use, delayed return to work, and possibly elevated rates of suicidal ideation or suicide and mortality (Fang et al., 2012). A supportive partner can help to buffer the adverse impact of cancer on patient distress and QOL (Kayser & Scott, 2008). For example, in a study of women with breast cancer, vulnerability to anxiety was moderated by the degree of felt support from a partner (Borstelmann et al., 2015). In a study of head and neck cancer patients, partners who used positive social influence tactics were effective in boosting patient positive mood and encouraging patient self-care (Badr, Yeung, Lewis, Milbury, & Redd, 2015).

Despite the benefits of having a partner, research has shown that partners are often as distressed as patients and distressed partners may find it difficult to provide adequate support to the patient (Milbury, Badr, & Carmack, 2012). Anxiety and depression are the most commonly reported problems, with estimates for depression at 39% (Braun, Mikulincer, Rydall, Walsh, & Rodin, 2007) and for anxiety at 40% (Janda et al., 2007). Very little is known about the factors driving partner distress; however, the patient's functional decline and increasing distress coupled with the increased need for the partner to assist with symptom management and provide support are likely contributing factors. Partner anxiety levels, depression, and stress have all been shown to increase as the patient's functional status declines and the patient approaches death (Grover et al., 2005). Caregiver depression also appears to be sensitive to sleep deprivation; declines in the caregiver's physical health; the perceived burden of caregiving; changes in roles, responsibilities, and leisure activities; and social isolation. Two meta-analyses examined the relationship between the psychological distress levels of patients with cancer and

their spousal caregivers and found their responses to cancer to be interdependent: each person affected the other's level of emotional well-being (Hagedoorn, Buunk, Kuijer, Wobbles, & Sanderman, 2000).

Social and Relationship Well-Being

At times, people with cancer may experience difficulties relating to others. Despite this, research has shown the protective value of social relationships and social support on patients' cancer-related stress (Helgeson & Cohen, 1996). Social support is also important for partners and accounts for a significant amount of variance in their QOL. Real or perceived lack of support from social network and family members is associated with high levels of physical and psychological exhaustion, and many caregivers report that their social support decreases over time (Fineberg & Bauer, 2011). Worsening illness can burden caregivers, who become fatigued and lack the time to continue normal social activities. This loss of a social network increases a sense of isolation and is associated with worse psychological well-being.

Despite the importance of network support, patients report that their spouses or partners are the first persons from whom they seek support after a diagnosis. Research on cancer patients suggests that support from family members and friends does not compensate for a lack of partner support (Manne, Alfieri, Taylor, & Dougherty, 1999). Although partners can be supportive, they can also engage in unsupportive, negative behaviors that include minimizing the problem, forced cheerfulness, and insensitive comments. In a qualitative study, partners' denial and avoidance of cancer-related discussions was distressing to patients, made them change how they interacted with their partners, and strained the marital relationship (Badr & Carmack Taylor, 2006). In another study, patients and partners were videotaped as they discussed cancer-related concerns, and researchers coded the exchanges for socially supportive and unsupportive behaviors (Manne et al., 2004). When patients made emotional disclosures and spouses responded in an unsupportive fashion, patients reported greater distress and lower levels of marital satisfaction.

Poor marital satisfaction is associated with greater psychological morbidity among cancer patients. In contrast, patients who report greater marital satisfaction and better marital functioning report lower levels of psychological morbidity (see Kayser, 2005). In lung cancer, Carmack Taylor et al. (2008) found that a cycle of distress develops whereby one partner's distress exacerbates the other partner's distress and that this association is weaker when patients and partners report higher levels of relationship functioning. One explanation is that good relationships promote positive supportive dyadic coping whereby one person recognizes the other's distress and assists him/her in coping. Another is that people in good relationships are more likely to act in the best interest of their partner and their relationship. In this case, patients/caregivers may believe that managing their own distress is in the best interest of their loved one. Indeed, in surveying 158 patients and their spousal caregivers, Badr and colleagues found that even though 34.6% of patients and 36.4% of the caregivers in their sample were distressed, *both* were distressed in only 17.6% dyads (Carmack Taylor et al., 2008). Thus, for about half of the distressed patients, their caregiver was not distressed, and for half of the distressed caregivers, the patient was not distressed. This is not to say that having a loved one who is distressed is not distressing for patients and caregivers with good relationships; rather, they may be better than those with poor relationships at managing their own distress so it does not reach a clinical level.

Cancer treatment can challenge established patterns of communication, control, and responsibility in a relationship. Some patients report greater conflict, decreased intimacy, and strained

communication after diagnosis, whereas others report that the illness brought them closer together (Dorval et al., 2005). Each juncture of the cancer treatment process brings challenges to patients and partners' communication and joint coping. In the weeks following diagnosis, these include negotiating changes in roles and responsibilities, dealing with interference with life plans, and managing household and childcare responsibilities. Concerns about the impact of cancer treatment or the desire to undergo fertility preservation procedures can cause delays in cancer treatment. During treatment, declines in physical functioning may interfere with household responsibilities, and integrating cancer into the everyday routines of a couple can be demanding (Kayser & Scott, 2008). Increased caregiving responsibilities can also alter sexual expression and increase tension between partners. A survey study of men with prostate cancer and their wives found high rates of sexual dysfunction associated with cancer treatment. Greater sexual dissatisfaction was associated with poorer marital adjustment, particularly in the absence of constructive communication (Badr & Carmack Taylor, 2009).

Declining functional abilities may bring about the necessity for couples to discuss how care and assistance with daily activities will be provided to the ill partner. Couples also typically negotiate difficult choices regarding end-of-life treatments and care and cope with anticipatory grief (see McLean & Jones, 2007). For couples who enter the posttreatment survivorship phase, the primary challenge is negotiating the transition to "normal" life, including partners' respective pace preferences. For most, the main tasks include resuming a sexual relationship, discussing changes in life plans, discussing health behavior changes, dealing with disease and treatment-related late effects that may influence the patient's functioning, managing worry about disease recurrence, and discussing the meaning of the cancer experience for themselves and the relationship.

Research has consistently shown that open, supportive communication is vital to maintaining positive relationship experiences while coping with the side effects of treatment and progressive illness. From a couples' perspective, successful adaptation may be less dependent upon the circumstances of the illness than on how well the couple integrates cancer into their lives.

Effects of Couple-Based Interventions on Patient and Partner QOL Outcomes

Couple-based interventions acknowledge that both members of the couple and their relationship are profoundly affected by the cancer experience (see Hagedoorn et al., 2008; Manne & Badr, 2008). Badr and Krebs (2013) conducted a systematic review and meta-analysis of randomized controlled trials of interventions that aimed to improve the physical, psychological, and social well-being of cancer patients and their partners. Their systematic review featured 23 qualifying studies and a total of 2,645 participants. Studies had an average sample size of 115 participants each ($s = 77$). The average age of participants was around 55 years, and approximately 16% of study participants were non-White. Forty-seven percent of eligible patients declined to participate in the studies, and 25% of participants withdrew from the studies. The majority (60%) of these studies focused on either breast or prostate cancer. Meta-analysis of study outcomes found small but beneficial effects of the interventions on physical, psychological, and relationship outcomes for both patients and partners. Weighted mean effect sizes and 95% confidence intervals are summarized in Table 1.

Topics of intervention sessions varied with the theoretical model being used. Methods included techniques from cognitive behavioral therapy, psychoeducation, emotion-focused therapy, and

Table 1 Weighted mean effect sizes (g) and 95% confidence intervals of couple-based interventions on quality of life.

	<i>Physical</i>	<i>k</i>	<i>Psychological</i>	<i>k</i>	<i>Relationship</i>	<i>k</i>
Patient	0.31 {0.11–0.50}	12	0.25 {0.12–0.32}	17	0.28 {0.14–0.43}	10
Partner			0.21 {0.08–0.34}	12	0.24 {0.06–0.43}	7

k describes the number of qualifying studies included in each analysis.

Source: Adapted from Badr and Krebs (2013).

behavioral marital therapy. “Partner-assisted” interventions conceptualized the partner as a facilitator of skill learning for the patient. The other interventions more actively involved both partners, treating the couple as one unit. Most interventions were composed of six sessions. Most sessions were conducted in person, although some were conducted over the phone.

Badr and Krebs’ (2013) review highlighted a number of methodological limitations in the couples’ intervention literature. Often, researchers did not articulate a theoretical model. Few examined the mechanisms by which interventions affected outcomes, so there are questions as to whether the theoretical basis of the intervention was as hypothesized. Most studies had small sample sizes and thus were underpowered to examine changes in the multiple outcomes that were measured. Some did not include information on refusal or attrition rates, suggesting reporting standards could improve. One-quarter of the studies that were reviewed did not report outcomes for partners. A lack of patient improvement could be explained by negative but unexamined effects on the partner or the possibility that patients and partners respond to intervention at different rates. Thus, future studies should include both partners and ensure that the couple is the unit of analysis throughout the research process. Another limitation is that none of the studies evaluated the cost-effectiveness of couple-based interventions relative to interventions targeting the patient or caregiver alone. Studies evaluating the relative cost of different modes of administration (i.e., in person, over the phone, internet) are needed.

Future Directions

As our review suggests, significant empirical attention has been paid to the challenges facing cancer patients and their partners that impact their relationships and QOL. There are also a number of unexamined issues. Understanding why some patients do not get the support they desire or expect and why certain couples are at risk for poor psychosocial outcomes will help clarify the role of couple relationships in adaptation to cancer. We also still do not fully understand the mechanisms by which social relationships are beneficial for patient and partner psychosocial adaptation and QOL.

Researchers should recognize that relationships and relationship roles change over time. Most studies have evaluated outcomes shortly after diagnosis. Indeed, many couples report that cancer brought them closer together, but it remains unclear what happens over time as couples learn to deal with the long-term side effects of cancer. Greater attention to sociodemographic variables is also needed. Because the majority of couple-based studies have focused on single-gender cancers such as breast or prostate, it is difficult to disentangle gender and role (i.e., whether the individual is a patient or partner) effects. Thus, more studies should include both members of the couple and focus on cancers that affect men and women. In addition, couples at different stages of the life cycle or cancer trajectory (i.e., early-stage versus end-of-

life versus the posttreatment survivorship phase) may experience different physical, psychological, and relationship stressors and therefore have different expectations regarding not only social support but also interaction with their partners. To date, very few studies have investigated the cancer experience of minority couples, despite the fact that a recent comparative analysis showed that cultural differences do exist—particularly around the dimensions of family boundaries, gender roles, conception of personal control, and interdependence (Kayser et al., 2014). Individuals with low socioeconomic status and those in same-sex relationships have also been underrepresented in this literature.

Pre-illness relationship factors may affect couples' motivations to use cancer as an opportunity to enhance their relationship. Individual factors such as attachment style and interpersonal skills may also influence relationship processes and adjustment. Few measures of cancer-related communication exist, and those that do involve self-reports that assess the quantity rather than the quality or nature of discussions. Finally, relationship quality may be an important moderator or proxy for social support. Individuals who are more satisfied in their relationships may perceive greater support, may be more likely to explain away partners' unsupportive behaviors, and may benefit more from the support that they do receive.

Future intervention studies should target lifestyle behavioral changes, since partners can engage in unhealthy behaviors (e.g., smoking) that can interfere with patient adherence to medical recommendations after a cancer diagnosis. Partners who model healthy behaviors for one another, acknowledge each other's successes, provide constructive feedback, and work together to overcome barriers may benefit in terms of increased self-efficacy and lasting behavioral change.

Although couple-based interventions have had many positive effects, they are seldom implemented in clinical practice setting. Several barriers need to be addressed in order for this to happen, including increasing the awareness of caregivers' and couples' issues in practice settings, training healthcare professionals on how to deal with caregiver and couples' issues, and demonstrating the impact of these interventions on healthcare utilization, patient satisfaction, and cost savings. Supporting partners and couples can benefit the patient's recovery and clinical outcomes. Improved patient outcomes, such as reduced postoperative complications resulting from early identification of problems, better management of adverse effects, increased adherence to oral medication, and early detection of adverse events, would result in reduced healthcare use and costs.

In conclusion, couple relationships exert a strong influence on both patients' and partners' psychosocial adaptation and QOL. Future research may benefit from an increased focus on couples' interactions to address ways that couples can adaptively cope together. In addition, there is a growing need for healthcare providers to view the couple as the unit of care and provide more information and support to partners so they can provide effective caregiving for the patient while also maintaining their own health and well-being.

Author Biographies

Hoda Badr is a health psychologist and associate professor in the Department of Oncological Sciences at the Icahn School of Medicine at Mount Sinai. Her research program focuses on developing and implementing psychosocial interventions that leverage family support to improve patient and caregiver health behaviors and well-being across the cancer control continuum.

Zeba Ahmad is an associate researcher studying psychosocial interventions with Dr. Badr at Mount Sinai.

References

- Badr, H., & Carmack Taylor, C. (2006). Social constraints and spousal communication in lung cancer. *Psycho-Oncology*, 15(8), 673–683. doi:10.1002/pon.996
- Badr, H., & Carmack Taylor, C. L. (2009). Sexual dysfunction and spousal communication in couples coping with prostate cancer. *Psycho-Oncology*, 18(7), 735–746. doi:10.1002/pon.1449
- Badr, H., & Krebs, P. (2013). A systematic review and meta-analysis of psychosocial interventions for couples coping with cancer. *Psycho-Oncology*, 22, 1688–1704. doi:10.002/pon.3200
- Badr, H., Yeung, C., Lewis, M. A., Milbury, K., & Redd, W. H. (2015). An observational study of social control, mood, and self-efficacy in couples during treatment for head and neck cancer. *Psychology & Health*, 30(7), 783–802. doi:10.1080/08870446.2014.994633
- Beesley, V. L., Price, M. A., Webb, P. M., & Australian Ovarian Cancer Study Group; Australian Ovarian Cancer Study—Quality of Life Study Investigators. (2011). Loss of lifestyle: Health behaviour and weight changes after becoming a caregiver of a family member diagnosed with ovarian cancer. *Supportive Care in Cancer*, 19(12), 1949–1956. doi:10.1007/s00520-010-1035-2
- Borstelmann, N. A., Rosenberg, S. M., Ruddy, K. J., Tamimi, R. M., Gelber, S., Schapira, L., ... Partridge, A. H. (2015). Partner support and anxiety in young women with breast cancer. *Psycho-Oncology*. doi:10.1002/pon.3780
- Braun, M., Mikulincer, M., Rydall, A., Walsh, A., & Rodin, G. (2007). Hidden morbidity in cancer: Spouse caregivers. *Journal of Clinical Oncology*, 25(30), 4829–4834. doi:10.1200/JCO.2006.10.0909
- Carmack Taylor, C. L., Badr, H., Lee, L., Pisters, K., Fossella, F., Gritz, E. R., & Schover, L. (2008). Lung cancer patients and their spouses: Psychological and relationship functioning within 1 month of treatment initiation. *Annals of Behavioral Medicine*, 36(2), 129–140. doi:10.1007/s12160-008-9062-7
- Dorval, M., Guay, S., Mondor, M., Masse, B., Falardeau, M., Robidoux, A., ... Maunsell, E. (2005). Couples who get closer after breast cancer: Frequency and predictors in a prospective investigation. *Journal of Clinical Oncology*, 23(15), 3588–3596. doi:10.1200/JCO.2005.01.628
- Fang, F., Fall, K., Mittleman, M. A., Sparén, P., Ye, W., Adami, H.-O., & Valdimarsdóttir, U. (2012). Suicide and cardiovascular death after a cancer diagnosis. *New England Journal of Medicine*, 366(14), 1310–1318. doi:10.1056/NEJMoal110307
- Fineberg, I. C., & Bauer, A. (2011). Families and family conferencing. In T. Altilio & S. A. Otis-Green (Eds.), *Textbook of palliative social work* (pp. 235–250). New York, NY: Oxford University Press. ISBN: 978-0-19-973911-0
- Grov, E., Dahl, A., Moum, T., & Fosså, S. (2005). Anxiety, depression, and quality of life in caregivers of patients with cancer in late palliative phase. *Annals of Oncology*, 16(7), 1185–1191. doi:10.1093/annonc/mdi210
- Hagedoorn, M., Buunk, B. P., Kuijer, R. G., Wobbles, T., & Sanderman, R. (2000). Couples dealing with cancer: Role and gender differences regarding psychological distress and quality of life. *Psycho-Oncology*, 9, 232–242. doi:10.1002/1099-1611(200005/06)9:3<232::AID-PON458>3.0.CO;2-J
- Hagedoorn, M., Sanderman, R., Bolks, H., Tuinstra, J., & Coyne, J. C. (2008). Distress in couples coping with cancer: A meta-analysis and critical review of role and gender effects. *Psychological Bulletin*, 134(1), 1–30. doi:10.1037/0033-2909.134.1.1
- Halford, W. K., Chambers, S., & Clutton, S. (2010). Couples coping with cancer. In N. A. Pachana, K. Laidlaw, & B. G. Knight (Eds.), *Casebook in clinical geropsychology: International perspectives on practice* (pp. 73–90). Oxford: Oxford University Press. ISBN: 978-0-19-958355-3
- Helgeson, V., & Cohen, S. (1996). Social support and adjustment to cancer: Reconciling descriptive, correlational, and intervention research. *Health Psychology*, 15, 135–148. doi:10.1037/0278-6133.15.2.135
- Janda, M., Steginga, S., Langbecker, D., Dunn, J., Walker, D., & Eakin, E. (2007). Quality of life among patients with a brain tumor and their carers. *Journal of Psychosomatic Research*, 63(6), 617–623. doi:10.1016/j.jpsychores.2007.06.018

- Kayser, K. (2005). Enhancing dyadic coping during a time of crisis: A theory-based intervention with breast cancer patients and their partners. In T. A. Revenson, K. Kayser, & G. Bodenmann (Eds.), *The psychology of couples and illness* (pp. 175–191). Washington, DC: American Psychological Association. ISBN: 978-1-59147-204-9
- Kayser, K., Cheung, P. K., Rao, N., Chan, Y. C. L., Chan, Y., & Lo, P. H. (2014). The influence of culture on couples coping with breast cancer: A comparative analysis of couples from China, India, and the United States. *Journal of Psychosocial Oncology*, 32(3), 264–288. doi:10.1080/07347332.2014.897292
- Kayser, K., & Scott, J. (2008). *Helping couples cope with women's cancers: An evidence-based approach for practitioners*. New York, NY: Springer Science + Business Media. ISBN: 978-0-387-74802-3
- Keefe, F. J., Ahles, T. A., Sutton, L., Dalton, J., Baucom, D., Pope, M. S., ... Syrjala, K. (2005). Partner-guided cancer pain management at the end of life: A preliminary study. *Journal of Pain and Symptom Management*, 29(3), 263–272. doi:10.1016/j.jpainsymman.2004.06.014
- Manne, S., Alfieri, T., Taylor, K., & Dougherty, J. (1999). Preferences for spousal support among individuals with cancer. *Journal of Applied Social Psychology*, 29(4), 722–749. doi:10.1111/j.1559-1816.1999.tb02021.x
- Manne, S., & Badr, H. (2008). Intimacy and relationship processes in couples' psychosocial adaptation to cancer. *Cancer*, 112(11 Suppl), 2541–2555. doi:10.1002/cncr.23450
- Manne, S., Ostroff, J., Sherman, M., Heyman, R. E., Ross, S., & Fox, K. (2004). Couples' support-related communication, psychological distress and relationship satisfaction among women with early stage breast cancer. *Journal of Consulting and Clinical Psychology*, 72(4), 660–670. doi:10.1037/0022-006X.72.4.660
- McLean, L. M., & Jones, J. M. (2007). A review of distress and its management in couples facing end-of-life cancer. *Psycho-Oncology*, 16(7), 603–616. doi:10.1002/pon.1196
- Milbury, K., Badr, H., & Carmack, C. (2012). The role of blame in the psychosocial adjustment of couples coping with lung cancer. *Annals of Behavioral Medicine*, 44, 331–340. doi:10.1007/s12160-012-9402-5
- National Alliance for Caregiving. (2009). Caregiving in the U.S. Retrieved from http://www.caregiving.org/data/Caregiving_in_the_US_2009_full_report.pdf
- Office of Cancer Survivorship. (2015). Retrieved from <http://cancercontrol.cancer.gov/ocs/prevalence/index.html>
- Portenoy, R., Thaler, H., Kornblith, A. B., Lepore, J. M., Friedlander-Klar, H., Coyle, N., ... Hoskins, W. (1994). Symptom prevalence, characteristics and distress in a cancer population. *Quality of Life Research*, 3, 183–189. doi:10.1007/BF00435383
- Sjövall, K., Attner, B., Lithman, T., Noreen, D., Gunnars, B., Thomé, B., ... Englund, M. (2010). Sick leave of spouses to cancer patients before and after diagnosis. *Acta Oncologica*, 49(4), 467–473. doi:10.3109/02841861003652566

Suggested Reading

- Kayser, K. (2005). Enhancing dyadic coping during a time of crisis: A theory-based intervention with breast cancer patients and their partners. In T. A. Revenson, K. Kayser, & G. Bodenmann (Eds.), *The Psychology of Couples and Illness* (pp. 175–191). Washington, DC: American Psychological Association. ISBN: 978-1-59147-204-9
- Manne, S., & Badr, H. (2008). Intimacy and relationship processes in couples' psychosocial adaptation to cancer. *Cancer*, 112(11 Suppl), 2541–2555. doi:10.1002/cncr.23450
- McLean, L. M., & Jones, J. M. (2007). A review of distress and its management in couples facing end-of-life cancer. *Psycho-Oncology*, 16(7), 603–616. doi:10.1002/pon.1196
- Badr, H., & Krebs, P. (2013). A systematic review and meta-analysis of psychosocial interventions for couples coping with cancer. *Psycho-Oncology*, 22, 1688–1704. doi:10.002/pon.3200

Direct-to-Consumer Testing

Angela M. Legg and May Reinert

Department of Psychology, Pace University, New York, NY, USA

Soon after the sequencing of the human genome, healthcare professionals could order genomic profiling for their patients. These genetic tests provided predictive and diagnostic information about patients' health risks and carrier status for illnesses ranging from breast cancer to Huntington's disease. Interested patients needed to consult with a physician or genetic counselor prior to receiving testing. These healthcare professionals then initiated an order for the patient to take the genetic test, typically for only one disease. To improve patients' understanding of their genetic risks, some physicians also required their patients to attend genetic counseling or additional consultations upon receipt of their results. However, the landscape of genetic testing dramatically changed in 2006 with the creation of direct-to-consumer (DTC) genetic testing services such as 23andMe. Interested patients no longer needed to receive physician or counselor approval before getting a genetic test. Patients also were not required to seek consultations or counseling sessions after receiving their results (although they were provided with this option). Completing a genetic test was as simple as collecting a cheek swab, shipping the kit, and awaiting the results. People could receive genetic risk information on a host of conditions ranging from cystic fibrosis, Alzheimer's disease, Crohn's disease, ovarian cancer, and even baldness. It is no wonder, then, that *Time* magazine deemed the "Retail DNA Test" the Invention of the Year in 2008 (Hamilton, 2008).

Despite the enhanced accessibility to genetic information brought on by the DTC wave, some controversy surrounds the use of and distribution of genetic risk data through DTC means. In 2013, the Food and Drug Administration (FDA) sent warning letters to several DTC testing companies, including 23andMe, prompted by concerns over releasing false health information and possible misinterpretation of results. In response, 23andMe discontinued the disclosure of disease risk information. While the company still provides full ancestry data, they now release only raw genetic data. These raw genetic data cannot be interpreted by untrained consumers and require consultation with a healthcare professional.

Although the scope of this entry focuses predominantly on DTC genetic testing, the empirical literature base remains quite small, perhaps reflecting the dramatically and rapidly evolving nature

of the DTC genetic testing landscape. Thus, when appropriate, we reference a related literature on DTC advertising of prescription pills to draw relevant connections. Although DTC genetic testing and advertising of prescription pills are distinct, they both provide consumers with opportunities to make health decisions prior to consultation with a healthcare professional. We suspect that where the DTC genetic testing literature currently lacks evidence, research on DTC advertising of prescription pills can offer informative avenues for future research.

The nature of DTC testing raises a number of questions relevant to health psychology, two of which are important for the scope of this entry. First, what psychosocial variables influence people's interest in DTC genetic testing? Second, what outcomes and downstream effects result from getting a DTC genetic test?

Psychosocial Predictors of Interest in DTC Testing

The introduction of DTC genetic testing opened up new research avenues to examine psychological predictors of interest in (and uptake of) DTC tests. In contrast to the traditional, in-office genetic tests, DTC genetic tests offer a more private, less expensive (~\$99), and more autonomous method for learning about health risks. However, the findings thus far do not paint a complete or clear picture regarding the psychosocial predictors of DTC testing, likely reflecting the nascent state of the literature.

Demographics

Before examining the subjective psychosocial variables that may influence the use of DTC testing, we first consider the role of demographics. Generally, people with the greatest interest in and use of DTC tests tend to identify as well educated, employed or retired, and Caucasian (Kaufman, Bollinger, Dvoskin, & Scott, 2012; Sweeny, Ghane, Legg, Andrews, & Huynh, 2014; Wilson et al., 2008). Although one study noted that older participants more frequently used an at-home test in the past, younger participants reported greater intentions to use a DTC test in the future (Wilson et al., 2008; but see Sweeny & Legg, 2011 for data showing no age differences).

A similarly inconsistent pattern emerges for gender. Some sources point to greater interest among women (Wilde, Meiser, Mitchell, Hadzi-Pavlovic, & Schofield, 2011), whereas other evidence suggests that men are more likely to use DTC testing (Wilson et al., 2008). Still other sources suggest no gender difference in DTC testing intentions (Sweeny & Legg, 2011).

Although no consistent link between income and DTC testing currently exists (Sweeny et al., 2014; but see Kaufman et al., 2012), some evidence from the DTC advertising literature suggests that people in lower income brackets are more persuaded by DTC advertising for prescription pills (Mathew, Spake, & Finney, 2008), a finding that may reflect lower health literacy levels in lower income individuals.

Self-Reported Reasons for and Against Testing

The most commonly reported reason for pursuing DTC genetic testing (or reporting intentions to do so) is that people are curious and desire detailed information about their genetic profiles (Goldsmith, Jackson, O'Connor, & Skirton, 2012; Roberts & Ostergren, 2013). Genetic counselors agree that DTC genetic testing can raise awareness and provide knowledge about risk factors (Hock, Christensen, Yashar, Gollust, & Uhlmann, 2011). They also report

that DTC genetic testing is advantageous for people in rural locations who live far from trained genetic counselors (Hock et al., 2011). Further, in a review of the DTC genetic testing literature, Goldsmith et al. (2012) indicated that many people believe genetic test results will motivate future healthy behaviors, facilitate family planning and discussion for future health conditions, and could be used in conjunction with a physician's oversight to monitor health.

In contrast, people's reported reasons against testing tend to focus on fears of their emotional and psychological reactions if they learn they are at risk for a disease (Goldsmith et al., 2012). From clinicians' perspectives, the possibility of misinterpreting results is a commonly listed reason against testing, especially in the absence of a trained healthcare professional (Hock et al., 2011). Ninety percent of these genetic counselors also listed the risk of feeling a false sense of security if a person learns that he or she is not a carrier for a certain disease. Finally, the possible risks of inadequate security protections or abuse or misuse of private information are frequently provided reasons against testing (Critchley, Nicol, Otlowski, & Chalmers, 2015; Mathews, Wayne, & Carter, 2012).

Perceived Barriers and Benefits to Testing

The costs and benefits associated with taking a test can affect whether people pursue DTC testing. Perceived barriers, or the costs and disadvantages associated with testing, decrease people's intentions to obtain DTC genetic testing (Sweeny & Legg, 2011). Barriers include, but are not limited to, concerns about the unethical disclosure of genetic results, financial cost of testing, difficulty taking the test, and difficulty returning the test back to the company.

In contrast, believing that a genetic test will offer advantages and benefits consistently predicts interest in general genetic testing (Sweeny et al., 2014) and also for DTC genetic testing (Sweeny & Legg, 2011). Some of these benefits include the additional privacy afforded by DTC genetic testing, less embarrassment if seeking information about stigmatized diseases, lower cost compared with in-office genetic testing, motivation to adopt other healthy and preventive behaviors, and greater ability to prepare for and cope with a possible disease. Although people may also report the autonomy afforded by DTC genetic testing as a benefit, it remains to be seen whether perceived benefits related to autonomy and privacy have shifted in light of the 2013 FDA regulation. Further, now that DTC genetic testing companies such as 23andMe only provide raw genetic data, the research currently makes no comment regarding whether potential customers now perceive greater barriers due to the need to seek out healthcare professionals to interpret their genetic profiles.

Knowledge and Awareness

An interesting phenomenon captured in the literature is that some knowledge about and awareness of DTC genetic testing positively relates to intentions to pursue testing, but upon viewing additional or more complete information, people's intentions to pursue DTC testing tend to decline, and preference for obtaining genetic test results through a physician increases (Wilde et al., 2011). A similar pattern emerges when considering the valence of information people view. Participants who read both positive and negative information about DTC testing reported similar levels of interest to those who viewed only negative information (both were less interested than people who viewed only positive information), suggesting that people are particularly sensitive to the downsides of DTC testing (Sweeny & Legg, 2011). Perhaps naive curiosity is a more powerful predictor of intentions to test initially, but people become more cautious upon considering the possible barriers and disadvantages of testing.

Anticipated Regret

Before deciding to take a DTC genetic test, people often imagine how they will feel after they receive their results or how they will feel if they miss out on the chance to take the test. This anticipatory regret over testing and over not testing predicts intentions to test (Sweeny & Legg, 2011). In Sweeny and Legg's (2011) study, participants who viewed positive information about genetic testing reported greater anticipated regret over not testing (i.e., missing out on the chance to test), and participants who viewed only negative information or a combination of positive and negative information about DTC testing reported greater anticipated regret over testing. Both forms of anticipated regret mediated the relationship between information valence and intentions to pursue testing.

Advertising and Risk Framing

Advertising for DTC genetic tests plays a key role in converting curious parties into paying customers. As noted above, people report more positive attitudes toward DTC genetic testing after viewing positive information, but not after viewing a mix of positive and negative information (Sweeny & Legg, 2011). DTC genetic testing companies can also strategically frame their product in ways that garner customer support. An analysis of 22 DTC genetic testing companies documented the variety of risk-framing strategies such as emphasizing the commonality of certain diseases, the predominance of genetic components of disease at the exclusion of environment factors, and the need to know this genetic information to empower oneself (Einsiedel & Geransar, 2009). Other advertising techniques found in this analysis included emphasizing the simplicity of DTC genetic testing procedures, enhancing trust and credibility, and aligning the company with the scientific community. Similar patterns emerge in DTC advertising of prescription pills: overly simplified, overly positive, and overly emotional advertisements that emphasize the biological causes of illness and how prescription pills offer a way for consumers to gain control again (Frosch, Krueger, Hornik, Cronholm, & Barg, 2007).

Consequences of DTC Testing

Prospective studies examining people's intentions for testing and reactions to testing are limited. Of note, researchers affiliated with 23andMe conducted a longitudinal study to follow nearly 1,000 customers who received their genetic test results (Carere et al., 2014). Until more thorough research emerges, the current literature provides only limited evidence about people's subsequent actions, comprehension, and behavioral and psychological changes after receiving DTC genetic results.

Subsequent Consultations with Healthcare Professionals

Prior to the FDA regulating the distribution of health risk data, customers of DTC genetic tests received information about their carrier and risk status for a range of diseases and health issues ranging from Huntington's disease to baldness. After receiving these data, 43% of surveyed customers reported seeking additional information about one of their results, and 28% of the customers reported discussing their results with a healthcare professional (Kaufman et al., 2012; see Bloss, Schork, & Topol, 2011 for similar patterns). Similarly, nearly half of

genetic counselors reported having a patient-initiated discussion about DTC genetic testing options, and 14% indicated that a patient had asked them to interpret DTC test results (Hock et al., 2011).

Comprehension of Results

Although many critics fear that consumers of DTC genetic testing will misinterpret their health risks, emerging evidence suggests the opposite effect. In their systematic review of the DTC testing literature, Roberts and Ostergren (2013) concluded that generally people can correctly recall and interpret their genetic risks. And despite advertising campaigns emphasizing the genetic causes of diseases, people receiving their genetic profiles understand environmental factors that contribute to illness above and beyond genetic predispositions (Kaphingst et al., 2012).

Changes in Health Behaviors

Receiving results from a DTC genetic test increases intentions to foster healthy behaviors such as dietary improvements and exercise, as reported by a third of Kaufman and colleagues' (2012) survey respondents. For example, a notable minority of respondents reported changing their medications or supplements or taking additional health tests after receipt of their genetic profiles (Kaufman et al., 2012). However, as is the case with many studies linking attitudes to behavior, people's intentions to increase their healthy habits after receiving DTC test results do not necessarily translate into actual behavior change. In fact, after receiving DTC genetic profiles, one study found no differences in people's dietary or exercise habits (Bloss et al., 2011).

Psychological Effects

After receiving results from DTC genetic tests, research points to changes in people's perceptions such that they tend to experience short-term increases in perceived risk for certain diseases, especially rare diseases (e.g., Graves' disease but not prostate cancer; James et al., 2011). However, this pattern no longer holds when people report their perceived risk a year after receiving their results. Contrary to what many people view as an important reason not to test, people do not seem to show increased levels of worry or anxiety after receiving their results (Bloss et al., 2011; James et al., 2011; Roberts & Ostergren, 2013).

In one of the first prospective studies on this topic, DTC genetic testing customers reported greater knowledge about genetics after receiving their test results but reported decreased genetics-related self-efficacy (Carere, Kraft, Kaphingst, Roberts, & Green, 2015). Regret over deciding to test negatively correlated with genetics self-efficacy. However, discussing results with a genetic counselor or other healthcare professional reversed this effect and predicted greater levels of genetics-related self-efficacy.

Opting Not to View Results

Finally, although it is interesting to consider the reactions of people who receive their results, what about the individuals who opt not to view their genetic profiles upon availability? Research on this topic is limited, but one study suggested that approximately 10% of participants choose not to view their test results when they became available (Bloss et al., 2011). Especially given

that DTC genetic testing customers now receive only raw genetic data that must be interpreted by a healthcare professional, examination of the reasons and mechanisms behind people's reluctance to view their results seems a beneficial avenue for future lines of research.

Conclusion

Undoubtedly, the field of DTC genetic testing is rapidly changing. With this change comes new opportunities for research. We see four important avenues for exploration. First, given the new FDA regulations, how will people's interest in DTC genetic testing compare with traditional, in-office forms of genetic testing? Second, what individual differences predict testing uptake and seeking interpretation guidance for raw genetic data? Third, as more prospective studies emerge, what are the consequences and benefits associated with DTC genetic testing? Finally, as telemedicine and DTC opportunities continue to evolve, what tools and training initiatives do medical practitioners need to best equip them to serve their patients who use DTC genetic testing outlets? These questions and many others offer numerous research opportunities to continue unraveling the psychosocial correlates of DTC genetic testing.

Author Biographies

Angela M. Legg is an assistant professor of psychology at Pace University. She earned a PhD in social/personality psychology from the University of California, Riverside, in 2014. Angela's research examines bad news delivery and genetic testing decisions. She has published in *Personality and Social Psychology Bulletin*, *Journal of Genetic Counseling*, and *Psychology & Health*. Her research has been featured in *Psychology Today*, *National Geographic*, and *Time*.

May Reinert is a graduate student in Pace University's Mental Health Counseling master's program. She earned her bachelor's degree in applied psychology from Pace University in 2015. May was a founding member of the Social, Health, and Relational Positive Psychology (SHARPP) lab. Her research examines predictors of genetic testing decisions. She is also interested in providing counseling services to the deaf and hard of hearing population.

References

- Bloss, C. S., Schork, N. J., & Topol, E. J. (2011). Effect of direct-to-consumer genomewide profiling to assess disease risk. *New England Journal of Medicine*, 364, 524–535. doi:10.1056/NEJMoa1011893
- Carere, D. A., Couper, M. P., Crawford, S. D., Kalia, S. S., Duggan, J. R., Moreno, T. A., ... Green, R. C. (2014). Design, methods, and participant characteristics of the Impact of Personal Genomics (PGen) Study, a prospective cohort study of direct-to-consumer personal genomic testing customers. *Genome Medicine*, 6, 96–110.
- Carere, D. A., Kraft, P., Kaphingst, K. A., Roberts, J. S., & Green, R. C. (2015). Consumers report lower confidence in their genetics knowledge following direct-to-consumer personal genomic testing. *Genetics in Medicine*. doi:10.1038/gim.2015.34
- Critchley, C., Nicol, D., Otlowski, M., & Chalmers, D. (2015). Public reaction to direct-to-consumer online genetic tests: Comparing attitudes, trust, and intentions across commercial and conventional providers. *Public Understanding of Science*. doi:10.1177/0963662513519937
- Einsiedel, E. F., & Geransar, R. (2009). Framing genetic risk: trust and credibility markers in online direct-to-consumer advertising for genetic testing. *New Genetics and Society*, 28(4), 339–362.

- Frosch, D. L., Krueger, P. M., Hornik, R. C., Cronholm, P. F., & Barg, F. K. (2007). Creating demand for prescription drugs: A content analysis of television direct-to-consumer advertising. *Annals of Family Medicine*, 5, 6–13.
- Goldsmith, L., Jackson, L., O'Connor, A., & Skirton, H. (2012). Direct-to-consumer genomic testing: Systematic review of the literature on user perspectives. *European Journal of Human Genetics*, 20, 811–816.
- Hamilton, A. (2008). Best Invention of 2008. Retrieved from <http://content.time.com/time/specials/packages/0,28757,1852747,00.html>.
- Hock, K. T., Christensen, K. D., Yashar, B. M., Roberts, J. S., Gollust, S. E., & Uhlmann, W. R. (2011). Direct-to-consumer genetic testing: An assessment of genetic counselors' knowledge and beliefs. *Genetics in Medicine*, 13, 325–332.
- James, K. M., Cowl, C. T., Tilburt, J. C., Sinicrope, P. S., Robinson, M. E., Frimannsdottir, K. R., ... Koenig, B. A. (2011). Impact of direct-to-consumer predictive genomic testing and risk perception and worry among patients receiving routine care in a preventive health clinic. *Mayo Clinic Proceedings*, 86, 933–940.
- Kaphingst, K. A., McBride, C. M., Wade, C., Alford, S. H., Reid, R., Larson, E., ... Brody, L. C. (2012). Patients' understanding of and responses to multiplex genetic susceptibility test results. *Genetics in Medicine*, 14, 681–687.
- Kaufman, D. J., Bollinger, J. M., Dvoskin, R. L., & Scott, J. A. (2012). Risky business: Risk perception and the use of medical services among customers of DTC personal genetic testing. *Journal of Genetic Counseling*, 21, 413–422. doi:10.1007/s10897-012-9483-0
- Mathew, J., Spake, D. F., & Finney, Z. (2008). Consumer attitudes toward pharmaceutical direct-to-consumer advertising. *International Journal of Pharmaceutical and Healthcare Marketing*, 2(2), 117–133. doi:10.1108/17506120810887916
- Mathews, R., Wayne, C., & Carter, A. (2012). Direct-to-consumer genetic testing for addiction susceptibility: A premature commercialisation of doubtful validity and value. *Addiction*, 107, 2069–2074.
- Roberts, J. S., & Ostergren, J. (2013). Direct-to-consumer genetic testing and personal genomics services: A review of recent empirical studies. *Current Genetic Medicine Reports*, 1, 182–200.
- Sweeny, K., Ghane, A., Legg, A. M., Huynh, H. P., & Andrews, S. E. (2014). Predictors of genetic testing decisions: A systematic review and critique of the literature. *Journal of Genetic Counseling*, 23(3), 263–288. doi:10.1007/s10897-014-9712-9
- Sweeny, K., & Legg, A. M. (2011). Predictors of interest in direct-to-consumer genetic testing. *Psychology & Health*, 26(10), 1259–1272. doi:10.1080/08870446.2010.514607
- Wilde, A., Meiser, B., Mitchell, P. B., Hadzi-Pavlovic, D., & Schofield, P. R. (2011). Community interest in predictive genetic testing for susceptibility to major depressive disorder in a large national sample. *Psychological Medicine*, 41, 1605–1614.
- Wilson, S., Ryan, A. V., Greenfield, S. M., Clifford, S. C., Holder, R. L., Pattison, H. M., ... McManus, R. J. (2008). Self-testing for cancer: A community survey. *BMC Cancer*, 8, 1–87. doi:10.1186/1471-2407-8-102

Suggested Reading

- Goldsmith, L., Jackson, L., O'Connor, A., & Skirton, H. (2012). Direct-to-consumer genomic testing: Systematic review of the literature on user perspectives. *European Journal of Human Genetics*, 20, 811–816.
- Roberts, J. S., & Ostergren, J. (2013). Direct-to-consumer genetic testing and personal genomics services: A review of recent empirical studies. *Current Genetic Medicine Reports*, 1, 182–200.
- Sweeny, K., Ghane, A., Legg, A. M., Huynh, H. P., & Andrews, S. E. (2014). Predictors of genetic testing decisions: A systematic review and critique of the literature. *Journal of Genetic Counseling*, 23(3), 263–288. doi:10.1007/s10897-014-9712-9

Disease-Specific Worry

Kevin D. McCaul¹, Renee E. Magnan²,
and Michael P. Mead¹

¹ *Department of Psychology, North Dakota State University, Fargo, ND, USA*

² *Department of Psychology, Washington State University Vancouver, Vancouver, WA, USA*

What Is Worry?

Definitions of worry historically have been derived from research on “worriers.” For example, Borkovec and colleagues defined worry as a chain of thoughts and images that are negatively affect laden and relatively uncontrollable (Borkovec, Robinson, Pruzinsky, & Dupree, 1983). Perceived lack of control may be the most important characteristic of trait worry, but we suggest that non-pathological worry need not involve uncontrollability. Instead, “normal” worry can be defined simply as a combination of negative *feelings* produced by unwanted, ruminative negative *thoughts*. Importantly, worry is neither exclusively an affective nor a cognitive experience; instead, it is a combination of the two. Although worry can occasionally persist over time, such as when one ruminates all night in bed about a forthcoming event, non-pathological worry is often short lived. McCaul, Branstetter, O'Donnell, Jacobson, and Quinlan (1998) called such experiences “emotional moments”—not full-blown emotional episodes represented by emotion descriptions such as fear or anger but rather the experience of relatively brief, negative feelings as one contemplates concerns having implications for the future.

We suggest that worry about disease can occur as a normal emotional moment and that normal worrying differs from two other related constructs: perceived risk and anxiety. Perceived risk is a cognitive construct, whereas we have defined worry as a combination of negative thoughts and negative affect. Moreover, the two constructs are potentially separable. One can, for example, perceive one's risk of contracting cancer as minimal but nonetheless worry greatly about the possibility; conversely, one can perceive one's risk of contracting cancer as likely but simply not worry about it, much as some cigarette smokers do. Research also suggests that worry about disease stemming from one's unhealthy behaviors predicts self-protective behaviors independently—and sometimes more strongly—than risk judgments (Magnan, Köblitz, Zielke, & McCaul, 2009).

Worry also differs from anxiety. Anxiety is accompanied by physical symptomology (e.g., arousal of the sympathetic system); worry does not necessarily involve symptoms. Worry has a particular target; anxiety can be general with no specific antecedent. Finally, as we shall see, worry often motivates problem solving; anxiety typically does not.

Do People Worry About Disease?

Worry can be directed toward many different life domains—one's family, job, finances, and friendships, for example. Interestingly, health concerns do *not* top the list of worries people report; indeed, a commonly used worry tool—the Worry Domains Questionnaire—does not include health or disease as a possible worry target. Survey data confirm the conclusion that disease does not head the list of worry domains. For example, young adult, healthy German women were interviewed about their worries (Becker, Goodwin, Hölting, Hoyer, & Margraf, 2003). The most common worries concerned work issues, closely followed by concerns about the health of others and then worry about their families. Lindsay et al. (2006) assessed worry among both men and women across the lifespan. They found that 30–40% of participants worried about relationships, finances, and work. Older adults reported having these concerns less frequently. Health-related worry was reported less often overall but remained consistent throughout the lifespan. In contrast, in a review of 21 studies assessing older adults, Hunt, Wisocki, and Roger (2009) noted that although few worries were reported, health concerns were prominent in their older samples.

Regardless of whether health concerns are the most intense or frequent worries people have, when people *do* worry about health and disease, some diseases attract more concern than others. In a sample of US adults, Wang et al. (2009) assessed worry about six different diseases: heart disease, diabetes, stroke, colon cancer, breast cancer, and ovarian cancer. Women worried most about breast cancer, followed by heart disease and diabetes. In contrast, men worried most about heart disease, reporting somewhat lower levels of worry for diabetes, stroke, and colon cancer. Senay, Alford, and Kaphingst (2012) conducted a similar survey study, assessing worry about 8 diseases among nearly 2,000 healthy adults. Both men and women reported worrying most about cardiovascular diseases (heart disease, high blood pressure, high cholesterol) followed by diabetes, colon cancer, lung cancer, skin cancer, and osteoporosis, and other survey studies reveal similar results.

Worry clearly depends on the specific disease assessed, with research suggesting that cardiovascular disease—the most common ailment affecting American adults—also attracts the most worry. However, it is also important to consider the *overall* degree of worry about disease. In the Wang et al. (2009) study, for example, participants rated their degree of worry during the last 4 weeks on a 1 (not at all worried) to 5 (worried almost all the time) scale. Overall, the frequency of worry was fairly low: only women describing worry about breast cancer reported a mean worry over 2. Such data suggest that many participants worry not at all about a particular disease. The mean level of worry described in Senay et al. (2012) was greater—perhaps because respondents were asked about (a) the intensity rather than the frequency of worry and (b) a different timeframe, lifetime versus the prior month. Specifically, participants were asked on a 1 (not worried at all) to 7 (very worried) scale how much they worried about developing each particular disease *throughout their lifetime*. Heart disease had a reported mean of 4.62, high blood pressure 4.59, high cholesterol 4.33, and diabetes 4.26. Colon cancer, skin cancer, lung cancer, and osteoporosis all had means below 4.

We were unable to uncover studies reporting absolute frequencies of worrying data across diseases, that is, studies addressing the proportion of a sample reporting *any or no* worry about different diseases. Some data from individual disease studies, however, suggest that the overall proportion of persons worrying a great deal could be relatively low. For example, Moser, McCaul, Peters, Nelson, and Marcus (2007) used nationally representative data from interviews with over 6,000 adults. The survey questions assessed worry for several cancer types. Approximately 2/3 of the respondents said they “never” or “rarely” worried about getting colon cancer, and a similar proportion of men said the same about prostate cancer. Over half of the women surveyed said they rarely or never worried about breast cancer. Moving forward, it would be important to obtain these sorts of data across many different diseases and to report categorical frequencies as well as scale means.

Research suggests the following in terms of how much we worry about disease: (a) overall levels are relatively low in comparison to worries about other aspects of life; (b) older people are more likely than younger adults to worry about their health, although worry is modest even among the former group; (c) worry about particular diseases ties in some way to disease likelihood because people worry more about diseases they are more likely to experience; and (d) many people worry minimally or even not at all about disease.

What Determines the Degree of Worry About Disease?

Many factors influencing the degree to which one experiences worry are associated with perceived disease risk. Perceived risk is composed of susceptibility judgments (the degree to which one feels they are likely to develop a disease) and severity judgments (how bad the disease consequences would be). Both of these risk components predict worry about developing a disease such that stronger perceptions of personal likelihood or severity are associated with more worry. However, worry’s associations with susceptibility judgments are stronger than its associations with severity (Cameron, Sherman, Marteau, & Brown, 2009). It could be that the difference in association strength between perceived likelihood and severity may be due to self-relevance. Worry about a disease and perception of personal likelihood of a disease are self-relevant in that one must look to the self to judge each. In contrast, severity judgments can be made without referencing the self. That is, one can perceive the outcomes as severe without necessarily thinking they are susceptible to experiencing such consequences.

The commonsense model (CSM) of illness representation (Leventhal, Brissette, & Leventhal, 2003) provides a general framework for how different disease-related variables influence risk and worry. This model posits that how people think about (“represent”) illness may elicit affective reactions such as worry. Illness representations include five content domains: identity (illness label and symptoms of illness), illness cause (e.g., heredity, behavior), illness timeline (time to onset, duration), illness consequences, and illness control (personal or medical control of progression or cure). These domains should differentially influence likelihood and severity judgments, such that beliefs about illness identity, cause, and timeline should affect likelihood estimates and beliefs about consequences and controllability should affect severity judgments. Importantly, all five illness representations should also influence worry about the disease.

Linked to the issue of likelihood estimates, Cameron (2008) found that illness identity (e.g., skin burns easily, dark moles), cause (e.g., hereditary, behavioral choices such as skin exposure), and timeline (e.g., developing skin cancer “at this age”) each predicted worry about

developing skin cancer. An important component of identifying an illness is beliefs about symptoms that indicate illness, such as shortness of breath indicating heart disease or a discolored mole indicating skin cancer. Illness symptoms should typically increase worry, although they could also reduce worry in some special circumstances. Moye et al. (2015) compared worry about developing melanoma before and after total-body digital photography among individuals with atypical mole syndrome. This approach creates high-resolution images that can identify changes in the skin surface. Worry scores were lower after the photography session than before but only among individuals with a history of melanoma. Assuming that individuals with a previous history of melanoma generally have heightened susceptibility perceptions, when viewing images that show no visual evidence of melanoma (suggesting that melanoma is not present), these individuals reported reduced worry.

“Cause” is another CSM component that influences susceptibility judgments and worry. Most prominently, individuals with a family history of disease report more frequent and more intense worry about developing a disease than individuals without a family history (Vassy & Meigs, 2011). A family history is likely to encourage beliefs that a disease has a genetic cause, thereby increasing susceptibility judgments. Another “cause” factor is the extent to which individuals believe that specific risky behaviors lead to illness or that health-protective behaviors prevent illness. Thus, individuals who engage in risky behaviors may perceive themselves at greater risk and be more worried about illness. Finney Rutton, Blake, Hesse, Augustson, and Evans (2011) found that compared with former and nonsmokers, current smokers were more likely to report their likelihood of developing lung cancer as “very high” and to report worrying about cancer “often or all of the time.” Cameron (2008) also showed that one facet of “timeline”—time to onset of a disease—was related to likelihood judgments and worry. Specifically, timeline beliefs (“People of my age are likely to develop skin cancer now—at this age”) were associated with personal likelihood of developing skin cancer and worry about contracting skin cancer.

The CSM also provides a framework for factors contributing to severity judgments. In particular, the CSM includes an “illness consequences” domain. The potential consequences of a disease are many. They could be medical (e.g., long-term disability), social (e.g., social life, money), and psychological (e.g., changes to one’s identity). This category would also include perceptions of the consequences of the disease treatment. Cameron (2008), for example, found that perceptions of more intense pain from treatment predicted greater worry about developing skin cancer. Similarly, women undergoing a genetic test for breast cancer reported more worry when they also thought that the consequences of a positive finding would be devastating (Cameron & Reeve, 2006).

Illness timeline perceptions are theoretically associated with likelihood and not severity judgments within the CSM framework, but they have been positively associated with severity judgments nonetheless (Cameron, 2008). Additionally, we were unable to find empirical evidence that control over disease outcomes (the other illness representation thought to be associated with severity judgments; Cameron, 2008) is associated with severity judgments, although it has been connected with worry. Specifically, Gibbons and Groarke (2015) found that stronger perceptions of control (personal and treatment) over breast cancer were associated with less worry about getting cancer in one’s lifetime. Importantly, severity judgments are less commonly assessed than likelihood judgments in the extant literature, and thus it is difficult to determine the extent to which these constructs factor into severity judgments—a task left for future research.

In the CSM, symptoms of illness are incorporated as a component of perceived likelihood. Yet, visibility of symptoms of disease may also be a contributing factor to severity as it is one

way that disease severity can be interpreted (e.g., consequences of mouth cancer could be a less severe sore on the lip or more severe tooth decay). Further, imagery of symptoms may promote greater worry about developing the disease because it keeps the consequences of the disease more salient or enhances concern of social consequences such as embarrassment or stigmatization. For example, messages about the consequences of smoking elicit greater worry when they are presented in graphic format (showing images of smoking consequences) compared with text-only formats (Magnan & Cameron, 2015).

The CSM describes a dynamic process in which illness representations, feelings about illness, and behavior change over time. As one has new experiences or acquires new information from one's own experiences, social network, physicians, or news/educational materials, representations of the various components may change. To this end, experiences such as familiarity with individuals who have experienced the disease and personal history of the disease, each of which is typically associated with greater worry about (re)developing the specific disease (Moye et al., 2015), may affect *all* illness representations. For example, a person with little knowledge regarding breast cancer may gain more insight into the symptoms (i.e., illness identity), cause, and timeline of breast cancer from helping a friend who is currently undergoing treatment. A person having previously experienced breast cancer may worry about recurrence because of firsthand knowledge of the disease consequences.

Representations of illness risk clearly influence the degree to which one worries about illness. Indeed, communicating risk information can increase worry about developing an illness (Magnan et al., 2009). Although factors linked to perceived risk may influence the degree of worry experienced, enhancing knowledge about health risks in and of itself does not always increase worry. Personal perceptions of risk are not necessarily rational estimates, but rather they can sometimes be subjective estimates derived from various characteristics one associates with risks in addition to aspects of feelings of risk. For example, in the case of a rare but serious disease, the actual risk of developing the disease is low, but dread associated with the disease may increase worry about the disease. Ultimately, although various representations of illness may contribute to perceived risk, they may also directly influence worry *without* influencing perceptions of risk. That is, one may worry about a disease without necessarily feeling "at risk." In short, risk and worry are not the same thing.

Worry Consequences

Worry can have both negative and positive consequences. One can worry "too much," and we would expect extremes of worry about disease to be accompanied by a lowered quality of life. Data confirming this possibility have been reported primarily among individuals who are already suffering from a particular disease. Worry about cancer recurrence among survivors, for example, is associated with a lower quality of life (Tewari & Chagpar, 2014). Extremes of worry could also promote avoidance of screening because of worries about finding the disease (Cameron, 2003). An interesting dichotomy appears here: Worry about the outcomes of a screening test motivates avoidance whereas worry about the disease itself motivates one to obtain a screening in the first place.

The consequences of worry need not always be negative. Worry about disease is related to problem solving and can facilitate active coping. In this regard, worry about disease promotes self-protective behaviors. For example, smokers who worry about disease think more about quitting (Magnan et al., 2009). People who worry more about cancer are more likely to have regular mammography screening and to have had a colon cancer screening test (Moser et al.,

2007). And, among women with an inherited risk of breast and ovarian cancer, those who worry more are more likely to adopt risk-reducing surgery (Portnoy, Loud, Han, Mai, & Greene, 2015).

We have previously proposed three reasons why worry might motivate self-protective behaviors. Worry can provide an additional, *rational* thought about taking health-protective action. One might reason, “If I’m this worried about having a heart attack, I should probably do something about it.” Second, worrying by definition involves intrusive thoughts. Thinking frequently about potentially suffering from disease keeps the issue “in mind,” serving as a persistent reminder that something needs to be done. Third, worry may motivate problem solving simply to end the negative feelings accompanying worry itself. Regardless of why, the point is clear: Worry can motivate planning and produce self-protective behaviors.

Conclusions

People worry about many aspects of their lives, only one of which is their health. Many people worry about specific diseases, although most individuals do not report a great deal of worry. People are more likely to worry about a particular disease if they feel at risk—especially if they feel susceptible, but also if they see the disease consequences as severe. Other variables that enhance worry in part through risk judgments include experiencing symptoms, a family history of the disease, perceptions of distressing disease treatments, a perceived lack of control over the disease, and negative images arising from thinking about the disease. Finally, some variables will increase worry separately from judgments of risk, such as feelings of dread about the disease. When people *do* worry, it can have either or both positive and negative consequences. Worrying a great deal about disease can lead to a lower quality of life, but it is important to also recognize that worrying about disease motivates many individuals to adopt behaviors that will effectively stave off disease and its consequences.

Author Biographies

Kevin D. McCaul is a professor of psychology at North Dakota State University. A social psychologist, Dr. McCaul’s most recent research has addressed relationships between cognitions, feelings, and self-protective health behaviors. In particular, he has connected thoughts about risk and feelings about worry in the context of cancer screening and smoking prevention and cessation. His empirical findings clearly suggest that worry is an important motivator of self-protective behaviors such as mammography screening and smoking cessation.

Renee E. Magnan is an assistant professor in the Psychology Department at Washington State University Vancouver. Her research uses social psychological theory to address issues in disease prevention and health promotion. A general focus of her work addresses how affect influences perceptions of health communications and health decisions and motivates self-protective health behaviors (e.g., smoking cessation, exercise, alcohol use).

Michael P. Mead completed his undergraduate work at Grand Valley State University and is presently a graduate student in the Health/Social Psychology PhD program at North Dakota State University, where he works in the SLEEP laboratory under the supervision of Dr. Leah Irish. His research interests include relationships between marital interactions and sleep quality, sleep hygiene behaviors, and mechanisms linking sleep and diet.

References

- Becker, E. S., Goodwin, R., Hölting, C., Hoyer, J., & Margraf, J. (2003). Content of worry in the community: What do people with generalized anxiety disorder or other disorders worry about? *The Journal of Nervous and Mental Disease*, 191, 688–691. doi:10.1097/01.nmd.0000092198.20420.fc
- Borkovec, T. D., Robinson, E., Pruzinsky, T., & Dupree, J. A. (1983). Preliminary exploration of worry: Some characteristics and processes. *Behaviour Research and Therapy*, 23, 481–482. doi:10.1016/0005-7967(83)90121-3
- Cameron, L. D. (2003). Anxiety, cognition, and responses to health threats. In L. D. Cameron & H. Leventhal (Eds.), *The self-regulation of health and illness behavior* (pp. 157–183). London: Routledge.
- Cameron, L. D. (2008). Illness risk representations and motivations to engage in protective behavior: The case of skin cancer risk. *Psychology & Health*, 23, 91–112. doi:10.1080/14768320701342383
- Cameron, L. D., & Reeve, J. (2006). Risk perceptions, worry, and attitudes about genetic testing for breast cancer susceptibility. *Psychology & Health*, 21, 211–230. doi:10.1080/14768320500230318
- Cameron, L. D., Sherman, K. A., Marteau, T. M., & Brown, P. M. (2009). Impact of genetic risk information and type of disease on perceived risk, anticipated affect, and expected consequences of genetic tests. *Health Psychology*, 28(3), 307–316.
- Gibbons, A., & Groarke, A. (2015). Can risk and illness perceptions predict breast cancer worry in healthywomen? *Journal of Health Psychology* (online ahead of print). doi:10.1177/1359105315570984
- Hunt, S. L., Wisocki, P. A., & Roger, P. R. (2009). “What, me worry?” An examination of the literature on worry and the older adult. *The Behavior Therapist*, 32(1), 9–16. doi:10.1177/1359105315570984
- Leventhal, H., Brissette, I., & Leventhal, E. (2003). The common-sense model of self-regulation of health and illness. In L. D. Cameron & H. Leventhal (Eds.), *The self-regulation of health and illness behaviour* (pp. 42–65). London: Routledge.
- Lindesay, J., Baillon, S., Brugha, T., Dennis, M., Stewart, R., Araya, R., & Meltzer, H. (2006). Worry content across the lifespan: An analysis of 16-to 74-year-old participants in the British national survey of psychiatric morbidity. *Psychological Medicine*, 36(11), 1625–1633. doi:10.1017/S0033291706008439
- Magnan, R. E., & Cameron, L. D. (2015). Do young adults perceive that cigarette graphic warnings provide new knowledge about the harms of smoking? *Annals of Behavioral Medicine*, 49, 594–604. doi:10.1007/s12160-015-9691-6
- Magnan, R. E., Köblitz, A. R., Zielke, D. J., & McCaul, K. D. (2009). The effects of warning smokers on perceived risk, worry, and motivation to quit. *Annals of Behavioral Medicine*, 37, 46–57. doi:10.1007/s12160-009-9085-8
- McCaul, K. D., Branstetter, A. D., O'Donnell, S. M., Jacobson, K., & Quinlan, K. B. (1998). A descriptive study of breast cancer worry. *Journal of Behavioral Medicine*, 21, 565–579. doi:10.1023/A:1018748712987
- Moser, R. P., McCaul, K. D., Peters, E., Nelson, W., & Marcus, S. (2007). Risk and worry as predictors of cancer health-protective actions: Data from the Health Interview National Trends Survey (HINTS). *Journal of Health Psychology*, 12, 53–65. doi:10.1177/1359105307071735
- Moye, M. S., King, S. M. C., Rice, Z. P., DeLong, L. K., Seidler, A. M., Veledar, E., ... Chen, S. C. (2015). Effects of total-body digital photography on cancer worry in patients with atypical mole syndrome. *JAMA Dermatology*, 151, 137–143. doi:10.1001/jamadermatol.2014.2229
- Portnoy, D. B., Loud, J. T., Han, P. K. J., Mai, P. L., & Greene, M. H. (2015). Effects of false-positive cancer screenings and cancer worry on risk-reducing surgery among BRCA1/2 carriers. *Health Psychology*, 34, 709–717. doi:10.1037/hea0000156
- Rutton, L. J. F., Blake, K. D., Hesse, B. W., Augustson, E. M., & Evans, S. (2011). Illness representations of lung cancer, lung cancer worry, and perceptions of risk by smoking status. *Journal of Cancer Education*, 26, 747–753. doi:10.1007/s13187-011-0247-6
- Senay, I., Alford, S. H., & Kaphingst, K. A. (2012). Judging risk for multiple diseases: The role of disease worry. *Journal of Health Psychology*, 18, 554–560. doi:10.1177/1359105312437263

- Tewari, A., & Chagpar, A. B. (2014). Worry about breast cancer recurrence: A population-based analysis. *The American Journal of Surgery*, 80, 640–645.
- Vassy, J. L., & Meigs, J. B. (2011). Despite underestimated familial risk by self-report, family history correlates with perceived risk and worry about chronic diseases such as coronary heart disease and diabetes. *Current Cardiovascular Risk Reports*, 5(1), 7–9. doi:10.1007/s12170-010-0137-1
- Wang, C., O'Neill, S. M., Rothrock, N., Gramling, R., Sen, A., Acheson, L. S., ... Ruffin, M. T. (2009). Comparison of risk perceptions and beliefs across common chronic diseases. *Preventive Medicine*, 48(2), 197–202. doi:10.1016/j.ypmed.2008.11.008

Suggested Reading

- Köblitz, A. R., Magnan, R. E., McCaul, K. D., Dillard, A. J., O'Neill, H. K., & Crosby, R. (2009). Smokers' thoughts and worries: A study using ecological momentary assessment. *Health Psychology*, 28, 484–492.
- McCaul, K. D., Magnan, R. E., & Dillard, A. (2009). Understanding and communicating about cancer risk. In S. M. Miller, D. J. Bowen, R. T. Croyle, & J. Rowland (Eds.), *Handbook of behavioral science and cancer*. Washington, DC: American Psychological Association.
- Portnoy, D. B., Ferrer, R. A., Bergman, H. E., & Klein, W. M. P. (2014). Changing deliberative and affective responses to health risk: A meta-analysis. *Health Psychology Review*, 8, 296–318. doi:10.1080/17437199.2013.798829
- Shiloh, S., Wade, C. H., Roberts, J. S., Alford, S. H., & Biesecker, B. B. (2013). Risk perceptions and worry about common diseases: A between- and within-subjects examination. *Psychology & Health*, 28, 434–449. doi:10.1080/08870446.2012.737464

Ecological Momentary Assessment for the Psychosocial Study of Health

Doerte U. Junghaenel¹ and Arthur A. Stone^{1,2}

¹*USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA*

²*Department of Psychology, Dana and David Dornsife College of Letters Arts and Sciences, University of Southern California, Los Angeles, CA, USA*

Measuring behaviors and subjective experiences via self-report is ubiquitous in health psychology and behavioral medicine. Traditional approaches to self-report have typically asked individuals to recall and summarize their experiences over a specific span of time (e.g., weeks or months). Assessments with briefer recall periods have also been used in laboratory or clinic settings in conjunction with study designs that maximize experimental control and facilitate standardized data collection. Over the past several decades, however, the use of momentary assessments has gained momentum and has moved the study of psychosocial factors and health behaviors from the clinic and laboratory into people's real-life environments. These momentary approaches fall under the framework of ecological momentary assessment (EMA; e.g., Shiffman, Stone, & Hufford, 2008; Stone, Shiffman, Atienza, & Nebeling, 2007). Here, we provide an overview of the concept and characteristics of EMA methods followed by current examples of EMA applications for the psychosocial study of health. We conclude this chapter with an outline of promising new future platforms for EMA applications in health psychology.

Defining Characteristics of EMA

The four defining features of EMA are (a) assessments of individuals' current states and behaviors to reduce bias in recall and memory construction, (b) data collection in people's natural habitat to maximize ecological validity, (c) careful attention paid to the timing (schedule) of assessments, and (d) collection of multiple assessments over time to capture the dynamic

interplay between various states and contexts that shape people's experience (Shiffman et al., 2008; Stone et al., 2007). It is important to note that EMA is not a single methodology but consists of a variety of measurement techniques that arose from diverse traditions. These traditions include written diaries, self-monitoring, experience sampling, behavioral/activity monitoring, and physiological ambulatory monitoring; the first three are being more central to the refinement of current EMA methods (Shiffman et al., 2008; Stone et al., 2007).

Written diaries are probably the oldest historical root for the development of EMA methods (Verbrugge, 1980) with formats ranging from open-ended narratives to standardized daily assessments. At times, diary assessments were also combined with measurement of physiological indices, such as heart rate or blood pressure. Self-monitoring approaches to subjective states and behaviors have also been influential in the development of EMA. Due to their focus on specific events, self-monitoring approaches did not concentrate on aspects of sampling or data capture outside of the specified target area (Shiffman et al., 2008).

The experience sampling method (ESM) is another term that describes momentary data collection of experiences, and it predated the development of EMA. The term was originally coined by Csikszentmihalyi and Larson (1987), who pioneered the real-time assessment of individuals' subjective states and experiences. ESM was developed to obtain a characteristic set of current experiences by random, signal-contingent assessments as participants went about their daily lives (Hektner, Schmidt, & Csikszentmihalyi, 2007). This was done by having participants' complete diary assessments repeatedly throughout the day.

EMA expanded the scope and more thoroughly defined the tenets of the sampling approaches for within-day monitoring (the four features mentioned above). Furthermore, the principles of EMA are not limited to self-reports of experiences, but may be applied to all manner of ambulatory monitoring tasks, for example, the assessment of ambulatory blood pressure and heart rate. Thus, in some respects EMA is a broader concept than ESM, and it has been widely adopted in the behavioral and medical sciences. Furthermore, there are "guidelines" for presenting the results of EMA studies (Stone & Shiffman, 2002), which serve as a useful evaluation standard; for example, the guidelines suggest considerable details be presented about the sampling schedules and compliance rates.

Advantages of EMA Over Traditional Recall Assessments

In this section, we describe some of the most prominent advantages EMA methods offer over traditional retrospective assessments.

A key feature of EMA is the collection of data from participants in their natural environment. Many, if not most, behaviors and experiences are context dependent, and there is growing concern that findings obtained in laboratory or clinic settings do not adequately capture people's real-world experiences. According to Brunswik (1956), data collection in people's natural habitats increases the ecological validity of assessments and generalizability of study findings. In other words, in order to achieve representativeness of individuals' experiences or behaviors, one has to carefully sample them in their everyday or natural contexts (Shiffman et al., 2008). In addition, studying participants in daily life can reveal important contextual information, which is usually not available in many traditional self-report instruments or interview assessments, and this information can help with the interpretation of subjective experiences and behaviors.

EMA methods advanced, in part, to circumvent the limitations inherent in retrospective recall. For many decades, research has relied heavily on recall to glean information about

people's psychosocial states and behaviors. It has been widely shown that retrospective reporting, however, is susceptible to a variety of biases and heuristics that can compromise its accuracy. Several possible sources of recall bias are noteworthy. For example, people are more likely to remember experiences that occurred closest in proximity (recency effect) and experiences that are in line with their current affective state (mood-congruency effect; see Stone et al., 2007).

The longitudinal nature of EMA methodology is particularly suited for the study of temporal sequences and dynamic processes as they unfold over time. Many content areas in health psychology are concerned with change in people's subjective experiences, naturally occurring or resulting from treatment. Understanding the dynamic interplay between affective, cognitive, and behavioral factors over a short period of time is, therefore, particularly relevant. Cross-sectional and prospective designs can only provide one or a limited number of snapshots, whereas EMA's dense sampling formats can enable a fine-grained and nuanced perspective (Shiffman et al., 2008).

Analyses of contextual relations are frequently of a cross-sectional nature because they examine the co-occurrence of situations and experiences, and not their sequential relationship, even in longitudinal data collection designs (Shiffman et al., 2008). Moreover, many traditional symptom questionnaires or clinical interviews do not assess context directly. For example, affective symptoms are usually assessed in terms of their frequency or intensity over a certain timeframe, but not with regard to specific contexts (Ebner-Priemer & Trull, 2009). EMA studies allow not only the establishment of a linkage between experiences and situations but also the disentangling of context-specific antecedents and consequences of individuals' experiences.

Another important consideration with regard to situational factors is the fact that context does not only exclusively refer to a specific situation or environment but may also embody another individual (e.g., a spouse or family member). Most EMA designs focus on a single person; however, it is possible to conduct dyadic sampling when it is of interest to determine how one person's feelings and actions impact the other (Laurenceau & Bolger, 2005).

Measurement Considerations in EMA

There are two key measurement concerns for the implementation of EMA: reactivity and compliance.

Reactivity can be defined as the potential for experience and behavior to be altered by the act of assessing it (Shiffman et al., 2008). In EMA, it is plausible that the intensive sampling methodology (along with auditory or visual prompts to complete assessments) could alter people's natural environment and their experiences by shifting their focus to the phenomena under study. To date, however, empirical evidence provides little support for measurement reactivity in EMA (e.g., Hufford & Shiffman, 2002).

Although the intensive sampling methods of EMA approaches offer the opportunity to obtain a fine-grained perspective of people's experiences and natural contexts, the completion of repeated assessments in daily life places a burden on study participants. Compliance with EMA assessments has, therefore, been a much-debated topic in the literature. Earlier approaches using written diaries have highlighted the issue that repeated sampling alone may not automatically guarantee momentary or real-world data collection. For instance, studies using paper diaries usually inferred compliance based on participants' date and time entries that were written on the diaries. However, studies have found that forward and backfilling of diary entries is quite common (Shiffman et al., 2008). In other words, participants may note that they

completed a report at a given time but may have actually completed their ratings after the fact or in advance of the specified time (Stone, Shiffman, Schwartz, Broderick, & Hufford, 2003). Another important piece of evidence concerning the authenticity of people's accounts pertains to treatment adherence, such as medication taking. Research has shown that compliance with a medical regimen as indicated by participants in written diaries does not necessarily correspond with adherence tracking performed by electronic devices. In fact, participants overstated their compliance compared with what was objectively recorded by the devices (Hufford, 2007). Current EMA data collection modalities enable the verification of when assessments were completed through electronic date and time stamping of entries.

Limitations of EMA Studies

Given the intensity of many EMA sampling schedules, it is not surprising that EMA studies are very demanding, both on participants and investigators. Participants must be willing to have their daily activities interrupted to complete assessments that can be disruptive and annoying (though many studies report excellent compliance). This raises the concern that only certain sorts of people will volunteer for and be able to complete EMA studies; moreover, it is likely that some professions (e.g., surgeons, bus drivers) are simply not capable of participating in real-time sampling. Thus, the demands of EMA may raise a threat to the generalizability of the findings, raising the possibility of selection bias. End-of-day diaries and day reconstruction methods (Kahneman, Krueger, Schkade, Schwarz, & Stone, 2004) provide alternatives that are less demanding; however, they also yield much less information about the day and are prone to some recall bias.

EMA Sampling Approaches

EMA sampling protocols can be broadly characterized as event contingent or time contingent, the latter including random assessment scheduling strategies and schedules with regularly spaced or fixed assessment intervals (Shiffman, 2007). The selection of a particular sampling scheme depends on what is being studied.

Event-contingent EMA methods are usually employed when the construct of interest is a concrete episode or behavior. Examples of these types of episodes in health psychology are physical activities like exercise, medication taking, pain flares, panic episodes, smoking, stressful events, and interpersonal interactions. Assessments are completed in response to a specific event, and it is usually the research participant who reports on the occurrence that may then trigger subsequent questions regarding the experience. When choosing an event-based approach, there are several considerations. First, what constitutes a specific event or episode needs to be clearly defined and communicated between researcher and participant. Second, the researcher needs to decide when a specific event will be recorded. Third, the usual frequency of an event should be taken into account when designing a sampling protocol. If the frequency of an event is very high, it may be too burdensome and unrealistic for participants to record an episode. If the frequency is very low, sampling needs to be conducted over a larger time span, and participants need to remember the recording protocol. Finally, verification of the authenticity of the event occurrence is difficult to establish since this approach often relies upon the participant reporting an episode (see Shiffman, 2007).

Time-based EMA sampling approaches are frequently implemented when researching phenomena that vary continuously over time and cannot be compartmentalized into discrete episodes, such as mood states and symptomatology (Shiffman et al., 2008). Time-based schedules vary in frequency and timing (Delespaul, 1995) but can broadly be divided into random and fixed interval assessments. The frequency of assessments should be based on the research questions. Random schedules usually deliver assessments at variable times throughout the day. These are the most commonly used schedules in EMA because they can provide a representative picture of respondents' environments, symptoms, and experiences, whereas all other schedules may not be truly representative of everyday life. Assessments can be programmed completely at random or randomized within strata of the day. In the latter, assessments can be randomly administered within certain predefined blocks of the day, such as in the morning and in the evening. For certain purposes, it may be advantageous to use a combination of random and fixed interval sampling. For example, if one is interested in studying the antecedents of panic attacks or the mood benefits of exercising, a combination of the two schedules may be preferable.

Success and Current Applications of EMA

The prominence and success of EMA is evident in numerous reviews and books that have appeared on the topic across the years. At this point, EMA has become a stand-alone acronym that is recognized in the fields of health psychology and psychosomatic and behavioral medicine. In addition to several books and chapters (e.g., see Hektner et al., 2007; Stone et al., 2007), reviews focus on the promise and challenges of EMA methods (Scollon, Kim-Prieto, & Diener, 2003) and applications of EMA to behavioral medicine (Robbins & Kubiak, 2014). We refer the reader to these sources for more detail than can be discussed in this chapter. In the following sections, we review recent examples of EMA applications that are of particular relevance for health psychology.

The ability of EMA methods to capture people's daily life as it unfolds has opened the door to the study of social behavior and interpersonal processes from the perspective of a real-time dyadic system. Social support, marital satisfaction, and dyadic adjustment, which have a long-standing tradition in health psychology due to their potent associations with health and illness, can be studied within this framework. Up until recently, research has often relied on patients' retrospective accounts about their interpersonal environment and relationship characteristics. This has provided an important yet incomplete glimpse into the complex dynamics of a person's social network for several reasons. First, studying interpersonal dynamics through the lens of an individual may not reflect how other dyad members view them and their behavior. This is of particular relevance when studying social support and behavioral responses toward the patient. Second, perceptions and behaviors of dyad and social network members mutually influence each other, and marital and family relationship experiences may change over time. In order to reveal an accurate and complete picture of social dynamics, it is therefore essential to study all network members of interest and examine how interpersonal processes unfold over time as couples or dyads go about their daily lives.

In clinical assessment, patients' medical progress has traditionally been assessed on a weekly or monthly basis, which requires patients to recall their symptoms and medical progress. However, as noted above, retrospective self-report measures are strongly influenced by current states and salient events. In addition, assessments conducted in the clinic setting limit the ecological validity of patients' self-reported symptoms. The successful integration of EMA

methodology into routine care is still in its infancy. However, real-time symptom assessments and patient tracking offer promising avenues to augment clinical assessment and can be suited to fit the needs of medical practice.

A logical extension to EMA data collection approaches has been the development of Ecological Momentary Intervention (EMI; for review see Heron & Smyth, 2010). Early implementations of EMA in clinical practice were primarily geared toward its use as an assessment tool. Bridging the gap between therapeutic consultations in the medical office and patients' experiences in their natural settings has not been an easy task in psychological treatment (Shiffman et al., 2008). However, clinicians have long aimed to effectively integrate treatment into patients' daily lives to facilitate practice and maintenance of skills between sessions (Heron & Smyth, 2010). EMI encompasses a variety of treatment approaches that mirror EMA methodology—delivery of treatments to people in real time in their natural environments (see Heron & Smyth, 2010).

The opportunities and benefits of introducing EMA methods into treatment delivery are vast. For one, the reach of EMI extends into patients' everyday lives. This means that the benefits of treatment are likely to be optimized. Second, the availability of treatment delivery in patients' natural contexts makes it possible for patients to integrate new skills and tools in their personally relevant contexts and settings. Third, practice and maintenance of new skills and behaviors is more readily achieved (Heron & Smyth, 2010).

Patient-reported outcomes (PROs) serve an increasingly important role in treatment evaluation and ultimately in health economics and health policy (Robbins & Kubiak, 2014). PROs refer to a variety of self-reported symptoms and behaviors pertaining to health and well-being, including pain, fatigue, psychological distress, and quality of life. The major US regulatory agency for medicine and devices, the Food and Drug Administration (FDA), issued "preliminary" guidance to the pharmaceutical industry emphasizing that real-time data collection, including EMA and end-of-day diaries, is preferable because PROs based on retrospective self-reports over long periods may have compromised validity due to retrospective reporting biases (FDA, 2009). To date, interventions in health psychology and behavioral medicine are slowly beginning to adopt EMA methods for the measurement of PROs. A recent example is a randomized controlled trial on mindfulness training in adults vulnerable to depression (Geschwind, Peeters, Drukker, van Os, & Wichers, 2011). Momentary sampling of positive emotions and of appraisal of pleasant daily activities 6 days before and after the intervention was conducted. The authors found that mindfulness-based cognitive therapy was associated with enhanced experience of positive emotions as well as greater appreciation and responsiveness to pleasant daily activities. Although studies like these are making their inroads into the clinical setting, much more progress needs to be made to fully integrate EMA methods into the standard toolkit for evaluating PROs in health psychology interventions.

Future Directions for EMA Methods

The versatility and richness of EMA methods have found their way into numerous settings and content areas. Technological advances are enabling new and flexible platforms for expanding the reach of EMA data collection. In particular, mobile phone applications for EMA have grown very rapidly. There are several new smartphone-linked capabilities that may be the future of EMA applications—combining self-report with socio-contextual, physical, and physiological momentary assessment (frequently referred to as mHealth methodologies).

Clearly, the future of EMA methods seems bright. Pending the appropriate implementation and validation of these new technologies, we believe that the effective and evidence-based integration of EMA technology into research and clinical practice will successfully propel the psychosocial study of health into the future.

Author Biographies

Dr. Doerte U. Junghaenel received her doctoral degree in social and health psychology from Stony Brook University. She is a research scientist at the USC Dornsife Centers for Self-Report Science and for Economic and Social Research at the University of Southern California. Her research focuses on the multimodal measurement of patient-reported outcomes, the examination of individual differences in symptom experience, and the study of interpersonal dynamics and how they may relate to favorable adaptation in chronic pain.

Dr. Arthur A. Stone is professor of psychology and director of the USC Dornsife Center for Self-Report Science at the University of Southern California. He was distinguished professor of psychiatry and psychology at Stony Brook University. Dr. Stone has been involved with the development of self-reports to measure end-of-day and within-day phenomena that led to EMA. More recently, he has been involved with the development of alternative methods for capturing daily experience, including the Day Reconstruction Method.

References

- Brunswik, E. (1956). *Perception and the representative design of psychological experiments*. Berkeley: University of California Press.
- Csikszentmihalyi, M., & Larson, R. (1987). Validity and reliability of the experience-sampling method. *Journal of Nervous and Mental Disease*, 175(9), 526–536. doi:10.1097/00005053-198709000-00004
- Delespaul, P. (1995). *Assessing schizophrenia in daily life: The experience sampling method*. Maastricht: Maastricht University Press.
- Ebner-Priemer, U. W., & Trull, T. J. (2009). Ecological momentary assessment of mood disorders and mood dysregulation. *Psychological Assessment*, 21(4), 463–475. doi:10.1037/a0017075
- FDA. (2009). Guidance for the industry. Patient-reported outcome measures: Use in medical product development to support labeling claims. Retrived from <http://www.fda.gov>
- Geschwind, N., Peeters, F., Drukker, M., van Os, J., & Wichers, M. (2011). Mindfulness training increases momentary positive emotions and reward experience in adults vulnerable to depression: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 79(5), 618–628. doi:10.1037/a0024595
- Hektner, J. M., Schmidt, J. A., & Csikszentmihalyi, M. (2007). *Experience sampling method: Measuring the quality of every day life*. London: Sage.
- Heron, K. E., & Smyth, J. M. (2010). Ecological momentary interventions: Incorporating mobile technology into psychosocial and health behavior treatments. *British Journal of Health Psychology*, 15(Pt 1), 1–39. doi:10.1348/135910709X466063
- Hufford, M. R. (2007). Special methodological challenges and opportunities in ecological momentary assessment. In A. A. Stone, S. Shiffman, A. A. Atienza, & L. Nebeling (Eds.), *The science of real-time data capture: Self-reports in health research* (pp. 54–75). New York, NY: Oxford University Press.
- Hufford, M. R., & Shiffman, S. (2002). Methodological issues affecting the value of patient-reported outcomes data. *Expert Review of Pharmacoeconomics & Outcomes Research*, 2(2), 119–128. doi:10.1586/14737167.2.2.119

- Kahneman, D., Krueger, A. B., Schkade, D. A., Schwarz, N., & Stone, A. A. (2004). A survey method for characterizing daily life experience: The day reconstruction method. *Science*, 306(5702), 1776–1780. doi:10.1126/science.1103572
- Laurenceau, J. P., & Bolger, N. (2005). Using diary methods to study marital and family processes. *Journal of Family Psychology*, 19(1), 86–97. doi:10.1037/0893-3200.19.1.86
- Robbins, M. L., & Kubiak, T. (2014). Ecological momentary assessment in behavioral medicine: Research and practice. In D. I. Mostofsky (Ed.), *The handbook of behavioral medicine* (pp. 429–446). Chichester, West Sussex: Wiley.
- Scollon, C. N., Kim-Prieto, C., & Diener, E. (2003). Experience sampling: Promises and pitfalls, strengths and weaknesses. *Journal of Happiness Studies*, 4, 5–34.
- Shiffman, S. (2007). Designing protocols for ecological momentary assessment. In A. A. Stone, S. Shiffman, A. A. Atienza, & L. Nebeling (Eds.), *The science of real-time data capture: Self-reports in health research* (pp. 27–53). New York, NY: Oxford University Press.
- Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological momentary assessment. *Annual Review of Clinical Psychology*, 4, 1–32. doi:10.1146/annurev.clinpsy.3.022806.091415
- Stone, A. A., & Shiffman, S. (2002). Capturing momentary, self-report data: A proposal for reporting guidelines. *Annals of Behavioral Medicine*, 24(3), 236–243.
- Stone, A. A., Shiffman, S., Atienza, A. A., & Nebeling, L. (2007). *The science of real-time data capture: Self-reports in health research*. New York, NY: Oxford University Press.
- Stone, A. A., Shiffman, S., Schwartz, J. E., Broderick, J. E., & Hufford, M. R. (2003). Patient compliance with paper and electronic diaries. *Controlled Clinical Trials*, 24(2), 182–199.
- Verbrugge, L. M. (1980). Health diaries. *Medical Care*, 18(1), 73–95.

Suggested Reading

- Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: Capturing life as it is lived. *Annual Review of Psychology*, 54, 579–616. doi:10.1146/annurev.psych.54.101601.145030
- Bolger, N., & Laurenceau, J. P. (2013). *Intensive longitudinal methods: An introduction to diary and experience sampling research*. New York, NY: The Guilford Press.
- Kumar, S., Nilsen, W. J., Abernethy, A., Atienza, A., Patrick, K., Pavel, M., ... Swendeman, D. (2013). Mobile health technology evaluation: The mHealth evidence workshop. *American Journal of Preventive Medicine*, 45(2), 228–236.
- Schneider, S., & Stone, A. A. (2016). Ambulatory and diary methods can facilitate the measurement of patient-reported outcomes. *Quality of Life Research*, 25(3), 497–506.
- Schwarz, N. (2007). Retrospective and concurrent self-reports: The rationale for real-time data capture. In A. A. Stone, S. Schiffman, A. A. Atienza, & L. Nebeling (Eds.), *The science of real-time data capture: Self-reports in health research* (pp. 11–26). New York, NY: Oxford University Press.

The Effects of Health-Related Policy

Robert García and Steve Sussman

Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

The role of policy often is to prevent an event from occurring that could do harm. This is especially clear when looking at health policy. The twentieth century saw many advances in the health status of much of the world's population due to the increased understanding of what effects health. We can think of many examples of how health policy improved health: tobacco product regulation, workplace safety, clean-air regulations, seat belt laws, child safety, sanitation, food handling, and removal of lead-based paints, as examples. While these policies have been effective at improving our health status, there are still other areas that need to be addressed (e.g., Should sales of unsafe tobacco products be permitted at all? Is the cost-effectiveness of removing toxins from the workplace too low?). Further, with ever changing technology it is important to adapt our policies in a similar rapid and effective fashion. We can learn important lessons from the successful history of tobacco policy to confront the new challenges to our health. In this chapter, we will examine how tobacco policy led to the change in societal norms and eventually the population's health.

A Brief History on the Growth of Big Tobacco

Many people have heard of the role of tobacco in the growth of the American colonies, both the English and Spanish domains. However, few know that up until around the mid-nineteenth century, those who smoked tobacco would not inhale (Proctor, 2011). The curing process that allowed tobacco to become milder and therefore inhaled was discovered by accident, when the fire being used to cure tobacco leaves died out and the workers quickly threw on charcoal to reignite the fire (Kluger, 1997; Proctor, 2011). The charcoal burned hotter and therefore provided a different curing process and mildness allowing users to inhale (Proctor, 2011). Along with creating a milder product, it also decreased costs as this method used a small fraction of fuel compared with the traditional wood fires (Kluger, 1997). Another major innovation that helped to spread the use of tobacco is automation. Specifically, the Bonsack

(cigarette rolling) machine invented in 1880 allowed for manufacturers to produce 210 cigarettes per minute (Kluger, 1997; Proctor, 2011). This invention could replace 500 individual employees (Proctor, 2011). Modern machines can produce nearly 20,000 cigarettes per minute (Proctor, 2011). The mechanization process allowed cigarette manufacturers to sell them cheaply further spreading nicotine addiction for pennies while making large profits.

Perhaps the largest contributor to the proliferation of tobacco use was its industry's agile and effective application of marketing. Over the decades, the tobacco companies have been able to respond accordingly to a changing world. One example often cited is how tobacco companies were able to capitalize on war. During the World Wars, the tobacco companies were able to use the war and home front to increase consumption of cigarettes. After World War I the consumption of cigarettes tripled (Brandt, 2007). During the Second World War, the tobacco companies were required to turnover a portion of their profits toward the war effort. They would use this to their advantage and market their products as a patriotic duty for American society. Cigarette consumption after that war doubled (Proctor, 2011). Tobacco companies continued to use current events in society to increase their reach. Other examples include the suffrage and civil rights movements in which cigarettes were marketed as an equalizer for marginalized populations (Brandt, 2007; Proctor, 2011). With the increasing use of cigarettes, the evidence of their detrimental effects on health became clearer that would eventually lead to more regulation of the industry. As cigarettes fell out of favor and became more regulated in most developed countries, the industry turned its attention to lower- and middle-income countries who welcomed the manufacturing jobs (Kluger, 1997; Proctor, 2011).

The Changing Tide: The Beginning of Tobacco Regulation

Two publications helped to start the regulation of tobacco products in the United States. First was an article in the December 1952 issue of *Reader's Digest* by Roy Norr titled "Cancer by the Carton" that was one of the first main stream, lay publications to link smoking to cancer (Brandt, 2007; Norr, 1952; Proctor, 2011). A little over a decade after this, the US Surgeon General released their first report about smoking and its effects on health in 1964. At that time 43% of the US adult population were smokers (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). Most of the policies that arose after the initial report focused solely on providing the consumers information on tobacco products and their consequences (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). To government officials at that time, it was up to the public to change their behavior, and the government was responsible to provide information about their behaviors and related consequences (Dinno & Glantz, 2009; Glantz, Barnes, Bero, Hanauer, & Slade, 1995; U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). The tobacco companies strongly refuted these claims (e.g., of lung cancer and cardiovascular smoking-related diseases) and conducted their own "science" to contradict the growing scientific consensus (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014).

The tobacco industry turned to marketing again, this time to maintain their consumers. While more people became aware of the risks carried with smoking, there was still a lag in behavior change due to the marketing campaigns that created doubt in the US public (Proctor, 2011; U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). Two years after the initial 1964 report, only 40% of the US public considered smoking a major cause of cancer compared with 90% in 1990 (U.S. Department of

Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). Over the next few decades, the information provided by governmental health agencies was associated with change in tobacco use prevalence. To combat declining prevalence the tobacco companies changed their advertising to start including terms that gave consumers a perception of health (Brandt, 2007; Glantz et al., 1995). Such terms included “light” and “mild” and were meant to help combat the negative health effects of cigarette ingredients such as tar (Grier & Kumanyika, 2010; Iglesias-Rios & Parascandola, 2013). The government’s approach to only providing information was limited. More policy action was needed to address the increasing rates of consumption in populations such as racial/ethnic minorities.

Marketing Regulation

With the increasing awareness of the health risks of tobacco products, there was an increase in the regulation of tobacco advertising. Consequently, the industry over the next decades would dramatically increase their advertising expenditures in unregulated media (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). For example, after the banning of television ads, the tobacco industry increased their advertising in magazines. While the ban on broadcast marketing did lead to a decrease in smoking behaviors, research has shown that limited marketing bans have limited impacts (Wakefield et al., 2002). Expenditures on magazine advertising increased from \$62 to \$152 million after the television ban (Proctor, 2011; U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). Further, tobacco companies used more imagery that promoted perceptions of healthier and cooler products, which were effective among youth and racial/ethnic minorities (Dauphinee, Doxey, Schleicher, Fortmann, & Henriksen, 2013; Ribisl et al., 2017; Siahpush, Jones, Singh, Timsina, & Martin, 2010). The lack of a complete policy on tobacco marketing assisted in the creation of large disparities in smoking behaviors among different racial/ethnic groups. African Americans and Native Americans consistently have a higher prevalence of smoking compared with non-Hispanic Whites (Centers for Disease Control and Prevention [CDC], 2005, 2014). As regulation continued to evolve, so did the tobacco industry’s effort to maintain their profits and market share.

In 1998, there were further advertising and promotional bans applied to the tobacco industry with the implementation of the Master Settlement Agreement (MSA). The MSA bans on advertising included the use of cartoon characters, billboards, public transit placements, product giveaways, and the branding of merchandise (Loomis, Farrelly, Nonnemaker, & Mann, 2006; Wakefield et al., 2002). In the decade after the MSA, tobacco companies retail environment advertising expenditures increased from \$6.7 to \$9.9 billion (Widome, Brock, Noble, & Forster, 2013). However, the tobacco industry advanced exposure to marketing and promotional materials in the retail environment, including convenience stores, which are frequented by youth. The ability to further control youth uptake in smoking was hampered by marketing in the retail environment and, likely, accounts in part to a plateau in the reduction of cigarette smoking since the late 1990s (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). The bans on marketing, however, did help to denormalize smoking in US society and brand it as unacceptable behavior (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). This denormalization of smoking helped governmental agencies in the implementation of other policies that dramatically decreased smoking and other tobacco use in the United States. Another benefit from the MSA was that it funded tobacco control programs at the state level.

Taxation: Beyond Increasing Costs

The most dramatic decrease in smoking behavior happened after the doubling of the federal tax on cigarettes in the early 1980s (Chaloupka, Cummings, Morley, & Horan, 2002; U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). Further, states with higher tax rates for cigarettes also demonstrated lower smoking rates (Carpenter & Cook, 2008; Meier & Licari, 1997). The decrease of smoking behavior, however, cannot be attributed solely to higher taxes but also to the funding those taxes provided to create new and effective tobacco control programs at all levels of government (Sutfin, Szykman, & Moore, 2008; U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014).

The revenue from the taxes made it possible to create effective anti-tobacco use advertisements and other programming relevant to different populations (Sutfin et al., 2008). Many of these campaigns targeted youth since the vast majority of smokers begin before the age of 26 (CDC, 2014). The youth-smoking prevalence has continued to decrease in recent years with 10.8% of youth reporting being current smokers in 2016 compared with 34.8% in 1999 (CDC, 2016). While financial investments in anti-tobacco marketing still are not at the same level as tobacco industry marketing, this infusion of revenue to state and local programs helped these and other grassroots programs gain strong footing to combat tobacco's influence on our youth.

The New Federal Mandate on Tobacco Regulation

By the time of the 50th anniversary report in 2014 the overall smoking rate in the United States had decreased to 19% (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014). The prevalence of smoking is at historical lows, and with new regulatory authority of the Food and Drug Administration (FDA) under the Family Smoking and Prevention Tobacco Control Act, there is promise that there can be further decreases in smoking and the burden of tobacco-related disease among the whole population. Efforts to give the FDA regulatory authority occurred as early as 1965, but they did not receive this authority until 2009 (U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2014).

The federal FDA recently released its final "deeming rule," which extends the agency's regulatory authority to all tobacco products, including electronic smoking devices, cigars, pipe tobacco, and hookah tobacco. The deeming rule extends several provisions of the federal Tobacco Control Act to these new tobacco products. For example, these products are now subject to the federal prohibition on sales to minors, the federal prohibition on free sampling, federal warning label requirements, and the requirement that tobacco manufacturers register with the FDA and seek the agency's review of new tobacco products.

Moving Forward: What Can We Learn From the Fight Against Tobacco

With the growing prevalence of chronic disease and the burdens of such diseases falling more on vulnerable populations such as racial/ethnic minorities, lower SES communities, and youth, it is important that we apply the lessons from tobacco policies and apply these to other areas. For example, one such area includes policies addressing sugar consumption. Many

initiatives have been undertaken to levy a tax on sugar-sweetened beverages, but the sugar/food industry has been able to use marketing, just as with tobacco, to defeat these measures. Passage of such measures could have the similar effect of tobacco taxes in providing funds to create campaigns to combat the influence of that industry on youth.

There is also a recognition of the effect of advertising on the consumption of unhealthy food among children and adults (Harris, Haraghey, Lodolce, & Semenza, 2018; The Federal Trade Commission, 2012). Similar to tobacco there is evidence on the need to reign in the manipulative marketing tactics such as depicting healthy activities (e.g., playing sports) in the promotion of unhealthy foods and beverages (Harris et al., 2018). Recent literature also shows the existence of disparities between racial/ethnic groups. Children of Hispanic/Latino descent, for instance, report lower usage of the Internet but have higher exposure to websites promoting food and beverages (Hyary & Harris, 2017). After the Federal Trade Commission's (FTC) initial report on the advertising of unhealthy food to children, the industry created the self-regulated Children's Food and Beverage Advertising Initiative (Powell, Harris, & Fox, 2013). The promotion of healthy lifestyle messaging is the goal of this industry-led initiative. The tobacco industry had similar approaches in an effort to prevent further regulation, which in the end did little to prevent youth uptake of tobacco products but did help in their public image (Proctor, 2011). Eventually research and programs like the Truth Initiative countered these efforts by the tobacco industry and helped to decrease the uptake in tobacco use behaviors.

It is important for public health professionals to look at how the government and scientific efforts to address smoking were responded to by the tobacco industry—and how to limit such unhealthy counter-responses. Some research has attempted to counter unhealthy food and beverage marketing by creating similar promotions of healthy food, but the results do not show increased interest or consumption among youth (Harris et al., 2018). Another approach will be necessary to counter unhealthy food and beverage marketing, perhaps one similar to the Truth Initiative and similar counter tobacco campaigns, where the campaigns address how consumption of unhealthy foods and beverages cannot only effect your health but also other aspects of a youth's life (e.g. social, sports, health). It may be possible to more effectively advance healthy lifestyles and decrease disease burden through policy advances such as stopping misleading advertisements, taxation of unhealthy products, and widening the net on attempts to sidestep policy.

Author Biographies

Robert García, MPH, is a doctoral candidate in the Health Behavior Research Program at the Keck School of Medicine of USC. He was a predoctoral trainee for the USC Tobacco Center of Regulatory Science for Vulnerable Populations. Robert has participated in numerous studies that use community-based approaches to cancer/tobacco prevention and control among Latinos. Robert is passionate about serving the Latino community and identifying culturally responsive practices to promote health behaviors and prevent tobacco use.

Steve Sussman, PhD, FAAHB, FAPA (PhD, UIC, 1984), is a professor of preventive medicine, psychology, and social work at the University of Southern California. He studies addictions etiology, prevention, cessation, and international translation research. He has 500+ publications. His EBP's include projects TNT, TND, and EX. He is the editor of *Evaluation & the Health Professions* (SAGE Publications). His newest text is *Substance and Behavioral Addictions: Concepts, Causes, and Cures* (Cambridge, 2017).

References

- Brandt, A. M. (2007). *The cigarette century*. New York, NY: Basic Books.
- Carpenter, C., & Cook, P. J. (2008). Cigarette taxes and youth smoking: New evidence from national, state, and local Youth Risk Behavior Surveys. *Journal of Health Economics*, 27(2), 287–299. <https://doi.org/10.1016/j.jhealeco.2007.05.008>
- Centers for Disease Control and Prevention (CDC). (2005). Cigarette smoking among adults—United States, 2004. *Morbidity and Mortality Weekly Report (MMWR)*, 54(44), 1121–1124. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/16280969>
- Centers for Disease Control and Prevention (CDC). (2014). *Tobacco product use among adults—United States, 2012–2013*. Atlanta, GA. Retrieved from <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6325a3.htm#Tab1>
- Centers for Disease Control and Prevention (CDC). (2016). Cigarette smoking among U.S. high school students at an all-time low, but e-cigarette use a concern. *CDC Online Newsroom*, June 9, 2016. Atlanta, GA: CDC. Retrieved from <https://www.cdc.gov/media/releases/2016/p0609-yrbs.html>
- Chaloupka, F. J., Cummings, K. M., Morley, C., & Horan, J. (2002). Tax, price and cigarette smoking: Evidence from the tobacco documents and implications for tobacco company marketing strategies. *Tobacco Control*, 11(Suppl. 1), i62–i72. https://doi.org/10.1136/tc.11.suppl_1.i62
- Dauphinee, A. L., Doxey, J. R., Schleicher, N. C., Fortmann, S. P., & Henriksen, L. (2013). Racial differences in cigarette brand recognition and impact on youth smoking. *BMC Public Health*, 13(1), 170. <https://doi.org/10.1186/1471-2458-13-170>
- Dinno, A., & Glantz, S. (2009). Tobacco control policies are egalitarian: A vulnerabilities perspective on clean indoor air laws, cigarette prices, and tobacco use disparities. *Social Science & Medicine* (1982), 68(8), 1439–1447. <https://doi.org/10.1016/j.socscimed.2009.02.003>
- Glantz, S. A., Barnes, D. E., Bero, L., Hanauer, P., & Slade, J. (1995). Looking through a keyhole at the tobacco industry. *JAMA*, 274(3), 219. <https://doi.org/10.1001/jama.1995.03530030039032>
- Grier, S. A., & Kumanyika, S. (2010). Targeted marketing and public health. *Annual Review of Public Health*, 31(1), 349–369. <https://doi.org/10.1146/annurev.publhealth.012809.103607>
- Harris, J. L., Haraghey, K. S., Lodolce, M., & Semenza, N. L. (2018). Teaching children about good health? Halo effects in child-directed advertisements for unhealthy food. *Pediatric Obesity*, 13(4), 256–264. <https://doi.org/10.1111/ijpo.12257>
- Hyary, M., & Harris, J. L. (2017). Hispanic youth visits to food and beverage company websites. *Health Equity*, 1(1), 134–138. <https://doi.org/10.1089/heq.2016.0026>
- Iglesias-Rios, L., & Parascandola, M. (2013). A historical review of R. J. Reynolds' strategies for marketing to hispanics in the United States. *American Journal of Public Health*, 103(5), e15–e27. <https://doi.org/10.2105/AJPH.2013.301256>
- Kluger, R. (1997). *Ashes to Ashes: America's hundred-year cigarette war. The public health, and the unabashed triumph of Philip Morris* (1st ed.). New York, NY: Vintage Books.
- Loomis, B. R., Farrelly, M. C., Nonnemaker, J. M., & Mann, N. H. (2006). Point of purchase cigarette promotions before and after the Master Settlement Agreement: Exploring retail scanner data. *Tobacco Control*, 15, 140–142. <https://doi.org/10.1136/tc.2005.011262>
- Meier, K. J., & Licari, M. J. (1997). The effect of cigarette taxes on cigarette consumption, 1955 through 1994. *American Journal of Public Health*, 87(7), 1126–1130. <https://doi.org/10.2105/AJPH.87.7.1126>
- Norr, R. (1952, December). Cancer by the carton. *Reader's Digest*, pp. 7–8.
- Powell, L. M., Harris, J. L., & Fox, T. (2013). Food marketing expenditures aimed at youth: Putting the numbers in context. *American Journal of Preventive Medicine*, 45(4), 453–461. <https://doi.org/10.1016/J.AMEPRE.2013.06.003>
- Proctor, R. (2011). *Golden holocaust origins of the cigarette catastrophe and the case for abolition*. Oakland, CA: University of California Press.

- Ribisl, K. M., D'Angelo, H., Feld, A. L., Schleicher, N. C., Golden, S., Luke, D. A., & Henriksen, L. (2017). Disparities in tobacco marketing and product availability at the point of sale: Results of a national study. *Preventive Medicine*. <https://doi.org/10.1016/j.ypmed.2017.04.010>
- Siahpush, M., Jones, P. R., Singh, G. K., Timsina, L. R., & Martin, J. (2010). The association of tobacco marketing with median income and racial/ethnic characteristics of neighbourhoods in Omaha, Nebraska. *Tobacco Control*, 19(3), 256–258. <https://doi.org/10.1136/tc.2009.032185>
- Sutfin, E. L., Szykman, L. R., & Moore, M. C. (2008). Adolescents' responses to anti-tobacco advertising: Exploring the role of adolescents' smoking status and advertisement theme. *Journal of Health Communication*, 13(5), 480–500. <https://doi.org/10.1080/10810730802198961>
- The Federal Trade Commission. (2012). *A review of food marketing to children and adolescents follow-up report*. Washington, DC. Retrieved from <https://www.ftc.gov/sites/default/files/documents/reports/review-food-marketing-children-and-adolescents-follow-report/121221foodmarketingreport.pdf>
- U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General. (2014). *The health consequences of smoking-50 years of progress: A report of the Surgeon General*. Atlanta, GA: Author.
- Wakefield, M. A., Terry-McElrath, Y. M., Chaloupka, F. J., Barker, D. C., Slater, S. J., Clark, P. I., & Giovino, G. A. (2002). Tobacco industry marketing at point of purchase after the 1998 MSA billboard advertising ban. *American Journal of Public Health*, 92(6), 937–940. <https://doi.org/10.2105/AJPH.92.6.937>
- Widome, R., Brock, B., Noble, P., & Forster, J. L. (2013). The relationship of neighborhood demographic characteristics to point-of-sale tobacco advertising and marketing. *Ethnicity & Health*, 18(2), 136–151. <https://doi.org/10.1080/13557858.2012.701273>

Suggested Reading

- Nestle, M. (2006). Food marketing and childhood obesity—A matter of policy. *New England Journal of Medicine*, 354(24), 2527–2529.
- Oreskes, N., & Conway, E. M. (2010). *Merchants of doubt*. New York, NY: Bloomsbury Press.
- Proctor, R. (2011). *Golden holocaust origins of the cigarette catastrophe and the case for abolition*. Oakland, CA: University of California Press.

Embodied Health

Ariel J. Mosley, Trevor James Swanson,
and Mark J. Landau

Department of Psychology, University of Kansas, Lawrence, KS, USA

Embodied health refers broadly to the study of health outcomes within the theoretical framework offered by perspectives on *embodied* or *grounded* cognition, which are increasingly influential in the cognitive sciences (e.g., Barsalou, 2008; Blascovich & Seery, 2007; Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005). These perspectives challenge the traditional assumption that mental representation and cognition can be understood in terms of abstract, symbolic information processing independent of having the particular types of bodies that we have. Instead, they propose the meanings people give to abstractions are intimately connected with their bodily (sensorimotor) states and routine interactions with the physical environment.

Because bodily states and experiences intersect with health outcomes in many diverse ways, the study of *embodied health* encompasses several interesting questions regarding the psychological processes underlying mental and physical well-being. For example, how do people “read” their bodily states to assess their health status? How do health-relevant behaviors like smoking and flossing become habitual motor routines?

This entry focuses on just one embodied process: using knowledge of bodily experiences to conceptualize health-relevant concepts. Many of these concepts are abstract because they refer to outcomes and causal relations that we cannot directly observe or experience, that are remote from our daily lives, or that we imagine occurring in the distant future, if at all. Few non-experts have a detailed mental model of, say, the biochemical processes by which ultraviolet radiation triggers malignant melanoma, or the effects of antidepressants on brain functioning. Consequently, these concepts can be difficult to understand in their own terms.

According to conceptual metaphor theory (Lakoff & Johnson, 1980), metaphor is a cognitive tool that people can use to understand (not just talk about) abstract concepts in terms of dissimilar concepts that are relatively more concrete. Metaphor supports understanding by creating a set of mental associations, called a *mapping*, between aspects of the abstract concept and corresponding aspects of the concrete concept. When people employ a metaphor to think,

the mapping enables them to draw on knowledge of the concrete concept as a framework for construing the abstract concept, even though the two concepts are unrelated at a surface level. In some (but not all) metaphors, the concrete concept is a sensorimotor state (e.g., increase in body temperature) or a familiar bodily activity (e.g., manipulating objects and containers). Researchers call these *bodily metaphors*.

Let's illustrate how bodily metaphors might shape mental models of health concepts. "I've *come a long way*," Claire says of a nasty cold, "but I'm not *out of the woods* yet." Of course, a cold is not a trip or journey in any literal sense, so why does Claire talk this way? She has difficulty grasping what a cold is and how it works. Employing the metaphor *cold recovery is a journey*, she maps colds onto an easily visualized schema for moving along a path. Now she can represent recovery as a *destination*, her persistent cough as an *obstacle*, and medication as *moving* her in the right *direction*. Colds and journeys both involve bodily experiences, but they are obviously concepts of a very different kind. That is the point: Claire capitalizes on a coherent *journey* schema to make sense of vague, complex, and occasionally threatening ideas about health.

But do people, in fact, use bodily metaphors to grasp health concepts?

Qualitative Evidence

Some evidence for the use of bodily metaphors comes from qualitative analyses of the language used by health practitioners and the general public to talk about those concepts. Susan Sontag's (1978) seminal book *Illness as Metaphor* revealed that metaphors pervaded discourse on cancer in the nineteenth and twentieth centuries. She showed that medical experts used a personification metaphor that attributes cancer agency and malevolent motives, referring to it as a growing, eating parasite, a demonic pregnancy, and an invasion of alien cells. Other metaphors portrayed cancer as a corrupting, corroding, and consuming force. Complementing Sontag's broad historical analysis, Penson, Schapira, Daniels, Chabner, and Lynch (2004) analyzed cancer patients' narratives and found that they, too, rely on concrete metaphors to communicate with doctors about the experience of having and treating cancer. Particularly common is a military metaphor that compares various aspects of cancer to analogous aspects of physical combat with a personified enemy, as reflected in phrases like "cancer *hit me hard*" and "I'm *battling* cancer."

Bodily metaphors also appear in narratives of substance addiction. Akers, Gordon, Reyna, and Severson (2013) analyzed over 2,000 online forum posts in a web-based cessation program for smokeless tobacco. They found that respondents frequently used metaphors to talk about tobacco, nicotine addiction, and substance cessation. Many of these metaphors were based on bodily experiences. For example, cessation was described as a journey to be completed, a physical battle to be won, and an escape from being physically restrained against one's will.¹

One other example will suffice this one from the domain of mental illness. Clients diagnosed with depression frequently compare depression to being spatially *down*, *low*, and *sinking* (McMullen & Conway, 2002). Other conventional metaphors compare depression to a physical weight holding one down and therapy as *easing* or *removing* that burden, or to a state of darkness (McMullen & Conway, 2002).

Qualitative evidence that linguistic metaphors pervade health discourse suggests that people use metaphors at a deeper conceptual level to represent and reason about health concepts. Yet an alternative interpretation of these findings is that people talk using conventional metaphoric

idioms, but they think about health concepts without the aid of metaphor. Therefore, we need to “peek under the hood” of language and examine whether bodily metaphors actively shape online health cognition.

Experimental Evidence

Toward this end, researchers in social and cognitive psychology have recently developed empirical strategies that can be used to test metaphor’s causal impact on information processing (Landau, Robinson, & Meier, 2014). One strategy is to expose people to a message that uses metaphoric phrases or imagery to compare an abstract concept to a superficially unrelated concrete concept—what researchers call a *metaphoric framing*. The reasoning is that if exposure to a metaphoric framing activates a metaphor in observers’ minds, it should trigger a cascade of associations entailed by that metaphor’s mapping. As a result, people should transfer emotions and cognitions associated with the framing’s concrete concept to interpret analogous aspects of the abstract concept.

A quick illustration: Jennifer hears on TV that a cold medication gives people a “bold step forward,” activating the bodily metaphor *cold recovery is a journey*. If that metaphor operates as a systematic mapping, as conceptual metaphor theory posits, then it should prompt Jennifer to pull from her knowledge of journeys to reason about corresponding aspects of colds, even those aspects of colds that are not mentioned explicitly in the original TV message (e.g., what causes colds). If the message had compared cold recovery to another concrete concept, such as evicting an annoying houseguest, or described it without metaphor, then we would not expect Jennifer’s understanding of colds to be colored by her knowledge of journeys.

Supporting studies show that even brief exposure to bodily metaphoric framings leads people to transfer emotions and cognitions associated with the relevant bodily states to interpret abstract health risks. In one representative study, Scherer, Scherer, and Fagerlin (2015) showed that exposure to various bodily metaphoric framings of the flu led participants to report stronger intentions to get vaccinated. Specifically, college students reported stronger intentions to get the flu vaccine if they read messages framing the flu as an *enemy combatant* (“an army attacking the body”), a *wild beast* attacking the body, or a *riot that revolts against the body*. What is the mechanism behind this effect? In line with our theoretical reasoning, exposure to these metaphoric framings prompted participants to transfer *emotional worry* associated with protecting their bodies from concrete hazards (getting assaulted by an enemy, dangerous animal, or angry mob) to assess how much the flu threatens their well-being. This heightened worry energized participants to take action to protect themselves.

In addition to motivating prevention behavior, bodily metaphoric framings influence decisions about which course of action to take to address health concerns. Hauser and Schwarz (2015) reasoned that in an actual state of war or physical combat, more active and aggressive actions (e.g., throwing punches) are generally seen as more appropriate than passive responses. Hence, they predicted that exposure to a war metaphoric framing of cancer (versus a non-metaphoric framing) would reduce people’s intentions to engage in so-called self-limiting cancer prevention behaviors—that is, those focused on restricting one’s own behavior (e.g., reducing consumption of red meat and alcohol)—especially when compared with more active prevention behaviors (selecting high-fiber foods). This is exactly what they found. Put another way, self-limiting behaviors are not typically associated with an aggressive battle setting, so the war metaphor portrays them as less appropriate for properly fighting the “war on cancer.”

Of course, in reality, these behaviors might be quite effective. Metaphor use may support understanding, but it does not guarantee accurate or healthy decisions (a point we return to in the next section).

Going further, our analysis suggests a more complex interaction between the metaphoric framing of an abstract disease and the metaphoric framing of a candidate treatment. To illustrate, imagine that Nader understands depression metaphorically as being *down* or *low*. He may infer that a medicine or therapeutic technique designed to treat depression needs to *lift him up* to be effective. Objectively speaking, depression is an abstract cognitive-affective condition, not a drop in vertical position, meaning that a treatment's associations with upward movement have no bearing on its efficacy. Still, it is possible that Nader thinks about depression by analogy to his experience of upward and downward movement and, as a result, he relies on that embodied knowledge to interpret a treatment's efficacy.

This phenomenon was demonstrated in a series of studies by Keefer, Landau, Sullivan, and Rothschild (2014). Participants who had been exposed a metaphoric framing subtly comparing depression to being *down* positively evaluated the effectiveness of a purportedly new antidepressant medication, named "Liftix," advertised as an "uplifting" treatment for people experiencing depression. Participants seem to have reasoned that the medication works if it "solves" the bodily problem of being down. In contrast, when depression was framed without a provided metaphor, participants were not especially attracted to Liftix. For them, the medicine's advertised *up*-ness was irrelevant.

In fact, participants actively denigrated a treatment if it was framed metaphorically as solving a *different* bodily problem than the one used to frame depression. When depression was framed as a problem of being *darkened*, participants rated Liftix as less effective than Effectrix, an equivalent medicine framed in non-metaphoric terms. If you're trapped in the dark, a drug that "lifts" your mood is not particularly effective.

Summing up our selective literature review, qualitative linguistic analyses show that bodily metaphors are commonly used in medical and lay discourse to communicate about a range of abstract health-relevant concepts. Converging experimental research provides stronger evidence that bodily metaphors operate at a conceptual level, shaping outcomes like health risk assessment, health behavior intentions, and treatment evaluations. Specifically, studies show that even incidental exposure to bodily metaphoric framings of health concepts leads people to make judgments and decisions about those health concepts that parallel their schematic knowledge of the bodily concepts to which they are compared. Even though those bodily concepts are unrelated and even irrelevant to the health concept at hand, they are nevertheless useful for gaining a confident grasp of the otherwise abstract, complex, and uncertain processes underlying health. This is one way in which health is embodied.

Practical Implications

Why does all this matter? Although health complications are partly determined by factors that are difficult for individuals to fully anticipate or control, ultimately their choices make a big difference. For example, it is estimated that 43% of all cancers could be prevented by changing high-risk behaviors and reducing exposure to environmental risk factors (Parkin, Boyd, & Walker, 2011). That is why health communicators have for many years publicized messages about health risks through various media outlets like public service announcements and consumer product labels. These messages are designed to motivate recipients to adopt and maintain lifestyle behaviors that reduce their risk of disease. Yet, despite the current health messages

in the media, people continue to engage in unhealthy practices, indicating that there is considerable potential to improve the power of these messages.

One way to accomplish this is to enhance these messages with bodily metaphors. The theory and research summarized above suggest that such metaphors are interpreted by observers not simply as colorful figures of speech; instead, they can systematically influence how people think about risk, treatment efficacy, and other operative constructs. Hence, developing communication strategies that guide the design of bodily metaphoric health messages can be a low-cost, theoretically grounded, and potentially powerful means of improving health message efficacy.

With this promise, however, come potential pitfalls. Metaphor usage is not guaranteed to help people *accurately* construe a health concept or motivate them along the path to lifestyle behavior change. That's why it is important to consider when bodily metaphors help and when they mislead or squelch motivation to make healthier choices.

When Bodily Metaphors Help

When a health risk seems abstract or removed from the practicalities of daily life, people may not feel particularly worried about the threat it poses, and hence they are not energized to change their current behavior. Metaphor can be used to compare that risk to a more concrete, easily visualized hazard, which might elicit the worry necessary to energize preventative behavior. This process was demonstrated in the aforementioned studies by Scherer et al. (2015): comparing the elusive and invisible flu virus to an aggressive army intent on attacking one's body raised observers' worry to energizing levels, prompting them to take action to get inoculated.

We can derive a more specific hypothesis from conceptual metaphor theory. If exposure to a metaphoric framing leads observers to recruit *their* knowledge of the concrete bodily experience to interpret the target health risk, then it should motivate action particularly among those individuals who, by disposition, are highly fearful of that bodily hazard. If, in contrast, observers are not especially disturbed by that hazard, then the metaphoric message will not elicit the worry necessary to motivate prevention. Landau, Arndt, and Cameron (2015) put this idea to the test. They built on Scherer et al.'s procedure but added an individual difference moderator: How much does the observer fear physical confrontations with aggressive others?

For individuals who strongly fear confrontations, exposure to metaphoric phrases and imagery comparing invisible ultraviolet radiation to an aggressive person pummeling one's skin increased worry about skin cancer risk and, furthermore, strengthened intentions to use sunscreen when going outside. But among participants who previously reported that they are not particularly disturbed by the idea of an aggressive person trying to hurt them, this metaphor had no effect on worry or motivation to apply sunscreen.

In addition to increasing energizing levels of worry, a metaphor can bolster so-called *response efficacy*. That is, it can bolster people's confidence that a recommended treatment behavior is effective at reducing their risk. Imagine that a doctor prescribes a new medication to help a patient fall asleep. If the patient lacks an intuitive understanding of how that medication works, he may not be sufficiently motivated to use it. A metaphor can compare the treatment's effect to a familiar aspect of the patient's ordinarily bodily experience. The sleep medication can be framed, for example, as "loosening the grip" of a nerve-wracked consciousness, thereby permitting sleep. The patient already has a familiar, confident sense of how manual gripping and "letting go" work, and a metaphor can transfer that schema to bolster confidence that a treatment works.

Similarly, metaphor has the potential to bolster *self-efficacy*, or confidence in one's ability to perform a behavior required to produce desired outcomes—in this case, reducing risk for health complications. Metaphors can reinforce feelings of self-efficacy by likening an abstract treatment or prevention behavior (e.g., “control your anxiety”) to something that is more concrete (e.g., “When that inner ball of anxiety starts shaking, turn your mind's gaze away to something pleasant”). More generally, metaphors help the person make meaningful sense of health-related concepts and situations that might otherwise seem overwhelming in their ambiguity and unpredictability (Teucher, 2003).

Additionally, because metaphors provide a stock of familiar images and schemas for how things work, they can facilitate communication between medical health professionals and the lay public (Krieger, Parrott, & Nussbaum, 2011; Penson et al. 2004; Reisfeld & Wilson, 2004). When treatment metaphors such as “fighting the war on cancer” are used in patient–practitioner communication, they can reduce perceptions of ambiguity and allow for increased understanding. This is important because an emerging and controversial topic is patient-based decision making, a practice that puts patients in the driver's seat and makes them more active in the decision making process (Mazur, 2015). However, endorsing this practice can be a double-edged sword. It can empower patients, but it can also expect them to make important decisions without sufficient background information. Without having an educated understanding of the choices available for treatment, these patients may feel confused. Metaphors can help to provide information about the disease and treatment options without subjecting patients to a long process of deliberation, questioning, and uncertainty.

Furthermore, although we saw in Hauser and Schwarz's (2015) work that understanding cancer as an enemy to be “fought” can reduce people's intentions to engage in self-limiting treatments (e.g., restricting one's diet), this was only true when the treatments were framed in exclusively non-metaphoric terms. “Restricting one's diet” in and of itself may seem like a rather ineffective weapon for “fighting the war on cancer,” but when told that this technique was *apt for fighting enemies*, participants viewed it as a useful and desirable strategy for treatment. This finding highlights the point that consistency between metaphorically framed health problems and their solutions not only increases motivation to seek treatment but also guides the individual to seek interventions that are easier to understand. By clearing the uncertainty that obscures our understanding of abstract health risks, metaphors can help us take responsibility and confidently address the issues that threaten our personal health and overall wellness.

When Bodily Metaphors Hurt

As alluded to earlier, a metaphor's utility for understanding is no guarantee of its accuracy, and in some cases the use of bodily metaphors can be misleading. By basing models of a health concept on concepts of a different kind, without due consideration of the health concept's unique properties, metaphors can perpetuate myths and negative stereotypes of individuals affected by disease.

Consider that, historically, the most pervasive metaphor for cancer is that of a military battle. Cancer discourse is replete with the vocabulary of war: *combatants*, *weapons*, *battles*, *soldiers*, *the wounded*, *warriors*, *survivors*, and *victories* (Mukherjee, 2011). However, a metaphor's mapping is partial and selective, meaning that it highlights certain aspects of the target concept (making them salient guides to thought and behavior) while at the same time playing down other aspects of the relevant ideas, pushing them to the periphery of consciousness.

Metaphors can mislead about the nature of a given health risk and a treatment's efficacy. For example, in a typical war, there is a single, clearly defined enemy. This may explain why oncologists in the 1960s narrowly tailored their efforts to find a "universal cure" for all forms of cancer. They imagined cancer to be *one* disease—a single, monolithic entity. The *war* metaphor may have underpinned the belief in the underlying singularity of cancer. Now, with scientific advancement, we are learning that cancer is a shape-shifting disease of immense diversity, and the same treatment cannot indiscriminately be applied to all forms.

The broader point is that studying how metaphors shape mental models of health concepts can not only improve health message efficacy but also shed light on unacknowledged sources of persistent misconceptions about health. The converse is that there may be a treatment or intervention that is, in actuality, quite effective, but if it is framed in a way that does not appeal to people's metaphorical conception of the health risk, it will not be endorsed.

Metaphors can foster misconceptions about not only a health risk or treatment but also those individuals who are affected by it. Sontag discusses this harmful consequence of metaphorical language, noticing the negative influences that cultural myths surrounding cancer had on her as a patient. She suggests that culture can potentially turn diseases into mythologies by perpetuating negative stereotypes and attributing stigma to both the disease and the afflicted individual. Sontag extends her critique of metaphors, purporting that AIDS has become the disease that is most demonized by society. Widely promulgated metaphors portray AIDS as pollution or decay, implying that afflicted individuals possess tainted moral values (reflecting the more general metaphor *immorality is dirt*; Lakoff & Johnson, 1980). Comparing this disease to a "plague" highlights not only the infectiousness of the physical virus itself but also the transmission of immorality. The diagnosed patient is perceived to be responsible for indulging in delinquency, and the illness is viewed as a punishment for social deviancy. This perception not only contributes to patients viewing themselves as socially devalued members of society but also may hinder them from seeking necessary treatment. Furthermore, it may incline public policy officials from supporting funding for research and interventions.

Additionally, metaphors can mislead about the individual's role in terms of having the agency or responsibility necessary for action and consequently may place the burden on the patient. Patients may begin to construct their own subjective experiences with the framework provided by the chosen metaphor, which in turn may promote feelings of shame, guilt, or hopelessness. Sontag suggests that as long as cancer is viewed as an evil enemy rather than a biological disease, victims of cancer will be demoralized upon learning that they have such a disease. Moreover, she asserts that the metaphor may actually distract from effectively treating the disease, leading to a greater number of fatalities by inducing paralyzing fear.

Although too much fear can be problematic, it is important to keep in mind that arousing a certain amount of fear can be beneficial for patients when making decisions about health behaviors. In particular, research on fear appeals in the health literature shows that inducing a moderate level of fear is the most effective way to motivate health behavior change. High levels of fear can backfire because people just shut down and repress awareness of the problem (Glanz, Rimer, & Lewis, 2002). Therefore, if metaphors transfer emotional worry from concrete hazards to evaluations of the target health risk, and that fear is very high, it may paralyze people from taking action. For example, envisioning how cancer cells "invade the body" and describing chemotherapy as "chemical warfare" can induce confusion and cause cancer patients to be overwhelmed by the threatening nature of the disease.

Similarly, doctors and health researchers can get caught in a web of beguiling metaphors that may lead to miscommunication. Aside from the public's response to metaphors, medical professionals and researchers may have their scholarly efforts guided by a superordinate

metaphor that is appealing for the sake of clear comprehension, but which leads them to overlook the unique properties of the target health concern and inject it with properties that it does not have. The implication is that interventions designed to reduce misconceptions and motivate behavior should pay particular attention to *which* metaphors are used in ordinary and medical discourse to frame health concepts.

Looking Ahead

The use of bodily states and physical experiences to inform health-related cognition and behavior is, most likely, not a single phenomenon produced by bodily metaphor or any other single mechanism. Instead, much like other complex social cognitive processes, it is likely to be produced by an underlying battery of interacting mechanisms. If this is true, the question then becomes, what are those mechanisms?

Besides bodily metaphor, candidate mechanisms are featured in influential views on embodied cognition (see relevant references at the outset of this entry). Consider Barsalou's (1999, 2008) perceptual symbols systems model. This model posits that concepts contain representations of bodily states (e.g., sensations) that customarily occur during interactions with relevant stimuli and contexts. To illustrate, people's *influenza* concept may contain, in addition to abstract knowledge of viruses and treatments, sensorimotor representations of bodily symptoms (e.g., fever) that routinely occur during experience with the flu. As a result, thinking about those concepts triggers the simulation, or neural reactivation, of associated bodily states, even when the individual is not currently interacting with the relevant stimuli (i.e., thinking about flu activates the bodily experience of nausea).²

The important point for now is that there is a large body of theory and research on embodied or grounded cognition that can be used to guide further work on embodied health. Developments in this area promise to shift us away from a narrow view of minds as disembodied information processors and reveal the vitally important roles that embodied experience plays in health-relevant cognition and lifestyle behaviors.

Acknowledgment

This material is based upon work supported by the National Science Foundation under Grant Number BCS-1222047.

Author Biographies

Ariel J. Mosley is currently pursuing a PhD in social psychology at the University of Kansas. She received her BA from California State University Sacramento in 2014. Her research focuses on processes of stereotyping, prejudice, and discrimination as they relate to stigma internalization and threats to existential motivations. Particularly, she is interested in issues of social power, group identity, and the psychological consequences of subtle forms of discrimination.

Trevor James Swanson is a PhD student of psychology at the University of Kansas and received his BS from Texas Christian University in 2014. His research is currently focused on understanding human motivation through the lens of existential psychology, with a particular

interest in how mortality concerns influence self-regulation. Additionally, he is interested in how different strategies of evaluative organization within the self-concept can serve as temporary adaptive solutions to stress and limited mental resources.

Mark J. Landau is an associate professor of psychology at the University of Kansas. He received his PhD from the University of Arizona in 2007. His research on metaphor's role in social thought and behavior is published in diverse academic journals and edited books. His research has been funded by the National Science Foundation and the National Cancer Institute.

Notes

- 1 Other common metaphors, less directly tied to bodily states, compared cessation to a job or the breakup of a dysfunctional friendship with tobacco. This variability illustrates that not all conceptual metaphors are embodied; rather, some capitalize on cultural schemas of social interactions.
- 2 Embodied simulation mechanism and metaphoric conceptualization are distinct mechanisms. Distinguishing them is not relevant for the purposes of this entry. For a detailed comparison (see Landau et al., 2014).

References

- Akers, L., Gordon, J. S., Reyna, S., & Severson, H. (2013). Metaphors of smokeless tobacco addiction and cessation. *Addiction Research and Theory*, 22(1), 49–56.
- Barsalou, L. W. (2008). Grounded cognition. *Annual Review of Psychology*, 59, 617–645.
- Blascovich, J., & Seery, M. D. (2007). Visceral and somatic indexes of social psychological constructs: History, principles, propositions, and case studies. In A. Kruglanski & E. Higgins (Eds.), *Social psychology: Handbook of basic principles* (2nd ed., pp. 39–66). New York, NY: Guilford Press.
- Glanz, K., Rimer, B. K., & Lewis, F. M. (2002). *Health behavior and health education: Theory, research and practice*. San Francisco, CA: Wiley.
- Hauser, D. J., & Schwarz, N. (2015). The war on prevention: Bellicose cancer metaphors hurt (some) prevention intentions. *Personality and Social Psychology Bulletin*, 41, 66–77.
- Keefer, L. A., Landau, M. J., Sullivan, D., & Rothschild, Z. K. (2014). Embodied metaphor and abstract problem solving: Testing a metaphoric fit hypothesis in the health domain. *Journal of Experimental Social Psychology*, 55, 12–20.
- Krieger, J. L., Parrott, R. L., & Nussbaum, J. F. (2011). Metaphor use and health literacy: A pilot study of strategies to explain randomization in cancer clinical trials. *Journal of Health Communication*, 16, 3–16.
- Lakoff, G., & Johnson, M. (1980). *Metaphors we live by*. Chicago, IL: University of Chicago Press.
- Landau, M. J., Arndt, J., & Cameron, L. (2015). *Effects of metaphoric cancer communications on risk perceptions and prevention intentions*. Manuscript in preparation. University of Kansas.
- Landau, M. J., Robinson, M. D., & Meier, B. P. (2014). *The power of metaphor: Examining its influence on social life*. Washington, DC: APA Press.
- Mazur, D. J. (2015). Social pressure on patient decision making through shifting mental models: Presenting evidence to patients. *Sociology Mind*, 5, 100–104.
- McMullen, L. M., & Conway, J. B. (2002). Conventional metaphors for depression. In S. Fussell (Ed.), *The verbal communication of emotions: Interdisciplinary perspectives* (pp. 167–181). Mahwah, NJ: Lawrence Erlbaum.
- Mukherjee, S. (2011). *The emperor of all maladies*. New York, NY: Scribner.
- Niedenthal, P. M., Barsalou, L. W., Winkielman, P., Krauth-Gruber, S., & Ric, F. (2005). Embodiment in attitudes, social perception, and emotion. *Personality and Social Psychology Review*, 9(3), 184–211.

- Parkin, D. M., Boyd, L., & Walker, L. C. (2011). The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010. *British Journal of Cancer*, 105, 77–81.
- Penson, R. T., Schapira, L., Daniels, K. J., Chabner, B. A., & Lynch, T. J. (2004). Cancer as metaphor. *The Oncologist*, 9(6), 708–716.
- Reisfeld, G. M., & Wilson, G. R. (2004). The use of metaphor in the discourse on cancer. *Journal of Clinical Oncology*, 22, 4024–4027.
- Scherer, A., Scherer, L. D., & Fagerlin, A. (2015). Getting ahead of illness: Using metaphors to influence medical decision making. *Medical Decision Making*, 35, 37–45.
- Sontag, S. (1978). *Illness as metaphor*. New York, NY: Farrar, Straus & Giroux.
- Teucher, U. (2003). The therapeutic psychopoetics of cancer metaphors: Challenges in interdisciplinarity. *History of Intellectual Culture*, 3, 1–15.

Suggested Reading

- Gibbs, R. W. (2006). *Embodiment and cognitive science*. New York, NY: Cambridge University Press.
- Landau, M. J., Robinson, M. D., & Meier, B. P. (2014). *The power of metaphor: Examining its influence on social life*. Washington, DC: APA Press.
- Sontag, S. (1978). *Illness as metaphor*. New York, NY: Farrar, Straus & Giroux.

Emotion Regulation

Emily W. Shih and Elizabeth L. Davis

Department of Psychology, University of California, Riverside, CA, USA

Emotions are adaptive because they provide information that usually carries consequences for people's health-related goals. For example, feeling a rush of fear in response to a rustling sound in the bushes signals the possibility of an adverse situation (e.g., a snake in the underbrush) and prompts a reaction (e.g., freeze, flee, fight) designed to enable survival. Our emotional experiences, whether generated by the small-scale hassles of daily life or true life-and-death situations, can impact our overall health and well-being by triggering a cascade of stress responses. These responses are extremely useful under conditions of objective threat (there really is a snake in the underbrush) but can be corrosive in situations in which the emotional response and resulting behaviors are poorly matched to the situation (a person feels extreme fear, but the rustling sound was caused by the wind; even worse, a person feels no fear, but the rustling sound was actually caused by a snake ready to strike). Thus, our ability to regulate our emotions once they are evoked—to downplay, calibrate, or otherwise modulate them—can weigh heavily in how we experience and respond to stress and, in turn, our susceptibility to poor physical health.

This entry is organized into two parts. The first describes and defines emotion regulation as a construct and highlights the known links between emotion regulation and physical health. Given the conceptual similarities between experiencing negative emotions and experiencing stress and distress, we discuss emotion regulation in relation to stress responding, focusing on why and how emotion regulation abilities are vital to promoting, sustaining, and enhancing our health. The second section moves beyond the direct association between emotion regulation and health, highlighting sources of the inherent variability in people's emotion regulation skills and how this maps on to susceptibility to illness and disease—we consider individual and developmental variability in skill and the importance of context, as moderators of the emotion regulation-health link that are ripe for future work to examine. Though research on these boundary conditions and constraints is growing, this is a relatively new area of focus, and we conclude with recommendations for health psychologists who wish to further understanding of emotion regulation's role in promoting or undermining people's health.

Emotion Regulation

The emotions we experience not only can influence our behavior in the short term but also can carry long-term consequences for how we understand and process our experiences, which in turn influences our future decisions. This process can apply not only to basic, daily life tasks like deciding which movie to see (avoiding one that looks likely to be scary, if the last scary movie you saw scared you so much, you jumped in your seat and spilled your drink all over your date) but also to life decisions that are much more consequential. For example, feeling upset about an awkward interaction with a new doctor can lead to negative emotions in the moment and, more damagingly, can preclude someone from seeking medical attention for later ailments. Poorly regulated emotion can interfere with successful health at the behavioral level, reflected in the decisions people make and the habits in which they engage, and at the physiological level, as reflected by dysfunction in stress response systems. We consider both behavioral and biological levels of analysis as crucially important to a complete understanding of emotion regulatory processes implicated in health.

Among the most robust findings in health psychology are those documenting the role that negative emotions and distress (or negative affect) play in the onset, maintenance, and exacerbation of health problems. Negative emotions appear to be associated with these health-relevant outcomes due to their potential to trigger a cascade of behavioral (e.g., self-medicating with substances) and physiological (e.g., allostatic load) responses that often result in enhanced susceptibility to certain illnesses. A classic example is work conducted by Cohen and colleagues (1995), in which researchers experimentally induced physical health reactions by systematically exposing participants to a respiratory virus. Measures collected during the trial included samples of nasal secretions for detection of infection, facial tissues for assessing mucus secretion weights, and daily self-reports of negative affect states and upper respiratory symptoms. All measures were also gathered before exposure to the virus to allow a baseline comparison. Most people got sick because of the virus exposure, but one of the most intriguing findings was that people ultimately developed a more severe form of the illness if they had reported more negative affect at the time of the exposure.

Despite the role that negative emotions may play in worsening experiences in some contexts, they can be extremely adaptive in others. For example, experiencing negative emotion signals a status change in a goal that a person holds, and detecting this change in one's goals prompts certain responses (behavioral, cognitive, physiological, motivational) in line with the specific emotion at hand. Consider a parent who reprimands a child for impulsively darting into a busy street. In the moment, the scolded child might burst into tears and feel that the parent's reaction is overly harsh, but to the extent that this reprimand slows or stops the child's future urges to run into traffic, it has ultimately served as an excellent deterrent (and health-promoting behavior).

Generally speaking, positive emotions are viewed as being protective—to one's well-being, mental health, or even physical health. People with more positive dispositions (e.g., those who experience positive affect more often or more intensely) tend to have stronger immune systems and cardiovascular function. Positive emotions are not always beneficial, however—context is crucial. Displays of positive emotion in certain (usually social) contexts can be highly inappropriate or offensive to others and thus confer substantial costs for the person who misjudged or miscalibrated the emotional response. For example, laughing uproariously during a somber funeral eulogy would probably introduce considerable tension to multiple relationships, costing the person social support resources and potentially introducing negative emotional states.

Thus, both positive and negative emotions may be adaptive or maladaptive, depending on the particular context. Context is critical for identifying, expressing, and regulating one's emotions in ways that promote adaptive functioning (social, behavioral, physiological). Emotions are informative (about the status of our goals) or protective (as in the case of a positive emotional disposition), but they can also be detrimental if they are miscalibrated for the context—of the wrong form, at the wrong time, or with the wrong intensity. In these cases, emotions must be regulated to promote adaptive or successful functioning. How people go about doing this, however, carries a separate set of consequences for functioning.

Broadly, emotion regulation encompasses any process that significantly influences the quality, intensity, timing, or dynamic features of an emotional experience (or response). According to this definition, emotion regulation refers to a wide range of biological, social, and behavioral regulatory processes (Gross, 2001). The way people regulate emotions depends on many factors, including the emotion at hand, the individual's point in the lifespan (e.g., infancy versus childhood versus adulthood), and the sum of one's experiences with emotion and regulation up to that point (e.g., experience or input from the environment). Though emotion regulation is often conceptualized as a means of "managing" one's emotions (i.e., the regulation *of* emotion), it can alternatively be viewed as the process by which one's emotions regulate behavior (i.e., regulation *by* emotion). Regardless of which view one takes, the relation between emotion and regulation is bidirectional, dynamic, and transactional over time—though emotion processes are often studied experimentally by inviting participants to the lab and evoking a target emotional state, in reality people rarely find themselves feeling "neutral" in their daily lives. People may feel mildly pleased about some good news they received this morning, or mildly anxious about an impending deadline, but regardless of whether emotions are intense enough to require active attempts at regulation, people are experiencing and modifying their feelings all the time. Often, emotion-related behavior (and motivation for regulating certain affective states) is driven by display rules, or sociocultural norms and standards for behavior. These norms vary across cultural groups and provide prescriptions for which emotional responses are appropriate in which contexts. Unsurprisingly, these standards for emotion expression have clear consequences for the way people regulate their emotions in the service of these superordinate sociocultural goals.

From this view, the incredible complexity of emotion regulation processes becomes clearer—for any individual person, effective regulatory behavior will depend on a host of factors, be constrained by multiple aspects of the context, and vary within person over developmental or historical time. Thus, no one way of regulating emotion is likely to be effective across all of these gradients of variability, so it is important for people to cultivate regulatory flexibility—in other words, to engage in regulatory processes that are readily adaptive, situationally responsive, and socioculturally specific. Effective emotion regulation predicts all manner of adaptive outcomes, including better social strategies when engaging with peers (e.g., Lopes, Salovey, Côté, Beers, & Petty, 2005) and greater success in cognitive delay tasks (e.g., Carlson & Wang, 2007), indicating the importance of considering emotion regulation processes for general well-being and healthy functioning.

Emotion Regulation Predicts Health and Well-Being

Emotions and the ability to effectively regulate one's emotions (e.g., change or modify emotional experience as desired or required) have been linked to physical health. For example, the chronic tendency to experience a particular emotional state (e.g., negative affectivity) predicts

illness symptoms (Watson, 2000). Thus, mood states that are longer lasting have clear implications for people's health. One explanation for the link between negative emotions and poor health outcomes centers on stress regulation as a mechanism by which unchecked (unwanted) negative emotions become overwhelming. Stress can be thought of as a process (e.g., noticing that you have been progressively feeling more "stressed" before a big exam), an outcome (e.g., a measurable biomarker that changes under conditions of challenge), or an experienced event (e.g., stressors, life events, or daily hassles). Here we consider stress as the process of reacting to input from the environment, in light of how this process physically taxes the body. Thus, stress typically arises when individuals perceive that they cannot effectively cope with the demands being made on them, or with threats to their well-being.

Stress reactivity can be defined as biological reactivity to environmental stressors, and individual differences in this response are thought to partially account for the broad between-person variability in susceptibility to stress-related illnesses. Generally, exaggerated stress reactivity is viewed as maladaptive and potentially toxic to one's overall well-being, placing individuals at heightened risk for mental and physical health problems. A common approach to describing the damaging effects of accumulating stress is to use an allostatic load framework, an approach that quantifies the "wear and tear" on the physiological systems of the body that are responsible for maintaining homeostatic function as well as supporting stress responses under conditions of challenge. Generally, the activation of the stress response system (the sympathetic branch of the peripheral nervous system, the system responsible for "fight-or-flight" responses) and return to pre-challenge levels and homeostasis after a challenge has ended or been handled (the parasympathetic branch of the peripheral nervous system, the "rest and digest" calming response to threat) is an extremely adaptive dynamic response. Under certain conditions like chronic stress or repeated exposure to intense stress, however, the system wears down. Prolonged or repeated activation of these allostatic systems results in the breakdown of their optimal functioning, providing evidence of allostatic load on the system, which brings a host of physiological and health-related problems.

Stress arises and persists under conditions of challenge and influences the physical functioning of the body. The more intense, chronic, or prolonged the experience of stress, the more consequences for the stress response systems and the greater likelihood of experiencing physical health decrements. Stress has been linked to all leading physical causes of death, including heart disease, cancer, and stroke (Cohen, Janicki-Deverts, & Miller, 2007). It is also commonly associated with development of major mental health issues such as depression, post-traumatic stress disorder, and pathologic aging (Marin et al., 2011). By logical extension, one can think of negative emotions as a potential source of intense, chronic, or prolonged distress that may ultimately have similar effects on physical health. Coupled with ineffective emotion regulation, distressing negative emotions can be especially damaging.

Many studies addressing the relation between emotion regulation and physical health have focused on how emotion regulation affects immune system functioning. People who regain or maintain positive emotional states through means of laughter or coping with humor when faced with stressful life experiences can show improvements in immune functioning (e.g., Tugade, Fredrickson, & Feldman Barrett, 2004). Reported positive and negative emotions have been linked with the release of secretory immunoglobulin A (S-IgA). S-IgA is considered the antibody first on defense against the common cold. Some research suggests that positive moods appear to enhance immune system responding, but this effect is compromised by negative moods. Emotion regulation, to the extent that it decreases negative emotion or enhances positive emotion, can thus arguably influence S-IgA levels.

Ineffective emotion regulation can also influence long-term health by increasing the risks of mental health problems like depression and anxiety. Dysregulated negative emotions like anxiety, fear, and sadness are implicated in many clinical symptoms and predict later disorders. For example, O'Leary (1990) found that when grief and sadness were not effectively regulated, these affective states often forecasted severe unipolar depression, which can result in immunosuppression. For example, Pisani et al. (2013) surveyed approximately 8,000 students about aspects of their lives including maladaptive emotion-related functioning: suicide attempts, difficulties in emotion regulation, lack of emotional clarity, limited access to emotion regulation strategies, and depressive symptoms. Difficulty regulating one's emotions (lack of clarity about feelings and limited access to useful strategies) related to increased depressive symptoms and likelihood of suicide attempts. Even after controlling for the presence of depressive symptoms, students who had difficulty with regulating their emotions showed elevated likelihood of a suicide attempt. Thus, ineffective emotion regulation is often implicated in affective psychopathologies like depression and anxiety disorders and can also affect this component of healthy functioning.

Additionally, stress, distress, and dysregulated emotion can interfere with behaviors that would enable someone to ameliorate physical health concerns. Abele and Hermer (1993) demonstrated that emotional states influenced people's beliefs about both their desire and their ability to engage in health-promoting behaviors. Individuals who were happier perceived themselves as better able to carry out health-promoting behaviors and had greater confidence that these behaviors would effectively treat their illness symptoms. On the other hand, individuals who were sad had less self-efficacy in relieving their illnesses, believing that they were limited in their ability to make themselves feel better, which interfered with subsequent seeking of or adherence to treatment.

Stress can also increase the likelihood of engaging in maladaptive behaviors (e.g., abusing substances, remaining in unhealthy relationships), which can have negative effects on physical health. These behaviors may represent maladaptive ways of regulating emotion. For example, considerable evidence suggests that people use and abuse alcohol as a result of their expectations of how it will influence their emotional states, as people tend to overdrink in attempts to regulate their negative feelings or to increase and heighten positive feelings (e.g., Sher & Grekin, 2007). Additionally, negative emotional experiences are an important antecedent to tobacco use (e.g., Shiffman & Waters, 2004). Smoking rates are considerably higher among individuals who have been diagnosed as clinically depressed, and people report that the desire to change how they feel is a primary reason for smoking.

Other behavioral evidence suggests that the consistent use of certain emotion regulation and coping strategies is also associated with health outcomes, even when introduced as part of an experimental intervention. Emotional disclosure is a form of emotion regulation, as it allows someone to revisit an upsetting experience as a way of processing the event or to enable the person to seek social support from others. Pennebaker (1997) found that even the simple act of disclosing emotional experiences in writing improves individuals' subsequent physical health. Students assigned to write about a traumatic emotional experience made fewer health center visits compared with students assigned to write about a neutral or mundane topic. Enhanced immunological functioning has also been observed as a result of emotional disclosure, in conjunction with decreases in self-reported physical symptoms and depression. In related work, Emmons and McCullough (2003) assigned participants to one of three groups: (a) a "count your blessings" group, (b) a "list your daily hassles" group, and (c) a control group. Individuals who counted their blessings weekly for ten weeks reported the best subjective health outcomes

(e.g., fewer physical complaints, increased exercise and sleep, better sleep quality), supporting the idea that cultivating better regulatory abilities can promote health.

Individual and Developmental Variability in Emotion Regulation Skill

The benefits of effective stress reduction and emotion regulation for adaptive functioning are hard to overstate. Stress reduction and emotion regulation skill leads to enhanced concentration, improved communication and relationships, more stable moods, and ultimately better physical health. Of course, there is considerable variation in how people regulate their emotions effectively and what constitutes “effective” regulation will necessarily change with time and across contexts. Here, we consider some of the external constraints and individual differences in effective emotion regulation that represent promising directions for future research into the effects of emotion regulation on physical health and functioning as the field moves toward identifying more precise targets for intervention. Specifically, we consider moderators of the relationship between emotion regulation and health. Clarifying the direct links between emotion dysregulation, distress, and health outcomes is an important first step, but we encourage scientists in this area to begin to examine the putative moderators of the associations (e.g., for which individuals, under which circumstances, are a particular way of regulating emotion “effective”?) to advance knowledge in the field.

People vary in what they can do to regulate negative emotions and stress. Adults in particular have a diverse set of options from which to choose when regulating negative emotions and dealing with stress. Two strategies that have been shown to be effective across a variety of contexts are reappraisal and problem solving. Gross (1998) first highlighted the efficacy of reappraisal by showing that it results in positive emotional and physical responses to emotion-eliciting stimuli. Both reappraisal and problem solving are a common component of cognitive behavioral therapies for psychopathological disorders (e.g., Stewart & Chambless, 2009). In contrast, expressive suppression and emotional avoidance have been shown to be relatively maladaptive responses to stressors and are associated with risk for psychopathological disorders, including anxiety (e.g., Campbell-Sills, Barlow, Brown, & Hofmann, 2006).

In daily life, people encounter multiple opportunities to engage in context-appropriate regulation. For example, it is often necessary to actively regulate emotions in professional or social settings because the workplace requires sustained attention, and successful social interactions are governed by display rules. Work or school contexts may be sources of different amounts and types of stress at different developmental stages, and effective implementation of specific strategies will thus vary according to contextual demands. There has been growing interest in the importance of flexibility in implementing strategies to match contextual demands because some strategies have been shown to be more costly under certain circumstances. For example, in social contexts, suppressing the expression of one’s true feelings can interfere with establishing rapport or closeness to the extent that the suppressor appears disengaged and avoidant (rather than benignly neutral, perhaps, Butler et al., 2003, but see Butler, Lee, & Gross, 2007, for evidence that this negative effect of emotion suppression is qualified by culture). In contrast, expressive flexibility, which is the ability to flexibly enhance or suppress emotional expression across different contexts, protects against detrimental effects of cumulative life stress (Westphal, Seivert, & Bonanno, 2010). Considering the constraints that context can impose on individuals’ responses to stress and regulation of negative emotions represents an important direction for future work.

Differences in the effects of specific emotion regulation strategies are also important to acknowledge because an understanding of individual differences in emotion regulation abilities involves examination of the emotion regulation strategies that people acquire and use in their own lives. The way people choose to regulate their emotions and deal with stress and negative events is variable, and what may be successful for one individual may not be for another person, or for the same person in a different context. Stress levels, how people react to stress, and the effects of stress on well-being all represent sources of heterogeneity that must be unpacked to understand the effectiveness of different emotion regulation strategies. Identifying differences in the process of acquiring particularly adaptive and functional emotion regulation strategies across the lifespan can help us isolate the developmental origins of dysregulated behaviors that could put individuals at risk for illnesses and negative health-related outcomes. Such an approach would be useful for informing future intervention work by identifying children at increased risk for later physical health problems and allowing intervention efforts to target improvement of emotion and stress regulation.

Intrinsic and extrinsic factors contribute to individual differences in emotion regulation skill, and many of these originate very early in life. Intrinsic differences in biological stress response systems and temperament (biologically based individual differences in emotional reactivity and self-regulatory ability) can put children on a particular developmental trajectory toward (or away from) emotional, mental, and physical health. These intrinsic factors emerge early in infancy and are influenced by the infant's reactivity to environmental stimulation, as well as ability to self-soothe and regulate distress. These early temperamental factors influence later development of more explicit attentional and inhibitory skills that may be applied to regulating reactions. Of course, the development of emotion regulation skill and flexible strategy use in early life is shaped by dynamic transactions between the child and environment, including other people. Children's families, neighborhoods, schools, peers, and culture all play a role in emotional development, as do aspects of the individual child (e.g., in eliciting and seeking out certain kinds of input from the social world). Most research to date has focused primarily on caregiver and parental influences. For instance, a child's ability to effectively manage negative emotions or distress hinges on both the child's capacity for self-soothing and the sensitivity and responsiveness of a caregiver to meeting the emotional needs of the child. The developmental transactions and individual factors that contribute to the attainment of competent emotion regulation abilities will fluctuate as a function of the child's capabilities and resources during periods of skill acquisition. The inherent variation in these transactions thus contributes to individual differences in emotion regulation in childhood, adolescence, and into adulthood, with implications for physical health throughout development.

Conclusion

Emotions have enormous influence over a wide range of health outcomes. Effectively managing negative emotions in a way that mitigates their negative effects is vital for adaptive functioning. Effective emotion regulation abilities can play a crucial role in promoting improved physical health outcomes (e.g., stronger immune systems) and guarding against health-damaging behaviors (e.g., alcohol abuse, smoking). Thus, it is important for people to develop competent emotion regulation abilities to promote healthy outcomes. It is not possible to narrow the cause of competent emotion regulation abilities to a single determinant source. Instead, we can attempt to understand which emotion regulation strategies work for which

people in which contexts to begin to more fully understand the links between competent emotion regulation abilities and people's overall health and well-being.

Author Biographies

Emily W. Shih is a graduate student in the Developmental Psychology Program at the University of California, Riverside. Her research interests are centered around examining the behavioral and physiological effects of parent–child interactions on children's socioemotional development. Presently, her research examines how parents' emotion regulation abilities and socialization of emotion regulation strategies map onto children's growing abilities to regulate their own emotions, their psychopathology, and their physiological flexibility.

Elizabeth L. Davis is a developmental psychologist and an assistant professor in the Department of Psychology at the University of California, Riverside. Her research uses a biopsychosocial framework to clarify emotion processes that predict adaptive (e.g., academic success, social competence) and maladaptive (e.g., psychopathology, physical health problems) outcomes.

References

- Abele, A., & Hermer, P. (1993). Mood influences on health-related judgments: Appraisal of own health versus appraisal of unhealthy behaviours. *European Journal of Social Psychology*, 23, 613–625. doi:10.1002/ejsp.2420230606
- Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion*, 3, 48–67.
- Butler, E. A., Lee, T. L., & Gross, J. J. (2007). Emotion regulation and culture: Are the social consequences of emotion suppression culture-specific? *Emotion*, 7, 30–48. doi:10.1037/1528-3542.7.1.30
- Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Effects of suppression and acceptance on emotional responses of individuals with anxiety and mood disorders. *Behaviour Research and Therapy*, 44, 1251–1263. doi:10.1016/j.brat.2005.10.001
- Carlson, S. M., & Wang, T. S. (2007). Inhibitory control and emotion regulation in preschool children. *Cognitive Development*, 22, 489–510. doi:10.1016/j.cogdev.2007.08.002
- Cohen, S., Doyle, W. J., Skoner, D. P., Fireman, P., Gwaltney, J. M., Jr., & Newsom, J. T. (1995). State and trait negative affect as predictors of objective and subjective symptoms of respiratory viral infections. *Journal of Personality and Social Psychology*, 68, 159–169. doi:10.1037/0022-3514.68.1.159
- Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *Journal of the American Medical Academy*, 298, 1685–1687. doi:10.1001/jama.298.14.1685
- Emmons, R. A., & McCullough, M. E. (2003). Counting blessings versus burdens: An experimental investigation of gratitude and subjective well-being in daily life. *Journal of Personality and Social Psychology*, 84, 377–389. doi:10.1037/0022-3514.84.2.377
- Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74, 224–237. doi:10.1037/0022-3514.74.1.224
- Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*, 10, 214–219. doi:10.1111/1467-8721.00152
- Lopes, P. N., Salovey, P., Côté, S., Beers, M., & Petty, R. E. (2005). Emotion regulation abilities and the quality of social interaction. *Emotion*, 5, 113. doi:10.1037/1528-3542.5.1.113
- Marin, M. F., Lord, C., Andrews, J., Juster, R. P., Sindi, S., Arseneault-Lapierre, G., ... Lupien, S. J. (2011). Chronic stress, cognitive functioning and mental health. *Neurobiology of Learning and Memory*, 96, 583–595. doi:10.1016/j.nlm.2011.02.016

- O'Leary, A. (1990). Stress, emotion, and human immune function. *Psychological Bulletin*, 108(3), 363–382.
- Pennebaker, J. W. (1997). Writing about emotional experiences as a therapeutic process. *Psychological Science*, 8, 162–166. doi:10.1111/j.1467-9280.1997.tb00403.x
- Pisani, A. R., Wyman, P. A., Petrova, M., Schmeelk-Cone, K., Goldston, D. B., Xia, Y., & Gould, M. S. (2013). Emotion regulation difficulties, youth-adult relationships, and suicide attempts among high school students in underserved communities. *Journal of Youth and Adolescence*, 42, 807–820. doi:10.1007/s10964-012-9884-2
- Sher, K. J., & Grekin, E. R. (2007). Alcohol and affect regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 560–580). New York, NY: Guilford Press.
- Shiffman, S., & Waters, A. J. (2004). Negative affect and smoking lapses: A prospective analysis. *Journal of Consulting and Clinical Psychology*, 72, 192. doi:10.1037/0022-006X.72.2.192
- Stewart, R. E., & Chambless, D. L. (2009). Cognitive-behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *Journal of Consulting and Clinical Psychology*, 77, 595–606. doi:10.1037/a0016032
- Tugade, M. M., Fredrickson, B. L., & Feldman Barrett, L. (2004). Psychological resilience and positive emotional granularity: Examining the benefits of positive emotions on coping and health. *Journal of Personality*, 72, 1161–1190. doi:10.1111/j.1467-6494.2004.00294.x
- Watson, D. (2000). *Mood and temperament*. New York, NY: Guilford Press.
- Westphal, M., Seivert, N. H., & Bonanno, G. A. (2010). Expressive flexibility. *Emotion*, 10, 92–100. doi:10.1037/a0018420

Suggested Reading

- Aldao, A. (2013). The future of emotion regulation research: Capturing context. *Perspectives on Psychological Science*, 8, 155–172. doi:10.1177/1745691612459518
- Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, 75, 317–333. doi:10.1111/j.1467-8624.2004.00673.x
- Gross, J. J. (2013). *Handbook of emotion regulation* (2nd ed.). New York, NY: Guilford Press.
- John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of Personality*, 72, 1301–1334. doi:10.1111/j.1467-6494.2004.00298.x
- Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion*, 23, 4–41. doi:10.1080/02699930802619031

Everyday Coping Behaviors

Megan L. Robbins, Robert C. Wright,
and Alexander Karan

Department of Psychology, University of California, Riverside, CA, USA

Coping takes place over the course of daily life, yet measures of coping have not always reflected this context. This entry discusses how methodology has shaped what we know about coping and what we have learned so far about everyday coping behavior, beyond coping styles and strategies.

Traditional Conceptualization and Measurement of Coping

Initially, coping research focused on strategies and dispositions that influence the impact of a negative event on well-being. This research yielded important insights about different types of coping strategies (e.g., active versus passive coping, emotion versus problem-focused coping) and when these types of strategies are most adaptive. For example, active coping—taking direct, intentional action to alleviate a stressor or its impact (Carver, Scheier, & Weintraub, 1989)—is generally an adaptive strategy when faced with a problem that is possible to remedy or reappraise. In the face of a stressor that is uncontrollable and not persistent (e.g., a temporary positive result for a diagnostic test), disengagement from the situation can be adaptive immediately following the event (Taylor & Stanton, 2007).

These distinctions made great strides toward understanding adaptive and maladaptive ways of handling major stressful experiences, but researchers noticed that an important piece was missing from coping research: the role of everyday behavior and experiences. For example, we know that active coping is often a helpful strategy, yet we do not know the specific daily behaviors enacted during the active coping process. Past research has also yielded limited knowledge about the changes active coping produces in everyday life. Further, questions remain about the role of active coping in the face of minor stressors throughout the day. Researchers noticed that these limitations were largely due to the methods used to assess coping. Exclusive use of retrospective self-reports unduly restricts what we can learn about coping because they are

limited to what people can remember—often what was most salient or recent—and exclude less memorable behavior and contextual information (Stone & Neale, 1984). Further, general endorsement of a particular item on a coping questionnaire is more appropriate for assessing coping styles as an individual difference, but not in-the-moment coping behavior (Stone et al., 1998; Stone, Greenberg, Kennedy-Moore, & Newman, 1991). Empirical evidence for this notion came 20 years later, when one study found that “concordance between global and daily reports was weak” (Todd, Tennen, Carney, Armeli, & Affleck, 2004, p. 310). Thus, coping researchers moved away from solely using global coping reports and began to use methods that get closer to behaviors linked to coping with daily diaries and ecological momentary assessments (EMA).

Daily Diaries and Ecological Momentary Assessment of Coping

Daily diary methods are daily reports, typically completed at the end of each day, in which participants can record their use of coping-relevant constructs such as coping strategies, mood, and support received. Using daily diaries, coping research gained insight into the role that minor events play in the coping process, as well as how daily fluctuations in coping, mood, and other behaviors influence coping outcomes such as psychological adjustment. Early diary research sought to understand the role of everyday stressors (e.g., arguments, deadlines, minor illnesses) rather than solely major events (e.g., divorce, loss of a job, death of a loved one). For example, one study used daily diaries to assess everyday stressors and their impact on mental health (Bolger, DeLongis, Kessler, & Schilling, 1989). This study focused on married couples’ daily stressors and mood over 6 weeks. They found that mood was better explained by day-to-day stress than researchers previously thought and that interpersonal conflicts best predicted day-to-day mood compared with other daily events. Daily diaries, then, provided a more accurate picture of what accounts for everyday mood than global reports alone could provide.

One of the biggest benefits derived from daily diary methodologies within coping research was the ability to conduct within-person investigations throughout a variety of stressful experiences. A study by Bolger et al. (1989) found that interpersonal conflicts explained 20% of the within-person variance in mood when facing everyday stressors. Other diary studies have also revealed the associations between use of specific coping strategies and well-being. One study revealed that using catharsis—emotion expression to reduce negative feelings about the stressor—and social support seeking were related to increased negative affect among a sample of men coping with daily stressors (Stone, Kennedy-Moore, & Neale, 1995).

Daily measurement of coping also facilitates more in-depth examination of individual differences in between-person studies. For example, rheumatoid arthritis patients completed daily diaries about their pain, coping, and mood for 75 consecutive days (Affleck, Urrows, Tennen, & Higgins, 1992). This study revealed vast differences in coping strategies used by the participants beyond what past retrospective accounts had depicted. Specifically, some people went weeks without using any coping strategies for their pain, while others used multiple strategies each day. Further, the study found that some participants reported using only one type of coping strategy throughout the study, whereas others reported a vast array of strategies. These previously unidentified individual differences in coping styles were discovered due to the unique methodological advantages of daily diaries.

EMA further improved our knowledge of daily events and coping by extending to the momentary level with multiple daily assessments. Multiple studies have found that such momentary reporting significantly increased the precision of measurement of daily coping by

relying even less on memory than diary methods. Notably, one study investigated the difference between coping reported immediately and two days after a stressful event by using an event-contingent method, which instructs participants to complete reports each time a stressful event (or other event of interest) occurs (Stone et al., 1998). The researchers found only 30% agreement between the momentary report and the retrospective reports of coping, even though the retrospection extended back only 2 days. This study was the first to show the advantage of EMA to study coping over even very brief periods of retrospection.

EMA has been useful in uncovering in-the-moment coping strategies that are helpful for specific stressors, like asthma symptoms. In one study, participants with asthma reported coping style, symptoms, and peak expiratory flow rate multiple times a day using a signal-contingent EMA, in which participants respond immediately after being prompted. Researchers found that use of avoidant coping strategies was associated with worse-than-normal asthma-related symptoms (Nazarian, Smyth, & Sliwinski, 2006). Thus, the introduction of EMA to coping research further improved the fidelity of self-reports of coping.

Though daily diaries and EMA vastly improved upon retrospective measures of coping, researchers have noted that they only provide information about coping that participants can readily report (Todd et al., 2004). The participant's perspective on coping is undoubtedly important, but no single measure can provide every piece of relevant information about coping in everyday life. Even as early as 1984, Stone and Neale noted the limitation of their daily self-report measure of coping, stating that they "do not claim to have developed a fully valid measure of coping; the potential weakness of the measure—that it relies on self-report—is all too obvious" (p. 905). Like retrospective reports, EMA is still subject to some of the same biases (e.g., social desirability) and limited to what people notice about their coping. Though it seemed obvious to coping researchers in 1984, the field did not begin to shift beyond self-reports until much later, when technology made it more feasible to compare self-reports to behavioral measures.

Naturalistic Observation of Coping

By sampling participants' social environments, researchers gain access to coping-relevant behaviors that go unnoticed by participants. For example, two studies employing the electronically activated recorder (EAR) (Mehl, Robbins, & Deters, 2012), a naturalistic observation method that consists of participants wearing a device that records ambient sound, have revealed links between expressive behavior and coping outcomes. First, a small sample of women with rheumatoid arthritis wore the EAR over 2 weekends, revealing preliminary evidence that sighing is more an indicator of depression than pain (Robbins, Mehl, Holleran, & Kasle, 2011). Another study of women with either rheumatoid arthritis or breast cancer uncovered the potential interpersonal cost of swearing in the coping context. Analyses revealed evidence that swearing in the presence of others may repel support networks at the expense of psychological adjustment (Robbins, Focella, et al., 2011). These findings shed light on the importance of mundane behaviors enacted in everyday coping, beyond reported strategies.

Naturalistic observation also remedies the problem with exclusive reliance on participants' reports of their social environments (e.g., how often specific topics arise in conversation). Questionnaire methods inherently assume what is most impactful in the coping process because they limit responses to the topics researchers have deemed most important (Hagedoorn, Sanderman, Bolks, Tuinstra, & Coyne, 2008). It is possible that over the years, coping researchers have developed questionnaire measures that capture the most important facets of

coping; however, this claim has yet to be empirically tested using other types of data collection for comparison. Naturalistic observation can complement self-reports of coping by providing an unfiltered account of everyday coping behaviors.

In one study, couples coping with breast cancer wore the EAR for 1 weekend and completed health and adjustment measures while the patient was undergoing active treatment. This approach provided unique insight into couples' daily interactions while enduring a highly sensitive and stressful experience. Interestingly, this study revealed that on average, cancer came up in less than 5% of couples' conversations (Robbins, López, Weihs, & Mehl, 2014). This discovery contributes a fundamental, if simple insight that has been missing throughout decades of cancer coping research. Past studies of the interactions of couples coping with cancer have focused almost exclusively on cancer-related conversations. However, consistent with relational regulation theory (Lakey & Orehek, 2011), which underscores the importance of ordinary social interactions, these findings highlight that an exclusive focus on cancer conversations overlooks 95% of couples' interactions. A focus on mundane, daily interactions may help resolve a long-standing conundrum in the cancer coping literature: studies consistently find that the quality of social relationships is greatly important for adjustment, but social support interventions based on this knowledge are relatively ineffective (Coyne, Stefanek, & Palmer, 2007). It is possible that a focus on mundane topics and behaviors not historically deemed important to coping research would improve the efficacy of coping interventions—perhaps by uncovering coping strategies that people can more readily incorporate into their daily lives.

Examining the coping-relevant facets of mundane rather than problem-focused conversations is thus a critical next step in the study of everyday coping. So far, one study has revealed that word use within non-cancer conversations is linked to dyadic adjustment, a multifaceted measure of marital quality, among couples coping with breast cancer (Karan, Wright, & Robbins, *in press*). Specifically, patients' and spouses' positive and negative emotion words were indicative of their partners' better and worse dyadic adjustment, respectively. Further, conversational focus on the spouse, as indexed by patients' use of "you" and spouses' use of "I," was linked to better adjustment in both partners. This finding emphasizes the importance of balance in the marital relationship—paying attention to the spouse in everyday conversation—while facing a major stressor like breast cancer. Such findings would remain concealed without the use of naturalistic observation methods like the EAR.

Summary

The study of everyday coping is still in its infancy, and more questions than answers remain about how facets of daily life contribute to coping. So far, we have learned that daily stressors greatly influence mood, people differ in their utilization of coping strategies, and expressive behaviors untapped by self-report methods may be important to the coping process. Developments in coping methodology have added more complete representations of coping in everyday life to what we already knew about coping styles and general coping tendencies.

Each research method has contributed a unique piece of information about how people cope in everyday life. Retrospective reports inform us about general coping styles and perception of how people feel they cope, whereas daily and momentary reports contribute the experience and perception of coping as it unfolds in daily life. Naturalistic observation fills in the coping-relevant behaviors and aspects of social environments that are difficult or impossible to report. Methodological plurality in coping research has led to greater understanding of how people respond to both major and minor stressors in everyday life.

Future Directions

Theory Building

Many questions remain, including how knowledge about everyday coping will be incorporated into existing overarching coping theories, as well as contribute to new ones. There are almost no unifying theories on everyday coping to date. One notable exception is relational regulation theory, which posits that most of the variance in social support is accounted for by mundane social interactions (Lakey & Orehek, 2011). More theories like this one, based on the existing body of research on everyday coping, are needed.

Multiple Perspectives on Coping

Greater use of EMA, naturalistic observation, and other methods in coping research is warranted, chiefly for adding diversified perspectives to gain a more comprehensive understanding of everyday coping. In addition, ambulatory physiological measures could facilitate knowledge of the pathways between everyday coping behavior and health. Passive telemetrics—automated sensors that participants can wear—give insight into fluctuations in emotion throughout the course of a day. While most passive telemetrics have not yet been used to study coping, it is now possible to begin incorporating them into studies to automate assessment in daily life. One cellphone app, the frequent electrodermal activity and event logging (FEEL) system, labels social interactions such as phone calls or emails as low, medium, or high stress using skin sensors and information already available from the phone (Ayzenberg & Picard, 2014). This work has aided in the recall of stressful events and can be utilized in combination with EMA and naturalistic observation to fine-tune advice on the types of support that are beneficial at various points throughout the day. For example, a study could passively sense stress levels, observe supportive behavior, and collect momentary reports of satisfaction with support. Multimethod approaches that harness technological advances hold promise for contributing to a more thorough understanding of coping.

Coping Behaviors

In addition to unpacking specific daily coping processes, new methodologies can also aid in the consideration of broader issues. For example, the field has not yet agreed upon which everyday behaviors should be considered “coping.” Researchers debate whether the definition of coping should be limited to what people do to effortfully alleviate the impact of a stressor, or whether the definition should include more automatized, habitual behaviors (e.g., Coyne & Gottlieb, 1996). Mapping observations of everyday behaviors onto reported coping strategies can identify behavioral manifestations of well-established coping constructs and thus provide a clearer sense of what people view as “coping” in their own lives. As technology advances methodological opportunities, research on coping can increasingly include facets of everyday life that contribute to a more complete picture of how people cope as they go about their days.

Author Biographies

Megan L. Robbins has been an assistant professor in the Department of Psychology at the University of California, Riverside, since 2013. She received her doctorate in psychology from the University of Arizona and her bachelor’s degree from the University of Texas in Austin.

Her research focuses on understanding how people's daily social interactions are related to health and well-being.

Robert C. Wright is a graduate student in the Department of Psychology at the University of California, Riverside. He earned his master's degree in sport psychology from San Jose State University and his bachelor's degree in psychology from California State University, Sacramento. His research focuses on the daily coping of patients living with chronic illness.

Alexander Karan is a graduate student in the Department of Psychology at the University of California, Riverside. He received his master's degree in psychology at the University of California, Riverside, and his bachelor's degree in psychology from the University of California, Los Angeles. His research interests include close relationships, emotion, stress, and coping.

References

- Affleck, G., Urrows, S., Tennen, H., & Higgins, P. (1992). Daily coping with pain from rheumatoid arthritis: Patterns and correlates. *Pain, 51*, 221–229. doi:10.1016/0304-3959(92)90263-B
- Ayzenberg, Y., & Picard, R. W. (2014). FEEL: A system for frequent event and electrodermal activity labeling. *IEEE Journal of Biomedical and Health Informatics, 18*, 266–277. doi:10.1109/JBHI.2013.2278213
- Bolger, N., DeLongis, A., Kessler, R. C., & Schilling, E. A. (1989). Effects of daily stress on negative mood. *Journal of Personality and Social Psychology, 57*, 808. doi:10.1037/0022-3514.57.5.808
- Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology, 56*, 267–283. doi:10.1037/0022-3514.56.2.267
- Coyne, J. C., & Gottlieb, B. H. (1996). The mismeasure of coping by checklist. *Journal of Personality, 64*, 959–991. doi:1467-6494.1996.tb00950.x
- Coyne, J. C., Stefanek, M., & Palmer, S. C. (2007). Psychotherapy and survival in cancer: The conflict between hope and evidence. *Psychological Bulletin, 133*, 367–394.
- Hagedoorn, M., Sanderman, R., Bolks, H. N., Tuinstra, J., & Coyne, J. C. (2008). Distress in couples coping with cancer: A meta-analysis and critical review of role and gender effects. *Psychological Bulletin, 134*, 1–30. doi:10.1037/0033-2909.134.1.1
- Karan, A., Wright, R. C., & Robbins, M. L. (in press). Everyday emotion word and personal pronoun use reflects dyadic adjustment among couples coping with breast cancer. *Personal Relationships, 24*(1), 36–48.
- Lakey, B., & Orehek, E. (2011). Relational regulation theory: A new approach to explain the link between perceived social support and mental health. *Psychological Review, 118*, 482–495. doi:10.1037/a0023477
- Mehl, M. R., Robbins, M. L., & Deters, F. g. (2012). Naturalistic observation of health-relevant social processes: The Electronically Activated Recorder (EAR) methodology in psychosomatics. *Psychosomatic Medicine, 74*, 410–417. doi:10.1097/PSY.0b013e3182545470
- Nazarian, D., Smyth, J. M., & Sliwinski, M. J. (2006). A naturalistic study of ambulatory asthma severity and reported avoidant coping styles. *Chronic Illness, 2*, 51–58.
- Robbins, M. L., Focella, E. S., Kasle, S., López, A. M., Weihs, K. L., & Mehl, M. R. (2011). Brief report: Naturalistically observed swearing, emotional support and depressive symptoms in women coping with illness. *Health Psychology, 30*, 789–792. doi:10.1037/a0023431
- Robbins, M. L., López, A. M., Weihs, K. L., & Mehl, M. R. (2014). Cancer conversations in context: Naturalistic observation of couples coping with breast cancer. *Journal of Family Psychology, 28*, 380–390. doi:10.1037/a0036458
- Robbins, M. L., Mehl, M. R., Holleran, S. E., & Kasle, S. (2011). Naturalistically observed sighing and depression in rheumatoid arthritis patients: A preliminary study. *Health Psychology, 30*, 129–133. doi:10.1037/a0021558

- Stone, A. A., Greenberg, M. A., Kennedy-Moore, E., & Newman, M. G. (1991). Self-report, situation-specific coping questionnaires: What are they measuring? *Journal of Personality and Social Psychology*, 61, 648–658. doi:10.1037/0022-3514.61.4.648
- Stone, A. A., Kennedy-Moore, E., & Neale, J. M. (1995). Association between daily coping and end-of-day mood. *Health Psychology*, 14, 341–349. doi:10.1037/0278-6133.14.4.341
- Stone, A. A., & Neale, J. M. (1984). New measure of daily coping: Development and preliminary results. *Journal of Personality and Social Psychology*, 46, 892–906. doi:10.1037/0022-3514.46.4.892
- Stone, A. A., Schwartz, J. E., Neale, J. M., Shiffman, S., Marco, C. A., Hickcox, M., ... Cruise, L. J. (1998). A comparison of coping assessed by ecological momentary assessment and retrospective recall. *Journal of Personality and Social Psychology*, 74, 1670–1680. doi:10.1037/0022-3514.74.6.1670
- Taylor, S. E., & Stanton, A. L. (2007). Coping resources, coping processes, and mental health. *Annual Review of Clinical Psychology*, 3, 377–401. doi:10.1146/annurev.clinpsy.3.022806.091520
- Todd, M., Tennen, H., Carney, M. A., Armeli, S., & Affleck, G. (2004). Do we know how we cope? Relating daily coping reports to global and time-limited retrospective assessments. *Journal of Personality and Social Psychology*, 86, 310–319. doi:10.1037/0022-3514.86.2.310

Suggested Reading

- Carey, A. L., Rentscher, K. E., & Mehl, M. R. (this volume). Naturalistic observation of social interactions. In K. Sweeny & M. L. Robbins (Eds.), *The encyclopedia of health psychology* (Vol. 2). West Sussex, UK: John Wiley & Sons Ltd.
- Junghaenel, D. U., & Stone, A. A. (this volume). Ecological momentary assessment for the psychosocial study of health. In K. Sweeny & M. L. Robbins (Eds.), *The encyclopedia of health psychology* (Vol. 2). West Sussex, UK: John Wiley & Sons Ltd.
- Panjwani, A. A., & Revenson, T. A. (this volume). Coping with chronic illness. In K. Sweeny & M. L. Robbins (Eds.), *The encyclopedia of health psychology* (Vol. 2). West Sussex, UK: John Wiley & Sons Ltd.
- Stagl, J. M., & Carver, C. S. (this volume). Personality and coping. In K. Sweeny & M. L. Robbins (Eds.), *The encyclopedia of health psychology* (Vol. 2). West Sussex, UK: John Wiley & Sons Ltd.
- Stephenson, E., & DeLongis, A. (this volume). Coping strategies. In K. Sweeny & M. L. Robbins (Eds.), *The encyclopedia of health psychology* (Vol. 2). West Sussex, UK: John Wiley & Sons Ltd.

Expressive Writing

Vanessa Juth¹, Danielle Arigo², and Joshua M. Smyth¹

¹*The Department of Biobehavioral Health, The Pennsylvania State University,
University Park, PA, USA*

²*Psychology Department, The University of Scranton, Scranton, PA, USA*

An Introduction to Expressive Writing

Both conventional wisdom and empirical evidence support the benefit of disclosing strong emotions. One approach to emotional disclosure is writing; emotional writing has of course long existed in creative and expressive arenas, has been used in conjunction with psychological and behavioral therapies, and occurs naturally in many contexts (Smyth & Greenberg, 2000). Over the last three decades, however, there has been specific and continued interest in a particular technique of expressive writing (EW). In this approach, individuals are asked to write about their deepest thoughts and feelings surrounding a stressful or traumatic experience. Writing typically takes place over several sessions spanning days or weeks, usually confidentially and privately. One aspect of this approach that differentiates it from other disclosure processes—especially sharing with other people—is that privately writing about personal thoughts and feelings removes socially driven feedback that may arise when talking about them with someone else (for better or worse, helpful or unhelpful advice, feeling supported or being rejected or judged, etc.). Remarkably, engaging in just 15–20 min of EW at each of three or four time points often produces psychological and physical benefit. Given the relative ease and access to EW (it essentially can be done by anyone, anywhere, and anytime), it holds potential for individual use and as an adjuvant psychosocial treatment. We discuss these points below, along with a representative summary of the EW literature.

Early Work

The Early Years of EW Research

EW was first introduced in an experimental framework by James Pennebaker and Sandra Beall (in 1986) and was seen as a means for individuals to safely express and work through their

thoughts and feelings. In their initial study, college students assigned to the experimental condition wrote about their emotions and facts related to a prior trauma; those in the control group wrote about a different, emotionally neutral topic at each writing session (e.g., time management, daily activities). Results showed that those in the experimental condition experienced better subsequent health outcomes (e.g., fewer health center visits during the 6 months following the writing task) compared with those in the control group. This seminal study began a line of research that now includes over 300 studies and introduced several procedures and practices that still impact EW work today.

First, Pennebaker and Beall (1986) established initial “guidelines” for administering EW as an intervention: participants are randomized to either an experimental condition in which they write about personally emotional topics, or to a control condition in which they write about topics that are not associated with strong emotions (e.g., daily activities). Across 3–5 days, both conditions complete writing sessions for about 15–30 min in a private room without receiving any feedback about the content of their writing. These procedures were largely used in subsequent studies for years; more recently, the specific EW methods used in research and practice have become increasingly more variable as researchers and clinicians have begun testing EW’s effectiveness in different contexts. For example, whether the specifics of the writing instructions, or the way the intervention was administered, could be altered to increase effectiveness (i.e., have stronger effects on health outcomes) or efficiency (i.e., improve health with shorter writing times or fewer writing sessions). As such, there is now tremendous variation in how EW is administered in research and clinical settings (a topic reviewed later).

Second, Pennebaker and Beall (1986) were the first to provide empirical evidence for EW’s ability to improve writers’ long-term health (in this case, several months later). As such, this study put EW “on the map,” inspiring other researchers and clinicians to use it in their work. Indeed, in the decade following the initial study, EW was tested as a means to improve a wide range of health and well-being outcomes in a variety of contexts other than college students; these included prior life trauma in adults and in adolescents, recent job loss, workplace performance and absenteeism, and psychotherapeutic and healthcare settings (Frattaroli, 2006; Smyth & Greenberg, 2000).

The number of these studies grew into a sizable literature that allowed for the use of meta-analysis, a statistical method that combines data from all available studies on a given topic to estimate an effect size, in this case, testing the effect sizes for EW improving different outcomes. The first meta-analysis of a small number of early studies suggested that overall EW had a medium effect size, which is on par with effects found for psychotherapy and other psychological interventions (Smyth, 1998). More specifically, the meta-analysis found that EW’s benefits were the strongest for physiological outcomes (e.g., immune system antibodies, liver enzyme functioning) and psychological well-being (e.g., mood, depressive symptoms) with medium to large effect sizes. Similar medium to large effects were found for self-reported health outcomes (e.g., frequency of healthcare utilization, physical symptoms) and improvements in academic/workplace settings (e.g., workplace absenteeism, reemployment, academic performance, grade point average), respectively. Finally, contrary to expectations at the time, there was no evidence that EW positively impacted a (heterogeneous) group of health-relevant behaviors (e.g., exercise activity, diet, alcohol/drug use), finding a small and nonsignificant effect size for these behaviors.

One aspect of this early meta-analysis was noteworthy—positive effects were observed not only on self-reported outcomes (e.g., how well someone said that they were feeling) but also on objectively measured outcomes (e.g., being reemployed, various biological measures, such as liver enzyme function or indicators of the immune system). Some concern had existed that

much of the effect of EW may be due to “demand characteristics” or similar reporting biases; finding positive impacts on objective outcomes greatly reduced concerns that such biases were (at least largely) driving observed effects. More generally, the positive initial studies and early meta-analytic support provided a sufficient evidentiary base to spur a large number of (often quite ingenious) studies from an array of researchers, which tested the range and limits of EW.

This first decade of EW studies had another significant implication. The growing number of studies on EW suggested that people were willing and able to write about deeply emotional and highly personal topics when participating in research studies. Even among those individuals who might be expected to shy away from fully confronting past trauma in their writing, for example, persons with posttraumatic stress disorder (PTSD), EW appears to be a feasible and acceptable treatment method. One common and expected side effect of EW is a short-term increase in distress immediately after a writing session is completed, particularly when participants write about a prior traumatic event (as opposed to EW variants, such as writing about positive experiences or benefit finding, some of the range of alterations in writing instructions are discussed later). There do not, however, appear to be long-lasting negative side effects. In fact, most participants generally describe the activity as helpful, rewarding, and enjoyable. As such, EW is seen as a feasible and safe approach to use in controlled research environments, in medical or therapeutic settings, and perhaps even in private settings (e.g., participants’ homes). Of course, as with any form of treatment or intervention tool, care needs to be taken when applying EW to sensitive samples or individuals whose health or safety may be at risk as a result of confronting their deeply personal emotions and thoughts during writing (e.g., individuals who are acutely and severely depressed or suicidal).

A New Direction: EW and Physical Health

The exciting early work in EW often studied relatively healthy, typically well-adjusted samples (with some notable exceptions, such as maximum security prisoners). Given the findings that EW could impact some biological processes, as well as subjective well-being, researchers and clinicians began to apply EW to patients with medical/health conditions. The first explicit test of EW as an adjuvant treatment in patients with medical disorders was conducted in community volunteers with mild to moderately severe asthma or rheumatoid arthritis (RA; Smyth, Stone, Hurewitz, & Kaell, 1999). Participants were randomly assigned to an EW condition or a neutral writing task. Objective disease-specific health indicators were assessed at baseline and 2 weeks, 2, and 4 months after the writing intervention; spirometry measured pulmonary functioning in asthmatic patients and clinical examinations by a rheumatologist measured RA patients’ disease status. For each of these outcomes, EW led to clinically significant improvement in a greater proportion of the sample than did control writing.

Smyth and colleagues thus found that EW was a beneficial adjuvant psychosocial treatment for improving clinical health outcomes among patients with chronic illness. This study led to a number of additional questions about the use of EW as an intervention in medical patients. In particular, could EW be useful for improving clinical outcomes in other medical conditions and contexts? Could EW help patients adjust to the diagnosis of a serious illness? Are there specific contexts in which, or individuals for whom, EW works best? How does EW lead to benefits—what are the specific mechanistic pathways (e.g., cognitive or emotional processes)? These and other questions have led to continued interest in EW as a research and clinical topic and generated a large number of studies that tested EW as a treatment for improving clinical outcomes in additional patient populations and for helping healthy individuals better adjust to life stress.

To date, the EW literature is replete with empirical studies and a growing number of systematic and meta-analytic reviews on EW. Overall, this literature shows that EW can produce fairly reliable, although modest, improvements in psychological and physical well-being. These improvements appear to exist among both healthy individuals and those suffering from physical or mental illness. It is essential to note, however, that not all studies find significant improvements in psychological and clinical outcomes. Researchers continue with efforts to identify the exact conditions under which (and the individuals for whom) EW is effective.

Given the vast scope of the EW literature, we provide below a representative (rather than exhaustive) summary of the major topic areas based on recent work and seminal studies, along with some recommendations for future research directions that may help us better understand the potential uses and limitations of EW.

Health Benefits

Medical Outcomes and Physical Functioning

As noted, evidence for EW's positive impact on medically relevant and objectively verifiable disease indicators has been found in a handful of high-quality studies. For example, several researchers found significant improvements in immune function among healthy participants and other patient samples (e.g., patients with HIV; Frattaroli, 2006). Newer findings also have replicated the benefit of EW among asthma patients (Smith et al., 2015) and have shown faster wound healing after EW (e.g., Weinman, Ebrecht, Scott, Walburn, & Dyson, 2008). More prevalent are studies showing EW's positive effects on patients' subjective physical health and functioning (rather than core indicators of disease status). These include self-reported improvements in physical mobility, functioning, and symptoms (e.g., pain, fatigue, sleep quality) and decreases in medical visits (Faller et al., 2013; Frattaroli, 2006; Harris, 2006).

Other work, however, has not found support for EW improving medical outcomes or more general physical functioning. As an example, EW has been widely administered to patients with different types of cancer as it was hoped that these patients would show health benefits similar to those found in other patients. This work has produced a large literature, with studies demonstrating some capacity for EW to improve aspects of cancer patients' self-reported health (e.g., sleep quality, pain intensity, general health); however, many other studies have not observed such improvements (Merz, Fox, & Malcarne, 2014). In part, this may be attributable to the fact that there is tremendous variability across these studies (discussed further below); for instance, different types of cancer have disparate underlying disease processes that may be differentially impacted by EW, and the way in which EW is administered in these different studies (e.g., having patients write about positive aspects of their lives since their cancer diagnosis vs. a past traumatic event, having patients carry out EW shortly after their diagnosis vs. several months later) may alter its effectiveness in improving health outcomes.

This literature on EW in cancer patients is not unique; rather, it highlights the complexity and uncertainty of trying to make sense of a complex literature testing the capacity of EW to improve health outcomes. Indeed, similar to the literature on EW in cancer, we see inconsistent findings in many other domains, including physical symptoms and general physical functioning and health behaviors (e.g., substance use; Frattaroli, 2006; Niles, Haltom, Mulvenna, Lieberman, & Stanton, 2014). In sum, the exact underlying features and mechanisms that make EW an effective intervention for improving health are not yet fully known (Smyth &

Pennebaker, 2008); as such, many reviews of its benefits on physical health are limited to conclusions along the lines of “it appears to be helpful in many cases, but not always and not for all people.”

Psychological Functioning and Well-Being

As we noted in the introduction, emotional expression more broadly defined is often associated with a range of psychological benefits, and early EW studies showed improvements in self-reported mood states (e.g., depression and anxiety levels). Since that time, EW has shown effects on various aspects of psychological functioning in healthy individuals, including reducing rumination (i.e., repeated intrusive negative thought patterns), clinical depression, and psychological distress (e.g., anxiety). One major area of exploring EW's ability improve psychological health has been conducted among individuals with chronic disease as patients often suffer from psychological comorbidities (e.g., depressive symptoms). And as the majority of approaches to EW focus on expressing thoughts and feelings about a stressful or traumatic event, there has been keen interest in its effect on posttraumatic stress (PTS) symptoms; an emerging literature tests EW as a form of treatment for military veterans who experienced combat trauma, other trauma victims (e.g., rape, vehicle accidents), and dyads coping with shared trauma (e.g., couples dealing with extramarital affairs). As is seen in the previously mentioned literature on medical outcomes and processes, evidence that EW leads to positive psychological outcomes among clinical samples and healthy populations is not uniform. That is, some work has failed to show that EW produces any benefit on aspects of psychological health and well-being among patients with chronic illness, individuals with mental disorders, and healthy persons. Further research is needed to help identify what factors determine EW's ability to help individuals better their psychological adjustment in general life or in response to a severely stressful or traumatic experience.

Interim Summary

Emerging evidence indicates that EW has some restorative physical and psychological health effects among patients with illness and among healthy individuals. Some studies, however, find benefits in areas that are not specific to one disease process (e.g., sleep, pain) or do not find positive effects for EW on health outcomes at all (or only for some subset of participants in a given study). Thus, EW is clearly not a panacea for improving disease status in medical patients, or for improving health in general. The reason(s) for the inconsistency in these findings is unclear, although moderating factors (discussed next) may play a role.

Further research on whether EW is an effective tool for improving physical and psychological health, in the general population and among individuals with physical or mental health conditions, is needed to determine the utility and parameters of incorporating EW into routine healthcare services. Ongoing research and clinical efforts will further develop our understanding of EW and how it may best be used to improve health.

Moderating Factors

Given some of the aforementioned inconsistencies in the results of EW studies, researchers have attempted to identify factors that may influence *for whom* and *under what conditions* the intervention is effective. If certain characteristics influence whether or how much EW improves

health (i.e., if moderators are identified), the intervention could be refined and targeted toward specific subgroups who may experience the greatest benefit.

Individual Differences

Theoretical and clinical perspectives regarding disclosure, emotional processing, and cognitive processes (e.g., thoughts/beliefs) pointed researchers to a wide range of individual difference factors that may influence response to EW. Indeed, evidence suggests that EW's effects are often moderated by individuals' personal characteristics, such that benefits are stronger among individuals with different characteristics (e.g., gender), dispositional traits (e.g., tendencies to engage in expressing one's emotions to others), or levels of psychological distress or disease status at time of study entry.

In addition, several studies show that participants' social environments appear to moderate EW's effects on psychological outcomes. For instance, a number of studies in cancer patients have found that individuals with higher social constraints—perceived social hindrances for disclosing thoughts and emotions—are shown to benefit more from EW than those individuals with lower social constraints (Merz et al., 2014). Findings such as these suggest that EW may be more effective for individuals who lack a supportive social environment, but questions about the exact social conditions under which EW may be most effective remain.

Other possible moderators are just emerging in the literature. For example, the early work by Smyth and colleagues (1999) found that EW was effective in patients with asthma or RA but that sample was composed of patients with mild–moderate illness severity. A recent study by Smith and colleagues (2015) conceptually replicated the earlier work in asthma patients; they found that patients with moderate illness severity obtained benefits from EW, whereas patients with very mild or very severe disease severity did not. Although clearly preliminary, such findings suggest that EW may not be helpful for improving disease status when an illness is so severe that it can no longer respond to EW, or if it is so mild that there is little room for improvement. Future studies should continue to test EW's impact on clinical health outcomes to further substantiate illness severity as a moderator and to determine whether there are other moderating illness characteristics.

Features of the Intervention: Differences in Writing Instructions as an Example

The benefits of EW are also shown to vary depending on specific features of the intervention. Although there are many details to consider, here, we cover differences in writing instructions as many studies manipulate this aspect of the intervention; through this example, we hope to demonstrate the complexity inherent to EW and the intricate details that need to be considered when administering it in a therapeutic context.

The majority of studies that have altered the writing instructions have participants write about positive aspects of past traumatic experiences, such as new things that they have come to appreciate more and that they have learned about themselves (e.g., posttraumatic growth), or new friendships that have developed (e.g., “benefit finding” or “meaning making”). Much of this work has yielded beneficial outcomes (e.g., fewer physician visits, lower psychological distress, and depressive symptoms), but it is unclear whether the mere act of writing about something “positive” means that it is personally meaningful to the participant. That is, it could be that simply writing about emotive topics, whether they are positive or otherwise, is what confers the observed health benefits. Moreover, altering the writing instructions to be about something positive may also produce processes that can influence participants' emotions,

cognitions, and/or physiology; this makes it difficult to pull apart the influence of the positive instructions versus the intervention itself.

Interim Summary

Overall, individual differences such as personality traits and medical characteristics appear to moderate EW's effects, but findings are (once again) not consistent. It is possible that EW works best for a subgroup of individuals (e.g., those with moderately severe illness, those without supportive social environments). Given the wide range of outcomes and populations in which moderation effects have been observed, much work is needed to determine which personal characteristics influence EW's effects consistently in specific contexts (e.g., past trauma vs. current trauma, chronic physical illness vs. mental health diagnosis vs. no health problems).

Moreover, the manner in which the EW is carried out procedurally may also play a role in its effectiveness. In addition to the example of highly variable writing instructions presented above, there are many other differences in how EW is administered (e.g., timing after [traumatic] event, location where the writing takes place, characteristics of the person administering the intervention, length of follow-up to when health outcomes are assessed) that likely play a role in how effective the intervention is in improving health outcomes. Ongoing and future research and clinical efforts will further develop our understanding of EW and how it may best be used to improve health.

How EW Improves Health: Potential Mechanisms

We still do not fully understand precisely how EW leads to beneficial outcomes, although many potential accounts of mechanisms exist in the literature. There are dozens of potential explanations that have been offered in the literature, and evidence that is broadly consistent with each explanation has been observed. What is considerably more difficult, however, is finding ways to show that a particular mechanism or process is uniquely important, or that EW is targeting some processes but not others.

Given that the initial EW paradigm was centered on writing about a past traumatic experience, many theories about its underlying mechanism(s) stem from various perspectives on stress and coping. One theory that emphasizes integration of cognitive and emotional processes proposed that EW helps convert trauma-related memories into language and facilitates meaning making and processing. Engaging in EW was thought to help create coherent narratives about a prior traumatic experience that would help assimilate the trauma-related memories with other memories, which, in turn, would reduce unwanted reexperiencing of the trauma and physiological arousal associated with trauma-related memories (in other words, facilitate coping), thus, improving health. A related but distinct theory focused more directly on the aspect of trauma exposure. It proposed that EW helps individuals cope with past exposures to traumatic experiences by helping them habituate to them. That is, completing multiple writing sessions about an emotionally distressing or traumatic experience repeatedly exposes participants to their negative thoughts and emotions associated with that experience, allowing negative emotions associated with that experience (and, perhaps, associated thoughts, physiology, and behaviors as well) to dissipate over time.

Other theoretical models for how EW improve health have been centered on aspects of the writing itself, specifically, in the act of translating emotional experiences into words. For

example, one perspective proposed that translating deeply personal thoughts and emotions related to a specific experience would increase people's self-awareness. Self-awareness, in turn, could then improve people's ability to regulate or stabilize their cognitive, behavioral, and especially affective responses to emotionally charged situations. Another model purported that translating abstract thoughts and emotions into written words with a linguistic structure allows individuals to better understand their past experiences in a way that could help them integrate those experiences into their understanding of the world and of themselves. Related explanations suggested that translating experiences into written language could help individuals build a narrative framework for their trauma over the course of the writing. Such a narrative framework would lead to insights and help redefine and reconceptualize traumas in a more coherent manner that allow for "letting go" of past negative experiences.

There are clearly many other explanations as well, some drawn more directly from clinical literatures addressing therapeutic approaches (e.g., cognitive behavioral-based approaches, self-regulation), and yet others that rely on more specific mediational pathways (e.g., affect labeling and neural encoding, sleep processes). Although beyond the capacity of this brief summary to review them all, this brief and selective summary is meant to demonstrate that there are numerous theoretical models for the plausible mechanisms of how EW may lead to positive health outcomes. It is also worth noting that these models are difficult to tease apart as many share a common basis (e.g., cognitive and emotional processes, translating experiences or thoughts into words). As such, evidence offered in support of one mechanism or pathway (e.g., changes in emotional responding over time) is consistent with several (even most) theoretical explanations. Finally, although there is often a desire to identify a single (parsimonious) mechanism that can explain an intervention's effects, it is likely that EW operates by activating multiple pathways. There may be a cumulative benefit through the engagement of multiple mechanisms of change (e.g., self-regulation and cognitive integration) and/or with EW producing benefits via different mechanisms for individuals in different contexts (see Smyth & Pennebaker, 2008).

Future Directions

There are certainly many remaining research questions about EW. Perhaps most notably and as is likely clear from this review, further work is needed to better understand the circumstances under which EW will be helpful. How can we be sure to get EW to the right people, in the right context, at the right time, and in the right way to optimally promote positive health and well-being?

Given the considerable variability in writing instructions and intervention parameters (e.g., length and number of sessions, timing of follow-up), it is absolutely essential that all published research studies systematically and comprehensively report methods and analytic strategies. This would allow for more accurate evaluation of whether (and how) procedural variations in EW interventions that exist between studies (e.g., location of writing, contact with and characteristics of research staff, etc.) may influence the efficacy of EW as an intervention. Future research may also consider testing EW in combination and in comparison with other more laborious, time-intensive, and costly interventions, determining the added value and relative efficacy of EW in clinical settings (see Cummings, Hayes, Saint, & Park, 2014, for review).

Future mechanisms research might consider adopting a pluralistic approach that assesses multiple mechanisms. This would allow examining important questions—for instance, moderated mediation: does EW lead to health benefits via different pathways among some

individuals or contexts versus others (e.g., men vs. women, different types of trauma, healthy persons and patients)? Similarly, there may be unique combinations of processes that lead to positive outcomes or dynamic feedback systems that unfold over time (perhaps even over different timeframes). These are all tremendously important and exciting areas for future EW research.

Summary

Expressive writing (EW) allows individuals to freely disclose their personal thoughts and emotions about a strongly emotional experience (typically, but not always, one that is stressful or traumatic); this is usually done in a private, “safe” environment that removes the risk of negative or socially stigmatizing feedback. Researchers have used EW to facilitate positive health outcomes in college students, healthy community residents (sometimes with particular challenges, such as unemployment), and patients with medical illnesses (e.g., asthma, PTSD, and cancer, among others).

In general, existing empirical evidence suggests that EW produces modest but fairly reliable, physical, and psychological health benefits. There is also, however, evidence that EW is more or less effective in some contexts and is more beneficial for some people than others. A challenge for future research is to systematically test moderating factors that may help optimize EW’s benefits. Although the underlying processes by which EW confers its health benefits are not yet fully clear, multiple theoretical models provide a basis for testing plausible mechanisms, some of which may act simultaneously or sequentially. EW thus remains an area of theoretical and clinical interest, and further examination of its potential as an adjuvant treatment in medical and therapeutic settings is warranted.

Author Biographies

Vanessa Juth, PhD, MPH, is a postdoctoral researcher at the Pennsylvania State University in the Department of Biobehavioral Health. Her research identifies intra- and interindividual processes linking stress and trauma to health to inform interventions for preventing disease and fostering resilience within individuals and their social environments. Her recent publications examine how interpersonal contexts relate to health outcomes while coping with adverse experiences including chronic disease, loss of a loved one, and natural disasters.

Danielle Arigo, PhD, is an assistant professor of psychology at the University of Scranton in Scranton, PA, (USA), and is a licensed clinical psychologist in the state of Pennsylvania. Her research investigates the role of social processes in health and health behavior. This work emphasizes methods for optimizing online social networks and affective approaches such as EW, to promote health. She has coauthored several articles and book chapters relevant to clinical applications of EW.

Joshua M. Smyth, PhD, is a professor of Biobehavioral Health and of Medicine at the Pennsylvania State University. His work encompasses three broadly defined areas: (a) What are the effects of stress on health? (b) Can we assess stress, affect, and health in an ecologically relevant manner as it unfolds over time? (c) Can psychological interventions improve health and well-being? His recent work synthesizes these areas to use real-time, ambulatory data capture to dynamically tailor the implementation and delivery of in situ dynamic and adaptive interventions to promote health and well-being.

References

- Cummings, J. A., Hayes, A. M., Saint, D. S., & Park, J. (2014). Expressive writing in psychotherapy: A tool to promote and track therapeutic change. *Professional Psychology: Research and Practice*, 45, 378–386. doi:10.1037/a0037682
- Faller, H., Schuler, M., Richard, M., Heckl, U., Weis, J., & Küffner, R. (2013). Effects of psychoncologic interventions on emotional distress and quality of life in adult patients with cancer: Systematic review and meta-analysis. *Journal of Clinical Oncology*, 31, 782–793. doi:10.1200/JCO.2011.40.8922
- Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin*, 132, 823–865. doi:10.1037/0033-2909.132.6.823
- Harris, A. H. (2006). Does expressive writing reduce health care utilization? A meta-analysis of randomized trials. *Journal of Consulting and Clinical Psychology*, 74, 243–252. doi:10.1037/0022-006X.74.2.243
- Merz, E. L., Fox, R. S., & Malcarne, V. L. (2014). Expressive writing interventions in cancer patients: A systematic review. *Health Psychology Review*, 8, 339–361. doi:10.1080/17437199.2014.882007
- Niles, A. N., Haltom, K. E., Mulvenna, C. M., Lieberman, M. D., & Stanton, A. L. (2014). Randomized controlled trial of expressive writing for psychological and physical health: The moderating role of emotional expressivity. *Anxiety, Stress, and Coping*, 27, 1–17. doi:10.1080/10615806.2013.802308
- Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology*, 95, 274–281. doi:10.1037/0021-843X.95.3.274
- Smith, H. E., Jones, C. J., Hankins, M., Field, A., Theadom, A., Bowskill, R., ... Frew, A. J. (2015). The effects of expressive writing on lung function, quality of life, medication use, and symptoms in adults with asthma: A randomized controlled trial. *Psychosomatic Medicine*, 77, 429–437. doi:10.1097/PSY.0000000000000166v
- Smyth, J. (1998). Written emotional expression: Effect sizes, outcome types, and moderating variables. *Journal of Consulting and Clinical Psychology*, 66, 174–184. doi:10.1037/0022-006X.66.1.174
- Smyth, J., & Greenberg, M. (2000). Scriptotherapy: The effects of writing about traumatic events. In J. Masling & P. Duberstein (Eds.), *Psychodynamic perspectives on sickness and health* (pp. 121–154). Washington, DC: American Psychological Association press.
- Smyth, J., & Pennebaker, J. (2008). Exploring the boundary conditions of expressive writing: In search of the right recipe. *British Journal of Health Psychology*, 13, 1–7. doi:10.1348/135910707X260117
- Smyth, J., Stone, A., Hurewitz, A., & Kaell, A. T. (1999). Effects of writing about stressful experiences on symptom reduction in patients with asthma or rheumatoid arthritis. *JAMA: The Journal of the American Medical Association*, 281(14), 1304–1309. doi:10.1001/jama.281.14.1304
- Weinman, J., Ebrecht, M., Scott, S., Walburn, J., & Dyson, M. (2008). Enhanced wound healing after emotional disclosure intervention. *British Journal of Health Psychology*, 13(1), 95–102.

Suggested Reading

- Cummings, J. A., Hayes, A. M., Saint, D. S., et al. (2014). Expressive writing in psychotherapy: A tool to promote and track therapeutic change. *Professional Psychology: Research and Practice*, 45, 378–386. doi:10.1037/a0037682
- Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin*, 132, 823–865. doi:10.1037/0033-2909.132.6.823
- Lepore, S., & Smyth, J. (Eds.) (2002). *The writing cure: How expressive writing promotes health and emotional well-being*. Washington, DC: American Psychological Association Press.
- Pennebaker, J., & Smyth, J. (in press). *Opening up: The healing power of words*. New York, NY: Guilford Press.
- Smyth, J. M., Stone, A. A., Hurewitz, A., & Kaell, A. (1999). Effects of writing about stressful experiences on symptom reduction in patients with asthma or rheumatoid arthritis: A randomized trial. *Journal of the American Medical Association*, 281, 1304–1309. doi:10.1001/jama.281.14.1304

Failed Control of Health Behavior

Melissa V. Auerbach and Marci Lobel

Department of Psychology, Stony Brook University, Stony Brook, NY, USA

Addiction Risk

Health behaviors provide a potent context for examining self-control failure, as many health-relevant behaviors, including food consumption, sleep, substance use, and sexual behaviors, affect critical outcomes such as physical health, longevity, and even survival. Cigarette smoking is a particularly common topic when studying self-control failure. Millions of Americans try to quit each year, yet a large majority of these attempts fail within the first month (Dube, Asman, Malarcher, & Carabollo, 2009). These high rates of failed smoking cessation are due not only to the physiological addiction to nicotine but also to the emotionally and behaviorally addictive properties of cigarette smoking. Overconsumption of food and alcohol are also considerable domains of self-control failure, as over time and with repeated failures of self-control, such behaviors may become chronic and evolve into addictions (Bland, Melton, Welle, & Bigham, 2012) especially when a person's motives for these behaviors change or intensify (Hustad, Carey, Carey, & Maisto, 2009). For example, it has been shown that moderate drinkers who consume alcohol for social reasons are at risk for addiction if they start drinking in order to cope with stress.

However, there has been little attention toward understanding why people who are not addicted fail at controlling their health behavior. College students are a particularly important group to examine, as behaviors such as drinking and overeating are considered socially normative in college (Bland et al., 2012) but can become chronic and impair health and well-being. This entry examines behavioral control among nonaddicted people to further our understanding of the origins of addiction risk.

Social Cognitive Theories

Health behavior theories such as the health belief model and the theory of planned behavior are often used to investigate health behavior among nonaddicted individuals. These approaches

are especially valuable in describing the uptake of health-promoting behaviors such as exercise and eating healthfully. Many health behavior theories emphasize goal setting as an instrumental first step in implementing self-control processes (Legault, Gutsell, & Inzlicht, 2011). Cybernetics is an approach that has been used to model self-control processes in humans, reducing control to three components: goal setting, behavioral monitoring, and behavior implementation. For example, according to cybernetic principles of self-control, dieters first set a specific goal for the kinds of food they want to eat. Next, they monitor their eating behavior for instances when it is discrepant from their goals. If any discrepancies are detected, they change their behavior (e.g., “Put down the chips and grab the broccoli!”; Legault et al., 2011). Goal setting creates a discrepancy between what one wants to do and what one is currently doing, and this discrepancy is what sets self-control in motion.

Health behavior theories generally do not explain the adoption of health-impairing behaviors such as smoking or binge drinking. Furthermore, by focusing on future outcomes of health behavior, prevailing theories tend to underestimate the role of the present moment as a factor that influences behavior (Darlow & Lobel, 2012; de Ridder & de Witt, 2006). Healthy intentions can be overruled by acute reactions to challenging situations. For example, even for individuals who regularly exercise and eat healthfully, vulnerability during periods of stress may lead them to succumb to vices such as unhealthy snacking. Furthermore, because most people are involved in multiple goal pursuits and engaged in many roles concurrently, they often need to divide their resources. Focusing on health behavior in isolation is a limited approach, as other life responsibilities compete for time, energy, effort, and other resources. Therefore, it is important to take into account the influence of situational factors (e.g., stress and fatigue) involved in the immediate context when examining control of health behavior.

Self-Control Theories

Self-control theories are applicable when considering the influence of situational factors involved with health-impairing behavior. For example, studies show that self-control is influenced by previous attempts at self-control, by mood, and by working memory capacity. Therefore, health behavior can be compromised when other contextual factors, such as stress and fatigue, impede efforts at self-control.

A widely accepted perspective on self-control is that it can be characterized by two systems of behavior (e.g., Dvorak & Simons, 2009). One system involves control over goal-oriented behaviors, such as eating nutritiously or abstaining from smoking to promote good health. This control of goal-oriented behavior tends to focus on long-term outcomes such as improvements in health. The second system involves appetitive or hedonistic behaviors that serve more immediate and contextually related goals, often to alleviate stress, improve mood, or derive pleasure (Vohs & Heatherton, 2000). This immediate system includes responses to temptations and cravings, such as consuming highly palatable and easily available foods (de Ridder & de Witt, 2006). Dual-process theories tend to separate cognitive and emotional processes: the immediate system is more emotionally reactive to context (e.g., smoking a cigarette in response to distress), and the goal-oriented one is more cognitively effortful (e.g., exercising to promote longevity). Therefore, according to this perspective, when people experience self-control failure, they are unable to override dominant, hedonistic response tendencies despite their desire to regulate behavior or maintain health goals.

The “hot/cool system” theory (Metcalf & Mischel, 1999) is one relevant example of a dual-process theory of self-control. This theory suggests that the “hot system” pertains to

resisting temptation or impulses. The “cool system” pertains to acting on planned and premeditated actions. The cool system relies on long-term executive function resources, such as reasoning and strategizing, and is therefore considered a cognitively based system, whereas the hot system is emotionally based as it deals with immediate reactions to the context. This theory suggests that the cool system functions less when individuals are confronted with impulses requiring immediate control and that the hot system is used to pursue short-term, immediate goals (de Ridder & de Witt, 2006).

Strack and Deutsch (2004) suggested a similar dual-process theory of self-control. One mode, termed “effortful,” tends to rely heavily on information processing and regulatory control. The other mode, termed “impulsive,” is reactive and responds to emotional situations. Dual-process approaches thus suggest that when individuals struggle with self-control, they experience conflict between competing behavioral tendencies, one being more emotionally reactive (e.g., smoking a cigarette during a time of stress) and the other being more cognitively effortful and goal oriented (e.g., smoking cessation).

Objectives

Below we extend existing theories of self-control to examine why people fail at controlling their health behavior. *We define failed control of health behavior as compromising distal health goals for more proximal, immediately rewarding outcomes.* We theorize that failed behavioral control is composed of cognitive and emotional factors that are unique and somewhat independent of one another but also interrelated. First, we expand upon the role of the immediate context involved in contemporary theories of self-control failure. Recent self-control theories explain that failure to act in accordance with one’s long-term goals is the result of emotional impulses taking precedence over cognitive, effortful considerations (de Witt Huberts, Evers, & de Ridder, 2014). Although emotionally reactive impulses are often a leading predictor of failed behavioral control, we posit that people may sometimes abandon their long-term goals by employing cognitive strategies that invoke the present moment (e.g., “The future is uncertain. Eat dessert first.”). We will thus explore cognitive strategies involved with immediate fulfillment that have received little attention in prior work. Second, we aim to integrate cognitive and emotional factors that have previously been underemphasized in research on failed health behavioral control. Further research is needed to understand how these two systems (emotional and cognitive) operate together, particularly to understand failed control of health behavior.

Cognitive Factors that Lead to Immediate Fulfillment

Dual-process theories posit that emotionally driven and/or impulsive factors are the primary explanation for why people opt for an immediate reward rather than adhering to their long-term goals. However, we argue that the dual-process approach is limited, as both the emotional and cognitive systems may both be independently involved with immediate reward fulfillment. We posit that there are two unique cognitive systems involved with health behavioral control: one that is effortful and aimed at providing attentional awareness toward long-term health goals, as discussed in existing dual-process theories, and a second that is less well studied according to the dual-process perspective, which consists of moment-salient behaviors that tend to involve less effort or deliberation than goal-oriented cognition. Below we discuss cognitively driven moment-salient behavior.

Theory of Discounting

The theory of discounting (Rachlin, Raineri, & Cross, 1991) is one cognitive approach that suggests people may perceive the moment as more important than the future, which is distant and uncertain. The more an individual discounts the future, the more likely they will be to choose a short-term option over a long-term one (Rachlin et al., 1991). Therefore, this theory helps to explain why the moment may be prioritized over long-term health goals. Stritzke, Breiner, Curtin, and Lang (2004) found that smokers tend to hold conflicting beliefs: they believe it is foolish to risk their health by continuing to smoke, yet they also believe life is uncertain so they might as well enjoy the moment. This study indicated that some people believe that it is highly uncertain that they will collect a long-term reward, so they might as well choose the more immediate reward. Thus, people may temporarily suspend their long-term goals when faced with certain contextual demands.

Automatic Decisions

Quick and automatic decisions may also compel someone to abandon their long-term goals and to adhere to the moment. It is unlikely that all the choices people make necessitate the active involvement of the self. Many choices that are made, especially those that are quick and effortless, may not be deliberate. For example, skipping breakfast in the morning is considered to be an unhealthful behavior as it increases one's appetite throughout the day, deregulates glucose levels, and often leads to subsequent overeating and weight gain. However, the behavioral effects of skipping breakfast are often overlooked by individuals, especially young adults. Although automatic behaviors such as skipping breakfast are typically easy and effortless, the impact of such behaviors can jeopardize long-term health goals.

Habits are another pertinent aspect of automatized behaviors. In general, people tend to behave in routinized, repetitive, and mindless ways (Langer & Moldoveanu, 2000), which may lead to the development of poor health habits. Unconscious habits, or mindless behaviors repeated over and over, may lead one to rely heavily on established patterns (Langer & Moldoveanu, 2000). For example, it has been shown that many smokers who relapse are not experiencing negative affect but are smoking outside of their awareness (e.g., Tiffany, 1990). Poor health habits are also likely to persist if one is unaware of the long-term costs associated with these behaviors. For example, research indicates that heavy drinkers do not pay attention to the "warning signs" of previous alcohol consequences, such as poor judgment or alcohol-related accidents, and do not adjust their drinking patterns to avoid them further (Hustad et al., 2009). Such poor health habits can thus be outside of awareness and may be antithetical toward health goals.

Integrating Cognitive and Emotional Perspectives

Dual-process approaches suggest that the cognitive, effortful system is invoked less when individuals are confronted with impulses requiring immediate control and that the emotionally reactive system is used to deal with short-term, immediate goals (de Ridder & de Witt, 2006). Thus, these theories separate cognitive and emotional processes. However, this distinction is a limitation because there is considerable evidence that cognitive and emotional processes work in concert to influence behavior (Dvorak & Simons, 2009). According to contemporary theorists, emotion and cognition are fully integrated and without clear demarcation in the human

brain (e.g., Inzlicht, Bartholow, & Hirsh, 2015). Individuals confronted with an emotional impulse may experience two simultaneously occurring cognitive processes: a declined functioning of the goal-oriented cognitive system and a cognitive appraisal process. Cognitive appraisals help determine emotional responses, and the full experience of emotion typically includes thought and action impulses (Lazarus, 1982). Thus, health behavioral control is likely to elicit simultaneous occurrence of emotional and cognitive processes.

Cravings and Temptations

Momentary cravings and desires can trigger both emotional and cognitive processes involved with failed control. Cravings, or intense desires for a particular substance, are highly salient psychological phenomena that influence behaviors like snacking and lead to weakened self-control. Individuals with a goal to lose weight may at first be cognitively effortful as they inhibit their cravings for fatty snacks. However, after multiple occurrences of controlled and healthful eating, this inhibitory system may eventually break down either due to an overpowering stressor or due to cognitive overload. Cravings can be so powerful that they may temporarily alter attitudes or perceptions toward a substance. In one study (Sayette & Hufford, 1997), cigarette smokers assigned to a “high urge” condition abstained from cigarettes for a few hours; “low urge” participants had recently smoked a cigarette. Then all were instructed to list as many positive characteristics of smoking as they could within 90 s, followed by a 90-s period in which they listed negative characteristics. More positive items were generated by high urge smokers than by low urge smokers. Thus, cravings can influence attitudes toward a desired substance, at least in the short term. Selective accessing of positive information related to an unhealthful behavior such as cigarette use may increase the likelihood of that behavior (Sayette & Hufford, 1997).

Environmental cues may also integrate cognitive and emotional processing in ways that lead to failed control. Temptations toward unhealthful behavior are ubiquitous in modern society and include frequent exposure to environmental cues such as fast food advertising. Such temptations, particularly if they are continuous, can undermine attempts to refrain from unhealthful behavior such as overeating. When people are in a “hot” visceral or emotional state, the presence of temptation directs their cognition toward an impulsive action. Yet people are often unaware that the stimuli in their environment may be spontaneously activating these cognitive processes that determine their behavior. Additionally, the restraint involved in day-to-day temptations one experiences can deplete self-control. Other environmental cues, including the observed actions of companions or outright social influence by them, can trigger failure in controlling one’s health behavior in the current moment and also undermine long-term goals such as weight loss, physical fitness, and low cholesterol because these often require consistent adherence across time.

Intrapersonal Resources

Mental fatigue is another powerful contributor to failed self-control that has been found to be associated with cognitive processes. Mental fatigue refers to feelings that people may experience during or after prolonged periods of cognitive activity. These feelings are very common in everyday modern life and generally involve tiredness or even exhaustion, an aversion to continuing with the present activity, and a decrease in the level of commitment to the task at hand. Although considered to be a negative affective state, mental fatigue is often discrepant with an overarching goal and may be regulated by engaging with sources of immediate

gratification. One of the more influential theories to come from research on fatigue is the limited strength model, which suggests that all of the resources available for self-control are in a single domain (Vohs & Heatherton, 2000). In other words, the resources required for behavior like attentional control are the same as those that are used for unhealthy behavior like overeating. This model posits that self-regulatory strength is a finite, renewable resource that is drained when people attempt to regulate their emotions, thoughts, or behaviors. For example, stressful situations can be depleting and lead individuals to poorly regulate during subsequent attempts of self-control. Thus, the emotional and cognitive processes driven by fatigue are highly integrated as they pool from the same domain-general source.

Similar to fatigue, stress has shown to be an emotionally potent factor in failed behavioral control that can exhaust an individual's personal resources. Situational factors like stress may make individuals more responsive to cognitive appraisal and less responsive to goal-oriented processes. In particular, acute stress due to an emotionally demanding event may diminish the importance of long-term goals while increasing the salience of the immediate context. This process can explain why acute stress often leads to relapses of smoking and drinking as well as to diet breaking (Baumeister, Heatherton, & Tice, 1994). Baumeister et al. (1994) suggested that affectively charged moments focus attention on immediate stimuli, decreasing the resources needed to attend to higher-level goals. In other words, it may be difficult for people to be aware of their goal-oriented behavior when emotionally charged situations demand a focus on immediate stimuli.

Although some instances of self-control failure are emotionally driven and not cognitive or deliberate, there are also instances of stress-related failed control that involve deliberate decisions influenced by cognitive appraisal. For example, if a man is on a diet and makes a conscious decision to eat an unhealthy dessert everyday by thinking he "deserves" it for all the stress he has endured, this reasoning indicates that his appraisal processes may have overpowered his long-term diet goals. Similarly, if a woman with the goal of reducing alcohol consumption has successfully regulated her alcohol intake for a week, she might "reward" herself by having a drink. Therefore, certain cognitive appraisal processes may lead individuals to fail at self-control by justifying a break from healthy goal pursuits.

Threatening emotional states can also produce a bias toward short-term thinking. This bias may be considered adaptive, as responding to the present context is sometimes more important for immediate survival than its long-term, accumulated consequences. Research on emotion-focused coping offers insight as to why people may prioritize substances that help ameliorate the immediate context. Alcohol is thought to serve as a form of immediate self-medication, reducing negative emotional states for some individuals. Similarly, cigarette smokers often cite anxiety reduction as a primary reason for smoking, and escape from negative affect has been found to be an essential motive for drug use.

Similarly, improving a negative mood may also be triggered by a cognitive appraisal process that makes contextual demands particularly salient. Negative mood signals a need for action, often leading one to search for strategies that will improve the mood or one's environment. Although unhealthy, mood-improving behavior tends to be motivated by an emotionally impulsive system, it also may occur due to a conscious choice to suspend one's long-term health goals. Unlike bouts of quick intense emotion, negative moods can last for several hours or days.

A continuous, persisting negative mood can also lead to negative social and work-related consequences, as it impairs emotional inhibition and concentration. Therefore, unhealthy behavior may be a deliberate solution to relieve a persistent negative state. Research on "negative state relief" and tension reduction illustrates that people are more willing to participate in risky unhealthy behavior when attempting to ameliorate or avoid experiencing negative mood.

The perceived duration of one's mood can also determine whether or not to engage in unhealthful behavior. Tice, Bratslavsky, and Baumeister (2001) found that after inducing a negative mood, those who believed their mood was modifiable engaged in impulsive behaviors such as eating fattening snacks. However, when participants believed that their affective state was more permanent or long-lasting, the desire for impulsive behaviors diminished. Thus, people's perceptions of the duration of their negative moods can determine whether or not they should indulge in immediate forms of gratification to make themselves feel better.

Conclusion

There is growing interest in self-control failure as a risk factor for addictive behaviors such as alcohol abuse and overeating in individuals who are not currently addicted. Over time and with repeated failures of self-control, unhealthful behavior may become chronic and evolve into addiction. Self-control failure has previously been theorized as a dual-process phenomenon in which cognitive and emotional processes are separate. However, these theories are simplistic and have been poorly integrated. Considerable evidence suggests that emotional and cognitive processes are mutually causative and work in concert with social and situational factors to influence behavior. Together with the cognitive, emotional, and social processes that enable or inhibit self-control, a variety of relevant psychological factors including momentary cravings, mental fatigue, and stress also help to explain why people fail at controlling their health behavior.

Author Biographies

Melissa V. Auerbach, MA, is a doctoral candidate in social and health psychology at Stony Brook University. Her research has focused on psychosocial aspects of health behaviors among a variety of populations, including college students and pregnant women. A recent study demonstrated the conceptual and empirical value of differentiating health-promoting and health-impairing behavior in pregnant women. An ongoing project will test the model of failed self-control that is articulated in this entry.

Marci Lobel, PhD, is professor of psychology and director of the Doctoral Program in Social and Health Psychology at Stony Brook University. Her research investigates social psychological factors such as stress, coping, and perceived control that influence physical health and well-being. She is currently conducting studies of the health effects of discrimination experienced by pregnant women of color and the ways that women cope with discrimination and other types of stress during pregnancy.

References

- Baumeister, R. F., Heatherton, T. F., & Tice, D. M. (1994). *Losing control: How and why people fail at self-regulation*. San Diego, CA: Academic Press.
- Bland, H. W., Melton, B. F., Welle, P., & Bigham, L. (2012). Stress tolerance: New challenges for millennial college students. *College Student Journal*, 46, 362–375.
- Darlow, S., & Lobel, M. (2012). Smoking behavior and motivational flexibility in light and heavy smokers. *Addictive Behaviors*, 37, 668–673. doi:10.1016/j.addbeh.2012.02.001
- de Ridder, D., & de Witt, J. (Eds.) (2006). *Self-regulation in health behavior*. Essex, UK: Wiley.

- de Witt Huberts, J. C., Evers, C., & de Ridder, D. T. (2014). "Because I am worth it": A theoretical framework and empirical review of a justification-based account of self-regulation failure. *Personality and Social Psychology Review*, 18, 119–138. doi:10.1177/1088868313507533
- Dube, S., Asman, K., Malarcher, A., & Caraballo, R. (2009). Cigarette smoking among adults and trends in smoking cessation—United States, 2008. *Morbidity and Mortality Weekly Report*, 58, 1227–1232. Retrieved from <http://www.cdc.gov/mmwr/PDF/wk/mm5844.pdf>
- Dvorak, R. D., & Simons, J. S. (2009). Moderation of resource depletion in the self-control strength model: Differing effects of two modes of self-control. *Personality and Social Psychology Bulletin*, 35, 572–583. doi:10.1177/0146167208330855
- Hustad, J. T., Carey, K. B., Carey, M. P., & Maisto, S. A. (2009). Self-regulation, alcohol consumption, and consequences in college student heavy drinkers: A simultaneous latent growth analysis. *Journal of Studies on Alcohol and Drugs*, 70, 373–382. PMID: PMC2670743
- Inzlicht, M., Bartholow, B. D., & Hirsh, J. B. (2015). Emotional foundations of cognitive control. *Trends in Cognitive Sciences*, 19, 126–132. doi:10.1016/j.tics.2015.01.004
- Langer, E. J., & Moldoveanu, M. (2000). The construct of mindfulness. *Journal of Social Issues*, 56, 1–9. <http://dx.doi.org/10.1111/0022-4537.00148>
- Lazarus, R. S. (1982). Thoughts on the relations between emotion and cognition. *American Psychologist*, 37, 1019–1024. <http://dx.doi.org/10.1037/0003-066X.37.9.1019>
- Legault, L., Gutsell, J. N., & Inzlicht, M. (2011). Ironic effects of antiprejudice messages: How motivational interventions can reduce (but also increase) prejudice. *Psychological Science*, 22, 1472–1477. doi:10.1177/0956797611427918
- Metcalf, J., & Mischel, W. (1999). A hot/cool-system analysis of delay of gratification: Dynamics of willpower. *Psychological Review*, 106, 3–19. <http://dx.doi.org/10.1037/0033-295X.106.1.3>
- Rachlin, H., Raineri, A., & Cross, D. (1991). Subjective probability and delay. *Journal of the Experimental Analysis of Behavior*, 55, 233–244. doi:10.1901/jeab.1991.55-233
- Sayette, M. A., & Hufford, M. R. (1997). Effects of smoking urge on generation of smoking-related information. *Journal of Applied Social Psychology*, 27, 1395–1405. doi:10.1111/j.1559-1816.1997.tb01604.x
- Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and Social Psychology Review*, 8, 220–247. doi:10.1207/s15327957pspr0803_1
- Stritzke, W. G., Breiner, M. J., Curtin, J. J., & Lang, A. R. (2004). Assessment of substance cue reactivity: Advances in reliability, specificity, and validity. *Psychology of Addictive Behaviors*, 18, 148–159. <http://dx.doi.org/10.1037/0893-164X.18.2.148>
- Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: If you feel bad, do it! *Journal of Personality and Social Psychology*, 80, 53–67.
- Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. *Psychological Review*, 97, 147–168. <http://dx.doi.org/10.1037/0033-295X.97.2.147>
- Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: A resource-depletion approach. *Psychological Science*, 11, 249–254. doi:10.1111/1467-9280.00250

Suggested Reading

- de Witt Huberts, J. C., Evers, C., & de Ridder, D. T. (2014). "Because I am worth it": A theoretical framework and empirical review of a justification-based account of self-regulation failure. *Personality and Social Psychology Review*, 18, 119–138. doi:10.1177/1088868313507533
- Dvorak, R. D., & Simons, J. S. (2009). Moderation of resource depletion in the self-control strength model: Differing effects of two modes of self-control. *Personality and Social Psychology Bulletin*, 35, 572–583. doi:10.1177/0146167208330855
- Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. (2009). The strength model of self-regulation failure and health-related behaviour. *Health Psychology Review*, 3, 208–238. doi:10.1080/17437190903414387

The Geography of Health

Arseny A. Ryazanov¹, Kirstie K. Russell², and
Nicholas J. S. Christenfeld¹

¹*Department of Psychology, University of California San Diego, La Jolla, CA, USA*

²*Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada*

Many factors contribute to, or subtract from, human health and longevity, from before conception to the moments before death, from what one eats and breathes, to what sports one enjoys, and what one drives and how one drives it. One factor that has received considerable attention is geography. Where you live predicts where, when, and why you die.

While there are myriad of ways that geography can be linked to health, they can be usefully, if roughly, divided into two broad categories. The first is that geographical location can be a proxy for differences in demographics, based on current and historical immigration patterns. That is, certain kinds of people, prone to particular sorts of ailments, are concentrated in particular geographic regions. We refer to this as an indirect geographical factor. The other is that geographical features have an impact on the health of residents. Living in a certain place can change the occupations, exposures, and habits of residents. We term this a direct geographical factor.

This distinction between geography as demography, through attracting different sorts of people, and geography impacting people after they live in a particular place is not as simple as the latter being an argument for geography being causal and the former being just a marker. It is instead that the relationship between region and health can differ. For example, a sunny region might attract retired people, who could be more likely to die from heart attacks and less likely to die from choking on a Lego brick. It would not be sun exposure per se that protects one from the dangers of toy asphyxiation. Nonetheless, making an area sunnier (we do not take a stand here on anthropogenic climate change, but just offer this as a hypothetical) could reduce that cause of death for its occupants over time. The causal pathway in this case would be changing immigration patterns. On the other hand, adding bike lanes to urban streets could lower heart attack mortality, with a causal impact on the cardiovascular health of current residents.

Both mechanisms clearly have public health implications, though those implications do differ in an important way. Promoting the health of residents is a central civic function. Attracting

healthy residents can also be useful locally, even if it does not make any overall difference at a larger scale because people are no healthier on the whole, just differently distributed. In the same way, attracting wealthy people to a particular state can enhance that state's tax revenues, even though that gain comes with a parallel loss in revenue to the state from which those people moved.

Causal Geography

Distinguishing between geography's influence on who lives in a particular place and how it changes those who live there is not a simple matter. It is certainly possible, for example, that a factor could have both effects. Adding bicycle lanes to a city could attract more fit bicyclists to that city and also make the people who live there more likely to become fit bicyclists. To further complicate matters, cities that add bicycle lanes might tend to be those whose residents are already keen on bicycling and so vote and lobby for such lanes. In this case, cardiovascular health would cause the change in geography.

There are several ways that geographic factors can be potent predictors of health outcomes without being causally involved. One is that geographic factors can be just markers. Cities with more bicycle thefts, for example, may be the ones with greater cardiovascular health of their residents. This would likely be just a reflection of the greater number of bicycles in use, and not a sign that encouraging thieves, perhaps by providing free bolt cutters, would be a useful public health program. Likewise, factors that may have been historically causal may no longer be so. Regional differences in climate or amenities that influenced ethnic migration patterns that are reflected in demography even today may no longer have any causal role, as, for example, those influenced by much immigration of a certain period passing through Ellis Island.

One way to increase confidence in a factor playing a causal role in health, short of experimental manipulation, is to show that its effect persists when controlling for the likely confounding variables. In this way, for example, one might explore whether caffeine intake still predicted negative health outcomes when controlling for smoking—which covaries with coffee drinking and is itself clearly causal. However, this approach carries a conceptual risk of controlling not just for confounds but also for possible mediators of the effect. In this case, one would potentially wrongly conclude that the variable of interest was not causal and was just a marker. To stick with our bicycle lane example, one might be interested in exploring heart disease risks of populations in cities with and without bicycle lanes. At the start of the study, concerned that cities could differ in other ways, one might statistically control for differences in reported diet and non-bicycle exercise habits. Should the association between health and bicycle lanes vanish in such an analysis, one could conclude that healthy populations like bicycle lanes, but that they are not causal, and so adding them would not enhance the population's health. However, it could be that the bicycle lanes are causally important and that their effect is through the impact they have on people's orientation toward diet and exercise. That is, bicycle lanes might encourage people to eat more healthily and generally to exercise more.

If one is interested in how resources should be allocated, questions of causality are not essential. It may also not be critical if the causal factor is not one over which one has control—such as the number of hours of daylight during the winter months or the suitability of the terrain for skiing. However, in designing interventions of controllable factors to promote public health, understanding and establishing the causal pathway becomes critical.

Indirect Geographical Factors: Regional Demographic Differences in Health

In reviewing some of the research on regional demography and health, we will confine ourselves to domestic findings, though clearly there are enormous differences between countries on important outcomes, from infant mortality to life expectancy. It is useful, when exploring the question of geography's connection to health, to consider how regions in the United States differ in terms of their demographic makeup. Demographic makeup of a population consists of several factors including age structure, race, and ethnicity. The following serve as examples of how the three are associated with varying health outcomes.

Disability and Age

Increased lifespans put larger demands on public health services, particularly in terms of chronic illnesses, injuries, and disabilities. As the US and world populations age, health problems shift to these focuses. Global trends aside, regions in which the population has a higher concentration of elderly people exhibit more age-related health problems than regions with younger populations. In Utah, for example, 32% of the population is between the age of 0 and 18, and 11% is over the age of 65. In Florida, on the other hand, an attractive destination for retirees, only 22% of the population is between 0 and 18, and 17% is over the age of 65 (Kaiser Family Foundation, 2013). Consistent with the demographic differences, Florida has a higher rate of disability than Utah (Erickson, Lee, & von Schrader, 2014a, 2014b). Thus, variation in age structure between regions can contribute to age-related health disparities.

Sickle Cell Disease and African Americans

African Americans of sub-Saharan descent are particularly vulnerable to a genetic condition known as sickle cell disease: 1 out of every 500 African Americans is born with the condition (Centers for Disease Control and Prevention [CDC], 2015). This disorder can result in cerebrovascular disease, cognitive impairment, and even death in affected children. African Americans represent larger portions of the population in the southeast United States (CDC, 2015), and this region reports larger estimated populations of individuals affected by the condition than other areas. Concentrations of demographic groups with genetic predispositions to particular diseases can thus contribute to regional health disparities as well.

Liver Cancer and Asians

Asian Americans are almost three times more likely to develop liver cancer than Caucasians (Lin, Chang, & So, 2007). While liver cancer is not a particularly common cause of mortality in the United States in general, it is the second leading cause of mortality among Asian American men (Lin et al., 2007). Asian Americans are most heavily concentrated in the American West, in states such as California and Hawaii. Consistent with the demographic distribution, these regions experience elevated liver cancer mortality. As with the regional differences in sickle cell disease, the combination of Asian American men's predisposition to liver cancer and their geographic concentration leads to varied health outcomes between regions.

Tay–Sachs and Ashkenazi Jews

Tay–Sachs disease, a genetic lipid storage disorder caused by the lack of a vital enzyme, causes children to lose motor skills and mental functions. Although children afflicted by Tay–Sachs disease usually appear healthy at birth, by about 6 months of age, they generally start to show signs of slowed development. This genetic disease is most common among individuals of Eastern European Jewish (Ashkenazi) descent (National Human Genome Research Institute [NHGRI] 2011). Other Jewish groups and non-Jewish groups are a hundred times less likely to be afflicted by this disease. While 1 in 27 Jews is a carrier of the Tay–Sachs gene, only 1 in 250 people in the general population is a carrier. Since both parents must be carriers for the offspring to be affected, the 10-fold carrier rate produces a 100-fold incidence rate. Therefore, regions such as New York City, where many Ashkenazi Jews reside, exhibit elevated occurrence of the disease. Other demographic groups that show similarly elevated rates of Tay–Sachs include a French Canadian population living near the Saint Lawrence River and a Cajun community in Louisiana (NHGRI, 2011). Once more, concentration of at-risk populations in a particular geographic area, such as New York, can create regional health disparities.

Asthma and Puerto Ricans

A disproportionate number of Hispanic children are affected by asthma, a disease that affects the lungs, often manifesting itself in repeated episodes of breathlessness, coughing, and wheezing (CDC, 2015). A 1996 study of East Coast families found that 25% of Hispanic families had at least one asthmatic child, while this was true for 18% of non-Hispanic Black families and only 10% of non-Hispanic White families (Beckett, Belanger, Gent, Holford, & Leaderer, 1996). Two-thirds of the Hispanic children with asthma are Puerto Rican: Puerto Rican children are more vulnerable to developing asthma than any other ethnic or racial group in the United States. As a result, regions with large Puerto Rican populations show elevated asthma rates. New York City, which contains the largest concentration of Puerto Ricans in the mainland United States, had an asthma mortality rate three times the national rate from 1981 to 1985 (Weiss & Wagener, 1990). Part of this geographic concentration may be attributed to the susceptibility of the Puerto Rican population to asthma.

Suicide and American Indians

Suicide rates between 2005 and 2009 were highest among American Indian/Alaskan Native males (25.96 suicides per 100,000) compared with other racial and ethnic groups and women (CDC, 2015). In 2013, Montana and Alaska had the highest state suicide rates of 23.9 and 23.3 per 100,000, respectively (Drapeau & McIntosh, 2015). Montana and Alaska have notable American Indian and Alaska Native populations, respectively. The highest rate of suicide in Montana is among American Indians (27.2 per 100,000; Rosston, 2012). The average annual rate of suicide among Alaska Native people during 2003–2008 was double the rate for Alaska non-Native peoples. These American Indian and Alaskan Native populations thus influence overall regional suicide rates.

Direct Physical Geographical Factors

The particulars of a region's geography certainly influence the patterns of immigration, from proximity to Ellis Island for various waves of European immigration and proximity to the

Pacific for waves of Asian immigration, to familiarity of terrain for Scandinavian immigrants, to proximity to steep trails for immigration of the young and athletic. Geography can also have an impact on health patterns more directly by altering the behavior and exposure of people once they are there. We will offer various examples of such effects.

Latitude

Seasonal affective disorder (SAD) is a pattern of depressive or bipolar episodes. Winter SAD has been found to correlate with latitude, occurring more often at higher latitudes in North America. However, the relationship between latitude and SAD appears much stronger in North America than in Europe, suggesting that social context and genetic factors may moderate this effect (Mersch, Middendorp, Bouhuys, Beersma, & van den Hoofdakker, 1999). Warm regions can have other health concerns: regions of the United States such as parts of Arizona and Texas have increasingly become home to Africanized honey bees, which results in an increased risk of people experiencing systemic anaphylaxis from bee stings, as these bees are more aggressive than non-Africanized bees. Weather itself can have acute implications for public health as well: heat waves increase heat-related mortality, and increased temperature has been found to correlate with increased all-cause mortality as well (Stafoggia et al., 2006).

Natural Disasters

Natural disasters can influence health immediately and dramatically, and they can also impact outcomes beyond the immediate effects of the disaster. Experiencing an earthquake in the first trimester is associated with shorter gestation in pregnant women. Increases in communicable diseases follow in the wake of natural disasters such as tsunamis or earthquakes. A review of posttraumatic stress disorder following natural (and other) disasters found substantial evidence for resulting posttraumatic stress disorder (Neria, Nandi, & Galea, 2008). Certain areas are more prone to natural disasters than others—earthquakes, of course, are not randomly distributed across the country, but follow known fault lines, especially those on the West Coast. As a result, natural disasters can contribute to differing health outcomes between regions, even long after their immediate effects.

Occupation

Regional differences in the distribution of natural resources produce occupational differences, which in turn can result in regional differences in health. Coal mining, concentrated in states such as West Virginia, causes lung diseases such as pneumoconiosis and chronic obstructive pulmonary disease. Community exposure to mining in coal-mining areas has likewise been found to relate to worse health outcomes such as higher rates of cardiopulmonary disease, chronic obstructive pulmonary disease, and lung cancer. As a result, regional differences in occupation can have a significant impact on community health.

Direct Cultural Geographical Factors

Regional differences in culture also comprise geographical determinants of health. Such differences may be produced originally by topography, natural resources, farming potential, and the like, but are no longer so directly linked to the physical environment. Coal mining, for example,

continues in West Virginia not so much because they have a culture that values fossil fuel extraction, or because early immigrants were predisposed to that activity—though both of those may also be true—but because that is where the coal is. However, for the sorts of cases discussed below, the persistence of regional differences seems instead to depend on continuing regional cultural differences. Examples of cultural regional variation include differences in diet, activities, stress, income, and community structure.

Diet

Dietary differences between regions can impact health outcomes. The southeastern United States leads the country in risk of stroke and hospitalizations for congestive heart failure. In fact, the disproportionate stroke mortality in this part of the country has led it to be dubbed the “stroke belt.” The high prevalence of stroke in this region is partially attributable to dietary differences. Judd et al. (2013) found a direct relationship between consuming a traditional Southern-style diet and increased risk of stroke. This diet of fried foods, high-fat foods, and sugar-sweetened drinks partially mediated racial differences in stroke risk in this region as well (Judd et al., 2013). Unhealthy diets, which are differentially distributed across the United States, thus contribute to health disparities between regions.

Pastimes

Gambling is bounded by legislation to specific regions of the United States. Both visitors to and residents of gaming communities such as Las Vegas and Atlantic City exhibit abnormally high suicide levels. Elevated suicide levels for not only visitors but residents as well suggest that the increased prevalence of suicide is not the simply result of gambling attracting suicidal persons. Further research has confirmed regional effects for low suicide areas as well: being a visitor to or resident of an area with low suicide risk was associated with a decreased suicide rate (Shrira & Christenfeld, 2010). Firearm use, another activity that is differentially legislated by region, was found to correlate with suicide level as well, suggesting that these differences may in part be explained by availability of means to commit suicide (Shrira & Christenfeld, 2010). Activities characteristic of particular communities thus contribute to health outcomes such as suicide levels.

Stress

Stressors and stress levels vary geographically as well. Stressors can have acute, immediate effects on health or chronic effects, which manifest only as a result of cumulative exposure to the stressor. Residents of New York City appear to experience both acute and chronic stressors. Residents of New York City who die there do so 55% more often than the general population does from ischemic heart disease. People just visiting the city show a 34% increase in the proportion of mortality due to ischemic heart disease. New York City residents who die outside the city show an ischemic heart disease proportion that is only 80% of that shown by residents in the city (Christenfeld, Glynn, Phillips, & Shrira, 1999). This pattern of results suggests that exposure to New York City increases heart attack risk, and the visitor findings indicate that some of that risk is chronic and some acute. That is, just being in the city at the moment confers some risk, and having been in the city for some time adds to that risk. Geographical differences in stressors thus contribute to disparities in health outcomes between regions.

Income

Income varies not just by family but also by neighborhood, state, and country. It correlates with outcomes such as infant mortality: infants born into poverty have a 1.5 times higher risk of neonatal and postneonatal mortality, likely due to differential access to healthcare (Gortmaker, 1979). Indicators of low socioeconomic status have also been associated with birthweight, gestational age, and preterm delivery. As a result, regions experience differential infant mortality outcomes that are associated with varying income levels: one analysis found that infant mortality in any particular census tract (a subdivision of a county) in Ohio correlated with the percentage of low-income families that lived in the tract (Stockwell, Goza, & Balistreri, 2005). Exposure to stress may mediate some of income's effects on infant mortality. Among low-income women, chronic psychosocial stress is associated with low birthweight. Likewise, depression during pregnancy, which is twice as common among poor urban women than it is in middle-class women, is associated with preterm delivery and low birthweight.

Longevity, or life expectancy, also correlates with income. Poor Americans live an average of 5 years less than the most affluent Americans. Some argue that income inequality in its own right contributes to mortality as well, separate from the effects of absolute level of wealth (Kennedy, Kawachi, & Prothrow-Stith, 1996). States with less equal wealth distributions, such as Louisiana and Mississippi, were found to have greater all-cause mortality than states such as Vermont and New Hampshire, and wealth distribution across all 50 states correlated with all-cause mortality (Kennedy et al., 1996). Although findings on income inequality relating to health have been mixed, there seems to be a modest effect of inequality. Income, and perhaps even level of income disparity within a community, can contribute to regional differences in health outcomes.

Community Structure

Whether people live in an urban or rural setting can also influence longevity. Premature mortality, as measured in years of life lost before reaching 75, was found to be greatest in rural counties, particularly in the Southeast and Southwest (Mansfield, Wilson, Kobrinski, & Mitchell, 1999). Metropolitan and urban counties, by comparison, had lower premature mortality when using this metric (Mansfield et al., 1999). The gap between urban and rural life expectancies has widened over the past 40 years, largely due to increases in life expectancies in urban areas (Singh & Siahpush, 2014). Community structure itself can thus influence health outcomes.

Public Policy and the Geographical Influences of Health

Exploring the geography of health can have a variety of public health implications. Just as the cause of cholera was revealed by an analysis of the distribution of the disease along the Thames, regional differences in illness can provide information about its etiology. Furthermore, understanding the link between geography and health can help in determining where resources are needed or will be needed in the future. Beyond understanding or predicting disease, it can lead to interventions that do prevent illness. For example, the creation of walkable green spaces increases the longevity of urban seniors. Physically active lifestyles are associated with a variety of cardiorespiratory benefits. Policies intended to encourage active transport such as biking

and walking lead to healthier populations: the creation of bike lanes and separating cycling from other traffic are associated with increased cycling.

Public health can be improved not only by altering the physical environment to promote exercise but also with alterations to culturally based food habits. Areas that lack access to supermarkets and healthy food, termed food deserts, result in poor diets and the health problems that relate to poor diet such as diabetes and coronary heart disease. These areas are generally low income and either urban or remote. Retail development by supermarket chains working in conjunction with the city council in one such British neighborhood improved diet-related outcomes, albeit modestly, suggesting that policy that develops supermarkets in such neighborhoods may be an effective intervention (Wrigley, Warm, & Margetts, 2003).

Policies aimed at regulating the quality of the environment also have an effect on public health. The establishment of air quality-control policies in the Los Angeles basin, an area with a problematic combination of high volume of automobiles, large seaport, and natural landscape that contains the resulting air pollution, caused long-term reductions in airborne pollution, which improved lung function growth in children living in this region (Gauderman et al., 2015). These policies included emission control as well as fuel reformulations (Gauderman et al., 2015).

Understanding both how regions differ in demography and how they have a direct effect on the health of their inhabitants serves a useful function in informing public health policy. When deciding how health services should be distributed regionally, demographic differences may be enough to inform effective policy. However, the identification of controllable factors that are regionally caused, rather than their being the result of demographic distributions, can lead to a more effective use of public health resources in addressing certain public health concerns more generally. While it is useful to dispatch more cardiovascular health services to the Southeast to combat the high stroke rate, identifying the dietary cultural contributions to stroke risk can spur the development of dietary interventions to reduce the disproportionate need for cardiovascular health services over time. This is not possible for uncontrollable regional factors; we cannot diminish or more equally distribute the fault lines across the United States.

Geography is not necessarily static—there is an emergent field of study on the potential impacts of climate change on regional differences in health, as temperature, agriculture, water supplies, pollution levels, and the like are redistributed geographically. For example, the distribution of vector-borne diseases, such as malaria, could change in response to rising temperatures and affect new populations. As climate change progresses, there will be an increased need for understanding, predicting, and responding to its shifting effects on population health.

Simultaneously, the expanding understanding of the genetic factors involved in various diseases will also increase the potential for the discovery of new geographic disparities in specific diseases. Identifying new vulnerabilities among demographic groups will inform how to best distribute services relating to the vulnerability across the country and where environmental interventions may be most effective.

We are not entirely dependent on medical breakthroughs to improve a population's health: while researchers continue to work toward medical treatments for obesity, for example, we can begin to address geography-level causes of the condition, such as by restructuring the built environment to decrease commute times. We will certainly continue to have new and effective medical treatments for those suffering from poor health, but we can also limit the number of people who need to resort to treatment by better understanding how geography contributes to their condition in the first place.

Author Biographies

Arseny A. Ryazanov is a doctoral student in the psychology program at the University of California, San Diego. He studies multiple aspects of social psychology—how locus of control relates to treatment of others, what encourages prosocial behavior, and whether we can predict incremental change in group preferences over time.

Kirstie K. Russell, a masters of public health candidate at the Dalla Lana School of Public Health at the University of Toronto, is interested in the social and ecological determinants of health.

Nicholas J.S. Christenfeld, a psychology professor at the University of California, San Diego, has investigated a broad array of only partially overlapping topics, including the role of stress in health and interventions that can reduce the severity and duration of its impact, and psychological factors in mortality, from imitative physician-assisted suicide to the cardiac perils of living in New York City.

References

- Beckett, W. S., Belanger, K., Gent, J. F., Holford, T. R., & Leaderer, B. P. (1996). Asthma among Puerto Rican Hispanics: A multi-ethnic comparison study of risk factors. *American Journal of Respiratory and Critical Care Medicine*, 154(4), 894–899. doi:10.1164/ajrccm.154.4.8887582
- Centers for Disease Control and Prevention (CDC). (2015). Retrieved from <http://www.cdc.gov>.
- Christenfeld, N., Glynn, L. M., Phillips, D. P., & Shira, I. (1999). Exposure to New York City as a risk factor for heart attack mortality. *Psychosomatic Medicine*, 61(6), 740. doi:10.1097/00006842-199911000-00003
- Drapeau, C. W., McIntosh, J. L., & (for the American Association of Suicidology). (2015). *U.S.A. suicide 2013: Official final data*. Washington, DC: American Association of Suicidology. Retrieved from <http://www.suicidology.org>
- Erickson, W., Lee, C., & von Schrader, S. (2014a). *2012 Disability status report: Florida*. Ithaca, NY: Cornell University Employment and Disability Institute(EDI).
- Erickson, W., Lee, C., & von Schrader, S. (2014b). *2012 disability status report: Utah*. Ithaca, NY: Cornell University Employment and Disability Institute(EDI).
- Gauderman, W. J., Urman, R., Avol, E., Berhane, K., McConnell, R., Rappaport, E., ... Gilliland, F. (2015). Association of improved air quality with lung development in children. *New England Journal of Medicine*, 372(10), 905–913. doi:10.1056/NEJMoa1414123
- Gortmaker, S. L. (1979). Poverty and infant mortality in the United States. *American Sociological Review*, 280–297.
- Judd, S. E., Gutiérrez, O. M., Newby, P. K., Howard, G., Howard, V. J., Locher, J. L., ... Shikany, J. M. (2013). Dietary patterns are associated with incident stroke and contribute to excess risk of stroke in black Americans. *Stroke*, 44(12), 3305–3311. doi:10.1161/STROKEAHA.113.002636
- Kaiser Family Foundation. (2013). Population distribution by age. Retrieved from <http://kff.org/other/state-indicator/distribution-by-age/>.
- Kennedy, B. P., Kawachi, I., & Prothrow-Stith, D. (1996). Income distribution and mortality: Cross sectional ecological study of the Robin Hood index in the United States. *British Medical Journal*, 312(7037), 1004–1007. doi:10.1136/bmj.312.7037.1004
- Lin, S. Y., Chang, E. T., & So, S. K. (2007). Why we should routinely screen Asian American adults for hepatitis B: A cross-sectional study of Asians in California. *Hepatology*, 46(4), 1034–1040. doi:10.1002/hep.21784
- Mansfield, C. J., Wilson, J. L., Kobrinski, E. J., & Mitchell, J. (1999). Premature mortality in the United States: The roles of geographic area, socioeconomic status, household type, and availability of medical care. *American Journal of Public Health*, 89(6), 893–898. doi:10.2105/AJPH.89.6.893

- Mersch, P. P. A., Middendorp, H. M., Bouhuys, A. L., Beersma, D. G., & van den Hoofdakker, R. H. (1999). Seasonal affective disorder and latitude: A review of the literature. *Journal of Affective Disorders*, 53(1), 35–48. doi:10.1016/S0165-0327(98)00097-4
- National Human Genome Research Institute (NHGRI). (2011). Learning about Tay-Sachs disease. Retrieved from <https://www.genome.gov/10001220>.
- Neria, Y., Nandi, A., & Galea, S. (2008). Post-traumatic stress disorder following disasters: A systematic review. *Psychological Medicine*, 38(04), 467–480.
- Rosston, K. (2012, October 1). Suicide in Montana facts, figures, and facts, figures, and formulas for prevention formulas for prevention. Retrieved from <https://dphhs.mt.gov/Portals/85/suicideprevention/SuicideinMontana.pdf>.
- Shrira, I., & Christenfeld, N. (2010). Disentangling the person and the place as explanations for regional differences in suicide. *Suicide and Life-threatening Behavior*, 40(3), 287–297. doi:10.1521/suli.2010.40.3.287
- Singh, G. K., & Siahpush, M. (2014). Widening rural–urban disparities in life expectancy, US, 1969–2009. *American Journal of Preventive Medicine*, 46(2), e19–e29. doi:10.1016/j.amepre.2013.10.017
- Stafoggia, M., Forastiere, F., Agostini, D., Biggeri, A., Bisanti, L., Cadum, E., ... Perucci, C. A. (2006). Vulnerability to heat-related mortality: A multicity, population-based, case-crossover analysis. *Epidemiology*, 17(3), 315–323. doi:10.1097/01.ede.0000208477.36665.34
- Stockwell, E. G., Goza, F. W., & Balistreri, K. S. (2005). Infant mortality and socioeconomic status: New bottle, same old wine. *Population Research and Policy Review*, 24(4), 387–399. doi:10.1007/s11113-005-0088-2
- Weiss, K. B., & Wagener, D. K. (1990). Geographic variations in US asthma mortality: Small-area analyses of excess mortality, 1981–1985. *American Journal of Epidemiology*, 132(suppl), 107–115.
- Wrigley, N., Warm, D., & Margetts, B. (2003). Deprivation, diet, and food-retail access: Findings from the Leeds food deserts' study. *Environment and Planning A*, 35(1), 151–188. doi:10.1068/a35150

Suggested Reading

- Boyle, P., Curtis, S., Graham, E., & Moore, E. (2004). *The geography of health inequalities in the developed world: Views from Britain and North America*. Burlington, VT: Ashgate Publishing Ltd. doi:10.1111/j.1467-9272.2005.00504_5.x
- Brown, T., McLafferty, S., & Moon, G. (2009). *A companion to health and medical geography*. London, UK: Wiley. doi:10.1002/9781444314762.ch6
- Gatrell, A. C., & Elliott, S. J. (2014). *Geographies of health: An introduction*. Hoboken, NJ: Wiley.
- Meade, M. S., & Emch, M. (2010). *Medical geography*. New York, NY: Guilford Press. doi:10.1002/9781118410868.wbehibs204

Habits

Martin S. Hagger^{1,2,3,4} and Amanda L. Rebar⁴

¹*Health Psychology and Behavioral Medicine Research Group, School of Psychology and Speech Pathology, Faculty of Health Sciences, Curtin University, Perth, WA, Australia*

²*Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland*

³*School of Applied Psychology and Menzies Health Institute Queensland, Behavioural Bases for Health, Griffith University, Brisbane, QLD, Australia*

⁴*Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia*

“Healthy habits” are a frequent topic of conversation in everyday life. People want to “get into the habit” of exercise or healthy eating or “break bad habits” like smoking or drinking too much alcohol. These frequent references to habit tacitly acknowledge that good habits are difficult to form and bad habits difficult to stop. Psychological research indicates that habits have a strong “automatic” or “implicit” component. That is, individuals tend to perform habits with relatively little conscious thought or effort. The propensity for actions to be controlled automatically is an extremely adaptive human function, as it means that mundane actions can be carried out with little cognitive effort, which “frees up” cognitive “space” for processing higher-order goals and actions. Health behaviors that are habitual are, therefore, regulated and enacted in an efficient and effective manner. However, the same system can automate behaviors that are detrimental to health, making them difficult to change. Health psychologists are interested in studying habit to understand the processes that lead to habit formation and perhaps develop means to change habits by promoting healthy habits and breaking unhealthy ones.

Formally, habit is defined as “a process by which a stimulus automatically generates an impulse towards action, based on learned stimulus-response associations” (Gardner, 2015, p. 4). In contradiction to the colloquial use of the term, habit is not a behavior, but rather a psychological or cognitive determinant of behavior. Habit formation is the process by which regulation of health behavior shifts from being under conscious, deliberative control to being regulated largely by nonconscious, automatic processes (Gardner, 2015; Sheeran, Gollwitzer, & Bargh, 2013; Wood & Neal, 2009). When a person frequently and consistently enacts a health behavior in the context of a behaviorally relevant cue or—so as to form a strong mental association

between the cue and behavior—a habit is formed. Research has demonstrated that the strength of people’s habits is a strong influence on their health-related behaviors and this effect is independent of conscious regulation such as goal setting, planning, or intentions (Conroy, Maher, Elavsky, Hyde, & Doerksen, 2008; Gardner, de Bruijn, & Lally, 2011). Exciting advancements in the study of health-related habits demonstrate strategies that people may use to break unhealthy habits and the potential for targeting habit formation as a strategy for promoting maintainable healthy behaviors (e.g., Lally & Gardner, 2013; Webb, Sheeran, & Luszczynska, 2009).

Theory, Habits, and Health Behavior

Consideration for the role of habits in the study of health behaviors has been a relatively recent advancement, and as a result, most studies have incorporated a measure of habit¹ as a separate correlate of health behavior alongside psychological factors from conventional social cognitive theories of behavior change (Biddle, Hagger, Chatzisarantis, & Lippke, 2007). This research has been further advanced through the application of dual-process theories, which contend that behavior is a function of both nonconscious (e.g., habit) and conscious processes (e.g., goals, deliberation, attitudes, and values; Strack & Deutsch, 2004 #1742; Hagger, 2016 #8248). For example, Rothman, Sheeran, and Wood (2009) present a model for understanding which conscious and nonconscious processes are important for adoption versus maintenance of health-related behaviors and outline how habits likely play a key role in the maintenance of health-related behaviors. Similarly, Rhodes and de Bruijn propose habit as a key factor involved in the control of actions in health contexts (i.e., translating intentions into action; Rhodes & de Bruijn, 2013).

The common theme of dual-process models is that health behaviors can be predominantly controlled by either reflective consideration of the action (e.g., weighing up the advantages, disadvantages, consequences, and barriers with respect to the behavior) or impulsive processes that lead to automatic behavioral enactment. The influences from both types of regulatory systems are supported by evidence from neuroscience that shows that activity in the fronto-parietal and cingulo-opercular networks is associated with deliberative actions, whereas activity in subcortical areas of the mesolimbic reward system such as the nucleus accumbens is associated with habitual behaviors (Rebar, Loftus, & Hagger, 2015).

Given that habitual behaviors are rapid, efficient, and free of conscious control and occur beyond an individual’s awareness, they tend to be the “dominant” response when individuals are presented with cues that initiate their action (Gardner, 2015; Verplanken & Aarts, 1999). For an individual to change or break a habit, therefore, they must consciously monitor their environment for the habit-triggering cue and, on its presentation, actively inhibit the automatic behavioral response. Without sufficient deliberative control over cues and associated automatic responses, referred to as self-regulation, the dominant response is likely to pervade in situations where the cue is presented. Even when individuals identify health goals and form plans to achieve these goals, habits may compete with these, and, given that the habitual pathways to action tend to predominate, they may lead to failure to carry out the good intentions.

Research on Habits and Health Behaviors

Research has demonstrated that habit is associated with a number of health-related behaviors (e.g., Gardner et al., 2011; Hyde, Elavsky, Doerksen, & Conroy, 2012; Webb et al., 2009).

Research has also examined the factors that determine whether health behavior is likely to be under habitual control or controlled by more deliberative, conscious processes. For example, an increasing body of evidence is demonstrating that intentions to engage in physical activity or eating healthily are better predictors of behavior for people if habits of these behaviors are weaker (Gardner et al., 2011; Rhodes & de Bruijn, 2013), and more recent evidence suggests this could be a time-dependent process in that people act on their habits unless they make particularly strong intentions to engage in a counter-habitual behavior on a certain day (Rebar, Elavsky, Maher, Doerksen, & Conroy, 2014). Furthermore, research has shown that individuals find it difficult to accurately recall habitual actions because such actions do not depend on awareness or conscious control (Hyde et al., 2012).

Research is also beginning to provide insight into the process by which habits form. For example, research has shown that habit formation requires more than mere repetition of the behavior (Hagger, Rebar, Mullan, Lipp, & Chatzisarantis, 2015; Lally & Gardner, 2013) and that the habit formation process is different for each person, but typically occurs quickly at first and gradually slows when a person nearly reaches full habit development (Lally & Gardner, 2013). Factors that have been shown to determine the formation of habits are planning and behavior enactment, strong cue–behavior implicit associations, or increasing the intrinsic value of the behavior by providing incentives or rewards (Lally & Gardner, 2013).

In addition, research is starting to emerge on how the formation of habits can be targeted within interventions aimed at promoting healthy habits (Gardner, Lally, & Wardle, 2012; Lally & Gardner, 2013). Habits form through repetition of behavior in a specific context, so the habitual response becomes strongly associated with a triggering cue, making the habitual response the strong default and alternative behavioral choices less salient (Wood & Neal, 2009). Simple intervention strategies to promote frequent and consistent performance of the desired habitual behavior in the context of an accessible cue (e.g., in the same manner, at the same time of day or part of a daily routine, in the same place) through a list of tips, messages via emails and SMS, or face-to-face training have shown not only to enhance health behavior but also to make the behavior more habitual. Additionally, incorporating “boosters” following intervention completion focused on action planning, self-efficacy, and satisfaction behavioral outcomes may serve to maintain intervention effects on habit.

Promotion of a healthy lifestyle often requires a combination of forming stronger healthy behavior habits (e.g., healthy eating, physical activity, medication adherence) and breaking habits for unhealthy behaviors (e.g., smoking, snack eating, sedentary behavior). Research has demonstrated that effective breaking of “bad” habits requires goal setting, cue monitoring, and effortful self-regulation (Lally & Gardner, 2013). Such efforts can be quite taxing, and engaging in effortful control over habits may be counterproductive, as it may lead to cognitive fatigue and reduced capacity for self-control, leaving people susceptible to temptations that cue up unwanted habitual responses (Hagger & Chatzisarantis, 2014). An option for reducing habitual unhealthy behaviors may be to avoid or minimize accessibility or presence of triggering cues by, for example, moving locations or to weaken the mental cue–response association through training techniques such as evaluative conditioning (Allom, Mullan, & Hagger, 2016).

Future Directions for Research on Habits and Health-Related Behavior

A number of priorities for future research on habits in the context of health-related behavior have been identified. Researchers are beginning to differentiate between habitual instigation (i.e., the initiation of a behavior) and execution (i.e., the movement through the sequence of

the behaviors) (Gardner, Phillips, & Judah, 2016). A proposed model is that in early stages of taking up a new behavior, behavioral initiation is predominantly controlled by more deliberative, reflective processes in which an individual weighs up the benefits and detriments of the behavior in a given context and makes decisions on whether or not to act accordingly. This process becomes more “automated” or habitual over time with repeated decisions to engage in behavior raising the possibility of links being made between the behavior and the decision-making process and external or internal cues. However, while behavioral initiation may become more automated, more complex processes involved in executing the behavior may not become automatic as they require elaborate planning and deliberation. Such distinctions may provide a comprehensive framework for promoting complex health-related behaviors that involve more than a single act (e.g., physical activity, eating, sedentary behavior).

Another avenue of research, that is, essential for advancing theory of health-related habit is to understand how the influence of habit compares to and interacts with that of other nonconscious regulatory processes such as automatic evaluations (e.g., implicit attitudes) or primed motivation (Conroy et al., 2008; Hagger, 2016; Sheeran et al., 2013). Most research on health-related habit has been studied alongside consciously controlled psychological constructs, but this largely downplays and likely simplifies the impact that other nonconscious processes have on health behaviors. Researchers should consider merging what have, to this point, been largely separate avenues of research on habit and these other nonconscious regulatory processes that are not reliant on learning.

Finally, the role of implicitly presented cues in developing habitual responses should be explored. Research examining the potential for environmental contingencies, structural changes, and means to prompt behaviors (e.g., posters and advertisements) to promote healthy behavior, often beyond an individual’s conscious awareness, has shown promise (Hagger, 2016). However, the research on the mechanisms and processes by which these contingencies affect behavior and assist in the development of habits is relatively sparse. Future research should explore the longevity of such prompts in promoting lasting behavior change and whether these develop into habits.

Conclusion

A health behavior is considered habitual if it is predominantly determined by “automatic” or “nonconscious” processes. Habitual behaviors tend to be developed over time through repetition and reward or reinforcement by external or internal contingencies or reinforcing agents. Habits may be represented schematically in memory and activated by the presentation of cues or prompts in the environment with which enactment of the habituated behavior is associated. As links between prompts and habitual behaviors tend to be very strong, changing habitual behaviors that are detrimental to health is challenging. However, the strong links also present an opportunity for interventionists as they provide a means to develop healthy habits and lasting behavior change. Research on habits has provided insight into the processes by which habits impact health behavior and an evidence base on which to develop interventions to break or change habits. Breaking bad habits requires effortful, goal-directed behavior, and self-regulatory skills such as goal setting and cue monitoring have been shown to be effective strategies to break health behaviors. Future research should seek to examine the role of priming interventions, implicit constructs, and the distinction between initiation and execution of health behavior to provide further insight into the processes that lead to health behavior.

Author Biographies

Martin S. Hagger is John Curtin Distinguished Professor in the School of Psychology and Speech Pathology at Curtin University, Australia. His research applies social cognitive and motivational theories to understand and to intervene and change diverse health behaviors such as physical activity, healthy eating, smoking cessation, alcohol reduction, and medication adherence. He is also at the forefront of theory development and has contributed to the advancement of social psychological theory including theory integration and self-control.

Amanda L. Rebar is senior lecturer in the Centre for Physical Activity Studies in the School of Health, Medical and Applied Sciences at Central Queensland University, Australia. Her research interests include physical activity motivation—specifically automatic regulation of physical activity (e.g., habits, automatic evaluations) and the mental health benefits of physical activity. She is also interested in structural equation modeling, hierarchical linear modeling, and the open-source data analysis program, R.

Note

- 1 It is important to note the use of terminology in this context. Habits as a plural term, or preceded by the definite article (“a habit” or “the habit”), refer to the concept of automated, impulse-driven responses. Habit in singular usually refers to a measure or construct of habit in an analysis or theory.

References

- Allom, V., Mullan, B. A., & Hagger, M. S. (2016). Does inhibitory control training improve health behaviour? A meta-analysis. *Health Psychology Review*, 10, 168–186. doi:10.1080/17437199.2015.1051078
- Biddle, S. J. H., Hagger, M. S., Chatzisarantis, N. L. D., & Lippke, S. (2007). Theoretical frameworks in exercise psychology. In G. Tenenbaum & R. C. Eklund (Eds.), *Handbook of sport psychology* (3rd ed., pp. 537–559). New York, NY: Wiley. doi:10.1002/9781118270011.ch24
- Conroy, D. E., Maher, J. P., Elavsky, S., Hyde, A. L., & Doerksen, S. E. (2008). Sedentary behavior as a daily process regulated by habits and intentions. *Health Psychology*, 32, 1149–1157. doi:10.1037/a0031629
- Gardner, B. (2015). A review and analysis of the use of ‘habit’ in understanding, predicting and influencing health-related behaviour. *Health Psychology Review*, 9, 277–295. doi:10.1080/17437199.2013.876238
- Gardner, B., de Bruijn, G.-J., & Lally, P. (2011). A systematic review and meta-analysis of applications of the self-report habit index to nutrition and physical activity behaviours. *Annals of Behavioral Medicine*, 42, 174–187. doi:10.1007/s12160-011-9282-0
- Gardner, B., Lally, P., & Wardle, J. (2012). Making health habitual: The psychology of ‘habit-formation’ and general practice. *British Journal of General Practice*, 62, 664. Retrieved from <http://bjgp.org/content/62/605/664.abstract>
- Gardner, B., Phillips, L. A., & Judah, G. (2016). Habitual instigation and habitual execution: Definition, measurement, and effects on behaviour frequency. *British Journal of Health Psychology*, 21, 613–630. doi:10.1111/bjhp.12189
- Hagger, M. S. (2016). Non-conscious processes and dual-process theories in health psychology. *Health Psychology Review*, 10, 375–380. doi:10.1080/17437199.2016.1244647
- Hagger, M. S., & Chatzisarantis, N. L. D. (2014). It is premature to regard the ego-depletion effect as ‘too incredible’. *Frontiers in Psychology*, 5, 298. doi:10.3389/fpsyg.2014.00298

- Hagger, M. S., Rebar, A. L., Mullan, B. A., Lipp, O. V., & Chatzisarantis, N. L. D. (2015). The subjective experience of habit captured by self-report indexes may lead to inaccuracies in the measurement of habitual action. *Health Psychology Review*, 9, 296–302. doi:10.1080/17437199.2014.959728
- Hyde, A. L., Elavsky, S., Doerksen, S. E., & Conroy, D. E. (2012). Habit strength moderates the strength of within-person relations between weekly self-reported and objectively-assessed physical activity. *Psychology of Sport and Exercise*, 13, 558–561. doi:10.1016/j.psychsport.2012.03.003
- Lally, P., & Gardner, B. (2013). Promoting habit formation. *Health Psychology Review*, 7, S137–S158. doi:10.1080/17437199.2011.603640
- Rebar, A. L., Elavsky, S., Maher, J. P., Doerksen, S. E., & Conroy, D. E. (2014). Habits predict physical activity on days when intentions are weak. *Journal of Sport and Exercise Psychology*, 36, 157–165. doi:10.1123/jsep.2013-0173
- Rebar, A. L., Loftus, A. M., & Hagger, M. S. (2015). Cognitive control and the non-conscious regulation of health behavior. *Frontiers in Human Neuroscience*, 9, 122. doi:10.3389/fnhum.2015.00122
- Rhodes, R. E., & de Bruijn, G. J. (2013). What predicts intention-behavior discordance? A review of the action control framework. *Exercise and Sport Sciences Reviews*, 41, 201–207. doi:10.1097/JES.0b013e3182a4e6ed
- Rothman, A. J., Sheeran, P., & Wood, W. (2009). Reflective and automatic processes in the initiation and maintenance of dietary change. *Annals of Behavioral Medicine*, 38, 4–17. doi:10.1007/s12160-009-9118-3
- Sheeran, P., Gollwitzer, P. M., & Bargh, J. A. (2013). Nonconscious processes and health. *Health Psychology*, 32, 460–473. doi:10.1037/a0029203
- Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and Social Psychology Review*, 8, 220–247.
- Verplanken, B., & Aarts, H. (1999). Habit, attitude, and planned behaviour: Is habit an empty construct or an interesting case of goal-directed automaticity? *European Review of Social Psychology*, 10, 101–134. doi:10.1080/14792779943000035
- Webb, T. L., Sheeran, P., & Luszczynska, A. (2009). Planning to break unwanted habits: Habit strength moderates implementation intention effects on behaviour change. *British Journal of Social Psychology*, 48, 507–523. doi:10.1348/014466608X370591
- Wood, W., & Neal, D. T. (2009). The habitual consumer. *Journal of Consumer Psychology*, 19, 579–592. doi:10.1016/j.jcps.2009.08.003

Suggested Reading

- Gardner, B. (2015). A review and analysis of the use of ‘habit’ in understanding, predicting and influencing health-related behaviour. *Health Psychology Review*, 9, 277–295. doi:10.1080/17437199.2013.876238
- Ouellette, J. A., & Wood, W. (1998). Habit and intention in everyday life: The multiple processes by which past behavior predicts future behavior. *Psychological Bulletin*, 124, 54–74. doi:10.1037//0033-2909.124.1.54
- Verplanken, B., & Aarts, H. (1999). Habit, attitude, and planned behaviour: Is habit an empty construct or an interesting case of goal-directed automaticity? *European Review of Social Psychology*, 10, 101–134. doi:10.1080/14792779943000035
- Verplanken, B., & Orbell, S. (2003). Reflections on past behavior: A self-report index of habit strength. *Journal of Applied Social Psychology*, 33, 1313–1330. doi:10.1111/j.1559-1816.2003.tb01951.x
- Webb, T. L., Sheeran, P., & Luszczynska, A. (2009). Planning to break unwanted habits: Habit strength moderates implementation intention effects on behaviour change. *British Journal of Social Psychology*, 48, 507–523. doi:10.1348/014466608X370591

Health Behavior Change

Paul T. Fuglestad, Jody S. Nicholson,
and Lauren James

Department of Psychology, University of North Florida, Jacksonville, FL, USA

Most modern diseases and causes of death have substantial contributing factors that are behavioral. The Center for Disease Control and Prevention estimates that premature death and unnecessary healthcare costs can be greatly ameliorated by enacting behavioral changes. For decades, researchers across disciplines in the social and health sciences have sought to create theories of health behavior that can be used to encourage healthy change and inform intervention design and implementation. Although these theories and approaches to intervention share underlying similarities, they vary widely in terms of their behavioral foci, focal constructs, and processes and stages of change. For example, Michie, West, Campbell, Brown, and Gainforth (2014), in attempting to catalog all behavior change theories relevant to interventions, identified over 80 theories with more than 1,000 constructs. Furthermore, health behaviors are numerous and varied, including diet, physical activity, alcohol use, tobacco use, vaccinations, medication adherence, sexual behavior, toxin exposure, and screenings. Fortunately, many theories are applied to numerous health behavior domains, and there is substantial overlap in mechanisms of change among the most widely used health behavior theories (Sheeran et al., 2016; Sheeran, Klein, & Rothman, 2017). This entry begins by reviewing numerous influential theories and approaches that have been used to understand and influence health behavior change and concludes with a discussion of challenges to health behavior change and promising new directions.

Although health behavior theories can be categorized in many different ways (e.g., Do they focus on intentional/explicit processes or implicit/habitual processes? Do they emphasize cognitions regarding a threat or cognitions regarding a specific behavior?), a useful distinction can be made between continuum and stage-based models (Rothman, Baldwin, Hertel, & Fuglestad, 2011). Continuum models explain change as a steady process, whereas stage-based theories assume change is nonlinear and consists of steps.

Continuum models specify a set of target variables that are thought to always predict behavior regardless of individual differences or situations, and a person's behavior is thought to be a function of his/her standing on these specified variables. For example, a continuum model such as the *health belief model* might attribute changes in smoking cessation to incremental changes in the perception of susceptibility to a health threat (e.g., cancer) and barriers or benefits to quitting (e.g., quitting will reduce the likelihood of lung cancer). On the other hand, stage theories propose that people move sequentially through various stages in changing their health behavior and different variables are thought to be important for certain stages, but not others. For example, programming based on a stage-based model like the *transtheoretical model* would focus on different change variables for someone who is not planning to quit (*precontemplation* stage) and someone who is actively quitting (*action* stage). Using this model, a program might focus on *consciousness raising* to increase knowledge for someone in the *precontemplation* stage while using *stimulus control* to break association cues that illicit smoking behavior for those in the *action* stage of quitting.

Continuum Models

Health Belief Model and Protection Motivation Theory

The first major health behavior theory was the health belief model developed by Irvin Rosenstock and colleagues in the 1950s. This model attempts to specify when people will take action against some health threat, such as getting a vaccination to prevent an illness, using a condom to prevent STDs, or wearing sunscreen to prevent skin cancer. It specifies that people will be more likely to take action if they perceive that they are vulnerable to a threat (i.e., *perceived susceptibility*), that the threat would have negative consequences (i.e., *perceived severity*), and that the benefits of action outweigh the costs or barriers.

Similar to the health belief model, the protection motivation theory, developed by Ronald Rogers, proposes that taking action to change a health behavior is dependent on constructs such as perceived susceptibility, severity, and whether the recommended behavior actually prevents the threat. It was largely developed in the context of using fear appeals to encourage people to engage in protective action against health threats. Building on stress and coping research, the protection motivation theory focuses on the cognitive processes of *threat appraisal* and *coping appraisal* that feed into one's protection motivation. One's emotional response to a potential threat (i.e., *fear*) was added to the threat appraisal process to go along with perceptions of susceptibility and severity. Similarly, one's confidence to enact a particular behavior (i.e., *self-efficacy*) was added to the coping appraisal process to go along with perceptions of benefits and barriers. In a meta-analysis of the protection motivation theory, Milne, Sheeran, and Orbell (2000) found that variables related to threat appraisal (e.g., susceptibility, severity, fear) and coping appraisal (e.g., self-efficacy, response benefits and costs) were predictive of intentions and behavior. The impacts of threat and coping appraisals on health-related intentions and behavior were small to medium in effect size and generally less predictive of subsequent rather than concurrent behaviors. Small to medium effect sizes were also evident in a meta-analysis of the influence of experimentally manipulated perceptions of how likely one was to get an illness (i.e., perceived susceptibility) and how bad it would be to get it (i.e., perceived severity) on people's intentions and behaviors (Sheeran, Harris, & Epton, 2014).

Theory of Reasoned Action/Planned Behavior and the Prototype Willingness Model

Another widely used set of health behavior theories are the theory of reasoned action and the theory of planned behavior developed by Martin Fishbein and Icek Ajzen. In the theory of reasoned action, behavioral *intention* (e.g., one's intention to eat more fruits and vegetables) is posited to be the most proximal predictor of health behavior and is predicted by an individual's *attitudes* and *subjective norms*. Attitudes about the behavior are based on one's expectation of behavioral outcomes and the value of those outcomes (e.g., eating more fruits and vegetables will have positive effects on health). Subjective norms regarding the behavior are based on one's perceptions of norms regarding a behavior and whether one is motivated to comply with those norms (e.g., important others think it is a good idea to eat more fruits and vegetables). In recognition that some behaviors are less volitional or controllable, the theory of planned behavior adds a construct very similar to self-efficacy, *perceived behavioral control*, which is based on how easy/hard it would be to engage in the behavior (e.g. eating more fruits and vegetables is something that can easily be done).

Sheeran et al. (2016) summarized 18 meta-analyses that examined the associations of attitudes, norms, and self-efficacy with health-related intentions and behaviors (e.g. condom use, physical activity, smoking cessation, alcohol use, and screening behaviors). Averaged across the meta-analyses, these constructs were all moderately to strongly associated with behavioral intentions and actual behavior. Sheeran et al. (2016) also conducted a meta-analysis to examine the experimental impact of these constructs. To be included in the meta-analysis, an intervention had to experimentally manipulate attitudes, norms, or self-efficacy related to a health behavior. Importantly, all of these constructs were related to intentions and behavior. Although the effect sizes were somewhat smaller than those reported in meta-analyses of correlational studies discussed above, the effect sizes were generally in the medium range, suggesting that these common constructs are indeed important for promoting health behavior change.

Building on the theory of planned behavior, the prototype willingness model was developed by Meg Gerrard and Frederick Gibbons to more fully understand non-intentional but volitional behaviors (e.g., risky sexual or substance use behaviors). It adds the variable of *social prototypes* that measures one's perception of the prototypical person who engages in a behavior (e.g., someone who engages in unsafe sexual practices), including one's similarity to the prototype and/or one's overall evaluation of this prototypical person. Social prototypes are thought to primarily influence another additional construct—*behavioral willingness*—which is one's willingness to engage in a risky/unhealthy behavior under certain circumstances (e.g., an attractive partner wants to have sex without a condom). Evidence suggests that these constructs are important for predicting behavior when one is engaging in less thoughtful, more heuristic-based cognition (Gerrard, Gibbons, Houlihan, Stock, & Pomeroy, 2008).

Social Cognitive Theory

Social cognitive theory, developed by Albert Bandura, is a continuum model that explains how humans develop, learn, and maintain certain behaviors and is often described in relationship to a model of self-efficacy. Bandura defined self-efficacy as one's belief in one's ability to succeed in specific situations and viewed people as more self-regulatory than reactive to their environment. For example, how people perceive their situations can inform their decisions to behave in certain ways that can lead to an eventual change in their environments. Therefore, people's behavior can be predicted by knowing the beliefs they have about their own capabilities

and environments. People's self-efficacy beliefs are thought to be dependent on four main sources of influence: (a) coaching and feedback (i.e., *social persuasion*), (b) observational learning and modeling (i.e., *vicarious experience*), (c) efficaciousness when agitated or depressed (i.e., *emotional/physiological states*), and (d) prior success with a task (i.e., *personal mastery experiences*). Beyond self-efficacy, behavior is also posited to depend on one's perceptions of environmental constraints that facilitate or prevent behavior (i.e., *barriers to change*), as well as one's attitude toward the behavior and beliefs in the likelihood that good or bad things will occur as a result of performing the behavior (i.e., *outcome expectations and evaluations*). As noted above, meta-analysis suggests that self-efficacy and attitudes toward the behavior are causally linked to health behavior change (Sheeran et al., 2016).

Stage Models

Transtheoretical Model

In contrast to continuum models of health behavior change, the transtheoretical model, developed by James Prochaska and Carlo DiClemente, is a popular stage model that conceptualizes the process of intentional behavior change. The model proposes that people move through five stages of change: (a) no intention to change behavior in the near future (i.e., *precontemplation*), (b) thinking about changing but have not decided (i.e., *contemplation*), (c) deciding to change behavior but have not successfully done so (i.e., *preparation*), (d) successfully changed behavior (i.e., *action*), and (e) preventing relapse (i.e., *maintenance*). The model posits that different barriers—which are referred to as processes of change or behavioral strategies—affect movement through the various stages. Early on (precontemplation and contemplation stages), behavioral strategies such as increasing knowledge (i.e., *consciousness raising*) and increasing concern regarding unhealthy behaviors (i.e., *dramatic relief*) matter most. Later (action phase), processes such as providing rewards for positive behaviors (i.e., *contingency management*), helping relationships (i.e., *social support*), and avoiding or removing cues to unhealthy behavior and adding cues to health behavior (i.e., *stimulus control*) matter most. The transtheoretical model incorporates elements of self-efficacy by taking into account the degree of confidence the individual has in their ability to maintain their desired behavioral change. This model can be helpful in determining the most appropriate interventions in health behavior change suited for the individual's current stage. As compared with the other models, it recognizes that there are individual differences that might make an intervention ineffective for some participants if they are not ready to engage in health behavior change. Meta-analyses and systematic reviews of the transtheoretical model suggest that it is effective in modeling health behavior change (e.g., Norcross, Krebs, & Prochaska, 2011). However, like most stage theories, there is mixed evidence with respect to its effectiveness relative to continuum models, the number and sequence of stages, the process by which people move from one stage to the next, and applicability across various health behavior domains (e.g. Armitage, 2009; Riemsma et al., 2003). For example, in a review of the transtheoretical model literature, Armitage (2009) suggests that a two-phase model incorporating motivational and volitional processes better conceptualizes the behavior change process than the five stages of the transtheoretical model.

Precaution Adoption Process Model

The precaution adoption process model, developed by Neil Weinstein and Peter Sandman, provides a framework to describe the particular sequence of change in which an individual

might engage. The model focuses on an individual's mental state that impacts why they decide to initiate action and how that decision translates into behavior. Individuals progress through seven stages that range from being unaware of an issue (stage 1) or unengaged by an issue (stage 2), to acting upon an issue (stage 6) and maintaining the intent to act (stage 7). At stage 3, undecided about acting, an individual can proceed to either deciding to act (stage 5) and proceed along the stages, or decide not to act (stage 4), in which case there is no continued advancement along the stages and the individual is considered in a state of moratorium. In general, the precaution adoption process model differs from the transtheoretical model in that it is more focused on the mental state of each stage and less focused on time until an intended action. In the precaution adoption process model, individuals may regress back in stages, and it is critical to not assume equal spacing between stages. Some individuals may be in one stage for such a brief period; it seems they skipped it altogether. For example, a doctor's advice may move an individual from unengaged (stage 2) to acting (stage 6). Moreover, the difficulty of the action of interest may impact the utility of this model, as behavior change that is easy (e.g., changing toothpastes, wearing seatbelts) may make the stages matter less, making it possible to combine stages with a more comprehensive treatment.

Although the precaution adoption process model has not been tested as widely as the transtheoretical model or health action process approach (HAPA) (see below), its utility has been demonstrated in work on radon testing, osteoporosis prevention, and dietary change (Weinstein, Sandman, & Blalock, 2008). For example, in encouraging undecided people to commit to radon testing (moving from stages 3 to 5), an intervention demonstrating high risk was more effective than an intervention demonstrating the ease of testing. Conversely, in encouraging decided people to act (moving from stages 5 to 6), the ease of testing intervention was more effective than the risk intervention. Future research, however, should focus on providing a more comprehensive understanding of why an individual would transition between stages.

Health Action Process Approach

The HAPA, developed by Ralf Schwarzer, is a stage theory of health behavior change that proposes that individuals pass through varying mindsets on their way to behavior change and therefore interventions that tailor to these changing mindsets will be most effective. In this manner, the adoption, initiation, and maintenance of health behaviors should be conceived of as a structured process, including a *motivation* phase and a *volition* phase, with changing perceptions of specific types of self-efficacy throughout.

Similar to continuum models discussed above, constructs such as risk perceptions, outcome expectations (i.e., attitudes), and task self-efficacy are thought to predict the formation of goals and intentions, an important aspect of the motivational phase. In contrast, the volitional phase is impacted by self-monitoring behaviors and awareness of standards (i.e., *planning, action control*) and confidence in maintaining behavior change (i.e., *maintenance self-efficacy*) and responding to behavioral lapses (i.e., *recovery/coping self-efficacy*). The volitional phase is further differentiated between people that have translated their intent to action and those that have not (i.e., the *inactive stage*). Individuals in the inactive stage are motivated to change but do not act because they lack the skills and internal or external resources to translate intention to action. Therefore, planning becomes an important part of the process of change, and post-intentional planning becomes a mediator between intent and action. Planning can further be divided into *action planning* and *coping planning*. Action planning involves the initiation of health behaviors, whereas coping planning is required for initiation and maintenance. According to the model, perceived self-efficacy is required throughout the process of behavior

change. However, the challenges that individuals face change during the process, and therefore the type of self-efficacy required also changes. For example, changing diet and physical activity indicates the need for action efficacy early on (the confidence to make difficult changes) but maintenance or recovery efficacy later in the process (the confidence to resist temptation and respond to lapses).

In general, research is supportive of the HAPA model (e.g., Schwarzer, 2008), although certain tenets are not always supported (e.g., Barg et al., 2012; Sutton, 2008). For example, in a study of the determinants of physical activity intentions and behaviors, action self-efficacy predicted behavioral intentions, whereas maintenance self-efficacy predicted planning and behavior; however, contrary to the model, planning did not predict behavior (Barg et al., 2012). Some have argued that the HAPA model is best thought of as a continuum model that builds on prior models like social cognitive theory and the theory of planned behavior by elucidating the intention to behavior link (Sutton, 2008).

Are Stage Models More Effective Than Continuum Models?

Although there is evidence for stage-based models as discussed above, meta-analyses of smoking cessation interventions and diet and physical activity interventions suggest that continuum and stage-based models do not differ in terms of effectiveness (e.g. Prestwich, Whittington, Dombrowski, Rogers, & Michie, 2014; Riemsma et al., 2003). Interestingly, Riemsma et al. (2003) also found that studies explicitly referencing theory were not reliably more effective than studies that did not explicitly reference theory. Of course, the interventions that did not explicitly use a particular theory may have targeted theoretically important constructs (e.g., self-efficacy, attitudes toward the behavior, etc.). Furthermore, many of the theory-based interventions did not explicitly link intervention techniques to theory constructs or did not measure the purported theory-based mediating constructs. As discussed by Sheeran et al. (2017), in order to promote health and advance health behavior theory, researchers will need to fully test mechanisms of change to know why an intervention succeeds or fails. Because stage models are inherently more complex and more difficult to use than continuum models, it is necessary to show that their effectiveness goes above and beyond continuum models.

Challenges and Promising New Directions

As pointed out by many health behavior theorists (e.g., Milne et al., 2000; Sheeran et al., 2016), there is extensive overlap in the constructs of health behavior theories even though they are problematically assessed with different measures. For example, the construct of *attitude* in the theory of reasoned action/planned behavior is conceptually very similar to *costs and benefits* in the health belief model or *outcome expectations* in social cognitive theory. Similarly, concepts related to self-efficacy are in the majority of health behavior theories. For example, Sheeran et al. (2016, 2017) identified constructs similar to attitude (i.e., expectations and evaluations of the behavior/outcomes of the behavior) and self-efficacy in most major health behavior theories. An important goal in health behavior change will be to develop a unified way to classify and measure these constructs of interest (see Michie et al., 2014).

Another future direction for health behavior change will be to elucidate behavior change processes that are less intentional or deliberative. In a meta-analysis of experimental studies

by Sheeran et al. (2016), the associations of attitudes, norms, and self-efficacy with behavior remained predictive when controlling for intentions, perhaps indicating that these predictors of behavior change may not be entirely deliberative or controlled. Although few moderators were identified by Sheeran et al. (2016), one finding of interest was that attitudes and self-efficacy were less impactful for frequent versus infrequent behaviors. The authors suggest that these frequent behaviors may be more habitual or automatic. Newer health behavior theories do emphasize habit (e.g., habit theory; Wood & Neal, 2007) and implicit processes (e.g., health goal priming; Papies, 2016). Furthermore, many health behavior theories emphasize the processes that might enhance the associations between intentions and behavior. For example, the HAPA emphasizes action planning and coping planning (Schwarzer, 2008).

An important goal for health behavior theories going forward will be to specify what constructs are most important for what types of behaviors, what kinds of people, and what stage of change. For example, behavioral willingness (versus intention) is a better predictor of risky behaviors, whereas intention (versus willingness) is a better predictor of health protective behaviors (Gerrard et al., 2008). Sequential multiple assignment randomized trial (SMART) designs are becoming increasingly used to build individualized treatments to influence health behaviors. For example, Sherwood et al. (2016) are conducting a SMART intervention that is aimed at testing secondary intervention procedures with initial non-responders in a weight loss study. In this way, more intensive procedures can be utilized with only those that need them, and the most optimal procedures can be identified with respect to timing and content (e.g., introducing portion controlled meals versus introducing self-regulation training). Taking another tack, researchers have had success tailoring intervention messages and strategies to match people's underlying psychological dispositions (Rothman et al., 2016). In this way, interventions can capitalize on people's self-regulatory strengths by encouraging them to pursue goals in ways that fit their natural inclinations.

As discussed above, models of health behavior change are generally effective in promoting initial changes in behavior and generally focus on the processes related to deciding and starting to make a change. However, even people who succeed in making changes in their behavior often fail to maintain those changes (Sarwer, von Sydow Green, Vetter, & Wadden, 2009). Strategies to promote maintenance have begun to emerge, such as continually reinforcing the determinants of initial change (a continued care model), instilling the strategies and skills necessary for maintenance at the outset of intervention, and targeting maintenance-specific predictors once people have had initial success (Rothman, Baldwin, Burns, & Fuglestad, 2016). For example, Rothman et al. (2011) have specified which predictors are most important for initiating change (outcome expectations, self-efficacy) and maintaining change (satisfaction with outcomes, satisfaction with the behaviors). Interventions on smoking cessation and weight loss provide support for these propositions (Rothman et al., 2011). A direct comparison of a continued care treatment with a satisfaction-based maintenance treatment found that both were similarly effective in encouraging behavioral maintenance (Rothman et al., 2016).

Conclusion

Clearly, theory and research have made great progress in affecting positive changes in health behavior. As demonstrated in various meta-analyses, constructs such as self-efficacy, attitudes, threat susceptibility, self-monitoring, and stages of change have been shown to effectively predict and influence healthful changes in behavior (e.g., Norcross et al., 2011; Sheeran et al.,

2014, 2016). However, many challenges remain. For example, it is unclear whether continuum or stage-based models are more effective and/or useful in influencing behavior change (Prestwich et al., 2014). It is also unclear as to how to best classify and operationalize health behavior theories and constructs (Michie et al., 2014). Going forward it will be important to conduct direct comparisons of competing theories and to experimentally examine how interventions affect theoretical mechanisms of change and how those mechanisms lead/do not lead to behavior change (Sheeran et al., 2017). Finally, although theories of health behavior change are generally effective in getting people to make initial changes in their behaviors, long-term maintenance of those behavioral changes remains challenging.

Author Biographies

Paul T. Fuglestad is an assistant professor at the University of North Florida. His work applies theory from personality and social psychology to understand and affect health behavior change.

Jody S. Nicholson is a developmental psychologist and an assistant professor at University of North Florida. Her research focuses on parents' role in children's health behavior decisions.

Lauren James is a clinical research coordinator in the Center for Healthcare Delivery Science at Nemours Children's Specialty Care and works to improve health outcomes for children, adolescents, and their families. Through several grant-funded studies, her research involves working on the design, development, and testing of web-delivered resources for youth with type 1 diabetes and their parents.

References

- Armitage, C. J. (2009). Is there utility in the transtheoretical model? *British Journal of Health Psychology*, 14, 195–210. doi:10.1348/135910708X368991
- Barg, C. J., Latimer, A. E., Pomery, E. A., Rivers, S. E., Rench, T. A., Prapavessis, H., & Salovey, P. (2012). Examining predictors of physical activity among inactive middle-aged women: An application of the health action process approach. *Psychology & Health*, 27(7), 829–845. doi:10.1080/08870446.2011.609595
- Gerrard, M., Gibbons, F. X., Houlihan, A. E., Stock, M. L., & Pomery, E. A. (2008). A dual-process approach to health risk decision making: The prototype willingness model. *Developmental Review*, 28, 29–61. doi:10.1016/j.dr.2007.10.001
- Michie, S., West, R., Campbell, R., Brown, J., & Gainforth, H. (2014). *ABC of behavior change theories* (1st ed.). London: Silverback.
- Milne, S., Sheeran, P., & Orbell, S. (2000). Prediction and intervention in health related behavior: A meta analytic review of protection motivation theory. *Journal of Applied Social Psychology*, 30(1), 106–143. doi:10.1111/j.1559-1816.2000.tb02308.x
- Norcross, J. C., Krebs, P. M., & Prochaska, J. O. (2011). Stages of change. *Journal of Clinical Psychology*, 67(2), 143–154. doi:10.1002/jclp.20758
- Papies, E. K. (2016). Health goal priming as a situated intervention tool: How to benefit from nonconscious motivational routes to health behavior. *Health Psychology Review*, 10, 408–424. doi:10.1080/17437199.2016.1183506
- Prestwich, A., Whittington, C., Dombrowski, S. U., Rogers, L., & Michie, S. (2014). Does theory influence the effectiveness of health behavior interventions? *Health Psychology*, 33(5), 465–474. doi:10.1037/a0032853.supp

- Riemsma, P., R., Pattenden, J., Bridle, C., Sowden, J., Mather, L., Watt, S., & Walker, A. (2003). Systematic review of the effectiveness of stage based interventions to promote smoking cessation. *BMJ: British Medical Journal*, 326(7400), 1175–1177. doi:10.1136/bmj.326.7400.1175/r326/7400/1175
- Rothman, A. J., Baldwin, A. S., Burns, R. J., & Fuglestad, P. T. (2016). Strategies to promote the maintenance of behavior change: Moving from theoretical principles to practice. In M. A. Diefenbach, S. M. Miller, & D. J. Bowen (Eds.), *Handbook of health decision science* (pp. 121–132). New York, NY: Springer. doi:10.1007/978-1-4939-3486-7_9
- Rothman, A. J., Baldwin, A. S., Hertel, A. W., & Fuglestad, P. T. (2011). Self-regulation and behavior change: Disentangling behavioral initiation and behavioral maintenance. In K. Vohs & R. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 106–122). New York, NY: Guilford Press.
- Sarwer, D. B., von Sydow Green, A., Vetter, M. L., & Wadden, T. A. (2009). Behavior therapy for obesity: Where are we now? *Current Opinion in Endocrinology, Diabetes and Obesity*, 16(5), 347–352. doi:10.1097/MED.0b013e32832f5a79
- Schwarzer, R. (2008). Modeling health behavior change: How to predict and modify the adoption and maintenance of health behaviors. *Applied Psychology: An International Review*, 57(1), 1–29. doi:10.1111/j.1464-0597.2007.00325.x
- Sheeran, P., Harris, P. R., & Epton, T. (2014). Does heightening risk appraisals change people's intentions and behavior? A meta-analysis of experimental studies. *Psychological Bulletin*, 140(2), 511–543. doi:10.1037/a0033065
- Sheeran, P., Klein, W. M. P., & Rothman, A. J. (2017). Health behavior change: Moving from observation to intervention. *Annual Review of Psychology*, 68(1), 573–600. doi:10.1146/annurev-psych-010416-044007
- Sheeran, P., Montanara, E., Bryan, A., Klein, W. M. P., Miles, E., & Rothman, A. J. (2016). The Impact of changing attitudes, norms, and self-efficacy on health-related intentions and behavior: A meta-analysis. *Health Psychology*, May, 1–88. doi:10.1037/hea0000387
- Sherwood, N. E., Butryn, M. L., Forman, E. M., Almirall, D., Seburg, E. M., Lauren Crain, A., ... Jeffery, R. W. (2016). The BestFIT trial: A SMART approach to developing individualized weight loss treatments. *Contemporary Clinical Trials*, 47, 209–216. doi:10.1016/j.cct.2016.01.011
- Sutton, S. (2008). How does the Health Action Process Approach (HAPA) bridge the intention-behavior gap? An examination of the model's causal structure. *Applied Psychology*, 57(1), 66–74. doi:10.1111/j.1464-0597.2007.00326.x
- Weinstein, N. D., Sandman, P. M., & Blalock, S. J. (2008). The precaution adoption process model. In K. Glanz, B. K. Rimer, K. Viswanath, K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed., pp. 123–147). San Francisco, CA: Jossey-Bass.
- Wood, W., & Neal, D. T. (2007). A new look at habits and the habit-goal interface. *Psychological Review*, 114(4), 843–863. doi:10.1037/0033-295X.114.4.843

Suggested Reading

- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York, NY: W H Freeman/Times Books/Henry Holt & Co.
- Norcross, J. C., Krebs, P. M., & Prochaska, J. O. (2011). Stages of change. *Journal of Clinical Psychology*, 67(2), 143–154. doi:10.1002/jclp.20758
- Rothman, A. J., Baldwin, A. S., Hertel, A. W., & Fuglestad, P. T. (2011). Self-regulation and behavior change: Disentangling behavioral initiation and behavioral maintenance. In K. Vohs & R. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 106–122). New York, NY: Guilford Press.

- Sheeran, P., Klein, W. M. P., & Rothman, A. J. (2017). Health behavior change: Moving from observation to intervention. *Annual Review of Psychology*, 68(1), 573–600. doi:10.1146/annurev-psych-010416-044007
- Sheeran, P., Montanara, E., Bryan, A., Klein, W. M. P., Miles, E., & Rothman, A. J. (2016). The Impact of changing attitudes, norms, and self-efficacy on health-related intentions and behavior: A meta-analysis. *Health Psychology*, May, 1–88. doi:10.1037/hea0000387

Health Behavior Interventions

Casey K. Gardiner, Arielle S. Gillman, Courtney J. Stevens, and Angela D. Bryan

Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA

Of the 10 leading causes of death in the United States, 7 are chronic diseases that have associated behavioral risk and protective factors (Johnson, Hayes, Brown, Hoo, & Ethier, 2014). These data suggest that health behavior change interventions have an enormous potential impact in preventing morbidity and premature mortality. However, designing and conducting a successful health behavior intervention is not a simple process; there are myriad methodological, theoretical, and practical factors that warrant consideration when attempting to translate findings from the health psychology/behavioral medicine literature into a feasible intervention that is suitable for use with the general public. While other chapters in this volume address specific health concerns that may warrant interventions or how to evaluate intervention effects, the present entry endeavors to highlight key considerations for and offer suggestions that are broadly relevant to the implementation of health behavior interventions across domains. Our aim is to provide an overview of the important considerations that merit attention before conducting an intervention in order to maximize its impact.

What Is the Nature of the Behavior?

The first step when attempting to design an intervention for improving a specific health outcome is to specify the behavior that is to be increased or decreased. While this may seem intuitively obvious, the lack of explicit specification regarding the target behavior to be modified has led to misguided campaigns for health promotion in the past. For instance, Johnston and Dixon (2008) point out that when researchers first demonstrated the link between tobacco use and lung cancer, a great deal of effort was put into attempting to manufacture a safer cigarette (e.g., adding filters, reducing tar content). Thus, the focus was placed on modifying the lethality of the cigarette rather than on modifying the behavioral action of smoking. Granted, financial incentives may have influenced the push for “safer” cigarettes, but even if it were possible to achieve a safe

cigarette, the flaw in this logic is the focus on the outcome (i.e., avoiding cancer) rather on the modification of the behavior (i.e., smoking cigarettes) that engenders that outcome.

Consider the similar focus that is placed on weight loss in the health literature. Surely, weight loss is a desirable outcome for many individuals dealing with obesity and/or with illnesses made more severe as a result of being overweight/obese (e.g., heart disease, arthritis, asthma, etc.). However, *weight loss* is not a behavior that can be performed. Rather, weight loss is an outcome that may (or may not) be achieved via changes in specific daily behavioral routines (e.g., what foods are consumed, amount of time spent engaging in sedentary activities, number of hours slept, etc.). Thus, when designing interventions to promote health, it is imperative that researchers explicitly define what *behavior* is to be targeted (Michie & Johnston, 2004).

Once the behavior in question has been clearly identified, (e.g., time spent walking per day), additional concerns must be addressed regarding the details of when, where, how, and under what conditions a behavior will be carried out (Michie & Johnston, 2004). For instance, researchers need to specify guidelines regarding the duration, pace/intensity, and number of days per week the individual will be asked to walk. Reviews of the literature have found that the answers to these questions are rarely specified adequately in intervention research (Johnston & Dixon, 2008; Michie & Johnston, 2004), and this lack of specificity may contribute to the generally small effect sizes observed in health intervention research.

Numerous aspects of the intervention, including its content and behavioral goals, may vary greatly depending on the nature of the target behavior, given that behavior is a very complex phenomenon that varies across numerous dimensions, for instance, the extent to which a behavior is performed publicly or privately (e.g., blood donation versus cancer screening), the level of effort required to perform the behavior (e.g., teeth flossing versus cardiorespiratory exercise), the frequency with which the behavior ought to occur (e.g., yearly flu shots versus daily fruit and vegetable consumption), how socially acceptable the behavior is (e.g., seat belt use versus acquiring clean needles from a needle exchange program), and whether the behavior in question is to be approached or increased versus avoided or decreased (e.g., increasing sunscreen use versus avoiding alcohol consumption; see McEachan, Lawton, & Conner, 2010).

Without careful consideration of these dimensions that define the behavior in question, researchers may fail to consider the critical factors that prompt (e.g., cues in the environment) and reinforce specific behaviors, or may use the wrong intervention techniques to try to change those behaviors. For example, approach-/avoidance-based interventions that successfully decrease substance use (a behavior that should be decreased) do not appear to be effective in promoting healthy eating behavior (a behavior that should be increased; Becker, Jostmann, Wiers, & Holland, 2015; Gardiner & Bryan, 2015). This is likely due to key differences that characterize the behaviors in question in addition to the increase/decrease dimension, perhaps most notably that eating is necessary for survival and thus may be more resistant to change or may require change by different mechanisms. At best, this type of oversight may result in researchers and health professionals aimlessly embarking upon or recommending interventions that lack theoretical support and/or ignore critical information known to change the behavior in question (Michie & Johnston, 2004). At worst, this oversight may result in burn-out, attrition, adverse events, and/or new or worsening health conditions.

Whom Will the Intervention Target?

In addition to selecting a target behavior on which to intervene, it is necessary to identify the target population for the intervention. The first fundamental question to ask is whether the

potential intervention participants are available and likely to benefit from a health behavior intervention. Specifically, the targeted individuals should be in need of health behavior change. For example, a student or university employee might wish to conduct an intervention with college student athletes, given that they are an easy population to access and track through the university's athletic department. However, this population might not be the best choice for interventions on the full range health behaviors that would be relevant to the general population. For example, in spite of low levels of physical activity on a national level (e.g., Troiano et al., 2008), it is unlikely that college student athletes would gain much from an intervention designed to increase physical activity. Similarly, while interventions to change eating behavior for the general population may focus on decreasing consumption of unhealthy foods and increasing consumption of healthy foods, this may not be an appropriate behavioral goal for student athletes who may have completely different dietary issues (e.g., eating disorders, adequate caloric and nutrient intake). On the other hand, college athletes may be an ideal target population for behavior change interventions designed to decrease binge drinking or risky sexual behavior. In sum, determining whether a population stands to benefit from a particular behavior change intervention needs to be carefully considered based on experience with the population, epidemiological research, and broader health behavior research.

Once the population and behavior have been jointly identified, it is wise to tailor the intervention design to the population in question. Such tailoring would include selecting intervention delivery mechanisms that are most likely to be suitable for that group, based upon their environment and typical behavior. For example, a dietary intervention delivered at farmers markets (e.g., coupons to enhance access to fruits and vegetables, exposure opportunities designed to improve children's attitudes toward healthy foods) might be quite effective in Vermont, where there are 15 farmers markets per 100,000 residents, but such an intervention could reach a far smaller percentage of the population in Texas, where there are only 0.7 farmers markets per 100,000 residents (Centers for Disease Control and Prevention [CDC], 2013b). The selection of a population and setting can inform other characteristics of the intervention design, perhaps especially if the target population is specialized in some way (e.g., homeless youth, incarcerated individuals, patients with particular physical or mental health conditions). Intervention content can be delivered using a multitude of techniques, including (but not limited to) online formats, text messaging, television advertisements, print materials, and in-person sessions. When selecting and developing an intervention, the interventionist must determine what type of intervention modality might be most feasible and effective given the population target, health behavior(s), setting, and available resources.

How Will the Intervention Change Behavior?

Perhaps the most imperative question when designing an intervention is this: What are the determinants of the health behavior for the population that—when addressed in a successful intervention—will lead to behavior change? In answering this question, the literature is very clear; it is wise to choose a theoretical framework. Research suggests that behavior change interventions grounded in theories are more successful in changing behavior (e.g., Glanz & Bishop, 2010) than those that are not theory based. The literature in social and health psychology offers many possible theoretical foundations for health behavior interventions. One example is the theory of planned behavior (TPB; Ajzen & Madden, 1986), which has been applied to numerous health behaviors across many domains, including substance use, health screening, eating, exercise, sexual health, and oral hygiene (Godin & Kok, 1996). The theory

posits that behavior is most proximally predicted by intentions to engage in the behavior. Intentions are in turn predicted by attitudes (positive or negative evaluations) about the behavior, subjective norms (the perceived social forces to perform or not perform the behavior), and perceived behavioral control (beliefs regarding the ease or difficulty of behavior performance). An intervention based upon the TPB could seek to change any or all of those four constructs.

Social cognitive theory (Bandura, 1977) also has applications for health behavior change. This theory suggests that perceived self-efficacy, or the belief that one can successfully complete an action, is a critical determinant of behavior engagement, such that stronger self-efficacy should be associated with increased behavior. The theory also suggests methods for enhancing self-efficacy, including behavior performance (i.e., practice) and modeling, which can be components of health behavior interventions. Research has demonstrated that interventions including behavior practice (e.g., role-playing condom negotiations and applying a condom to a penile model; Bryan, Aiken, & West, 1996) cause increases in self-efficacy.

Self-determination theory (Ryan & Deci, 2000) presents yet another potential framework for health behavior interventions. The theory posits that by meeting innate human needs, such as autonomy, competence, and relatedness, individuals will be more able to self-regulate their behavior in a fashion that supports behavior maintenance (Ryan, Patrick, Deci, & Williams, 2008). Interventions based upon this theory may attempt to enhance feelings of autonomy or build intrinsic motivations to maintain behavior. For example, therapies that support the development of patient competence can enhance autonomous regulation of behavior and perceived competence, which are associated with improved health behavior outcomes (e.g., smoking abstinence, exercise behavior volume; Stevens & Bryan, 2015; Williams et al., 2015).

Beyond these commonly applied theories, there are scores of others, with a recent review noting over 80 extant theories that have been applied to health behavior change (Davis, Campbell, Hildon, Hobbs, & Michie, 2014). These include those that apply broadly to many behaviors, as well as theoretical models that are specific to particular health behaviors. Different theoretical orientations offer different strengths and weaknesses, and an examination of the literature can guide the selection of a theoretical model that may be most appropriate to the target behavior, population, and intervention approach. For example, intervention sessions conducted in person may offer opportunities to enhance self-efficacy through role play or behavior practice, while those delivered online or via print materials require different approaches and may be more successful in targeting other constructs. Overall, the selection of a theory provides a useful framework for understanding the mechanisms by which an intervention can cause behavior change, which can in turn drive the development of intervention content designed to affect each posited mediator, a process sometimes referred to as “intervention mapping” (see Bartholomew, Parcel, Kok, Gottlieb, & Fernandez, 2011).

What Materials Will You Use to Intervene?

After the target behavior, population, and theoretical basis of the intervention are identified, an important next step in selecting an appropriate intervention is to identify whether empirically supported interventions for the target behavior already exist. A great number of successful interventions based on a variety of theoretical perspectives are easily accessible to individuals who wish to implement such an intervention themselves. As such, we recommend that those attempting to conduct an intervention to change behavior do not “reinvent the wheel” and

instead save time and resources by implementing an empirically supported health behavior intervention if this is possible.

For example, the Centers for Disease Control and Prevention (CDC) website has a list of empirically supported HIV prevention interventions for sexual risk behavior reduction (CDC, 2013a), where specific interventions can be selected. Based on the behavioral target and population, one might choose the CLEAR (Choosing Life: Empowerment! Action! Results!) intervention (CDC, 2015), which is an individual-level intervention that uses a cognitive behavioral approach to change substance use and risky sexual behavior. As another example, the American College of Sports Medicine (ACSM) releases thorough position stands that provide recommendations for empirically supported intervention techniques for increasing exercise behavior (see Donnelly et al., 2009). These websites and articles often have detailed instructions to help individuals begin implementing these interventions. Each site also provides a set of references that detail the empirical support for the intervention and typically include instructions for implementing the intervention as well as information about any specialized training that might be required.

Literature searches for primary articles in academic databases can also serve as an important resource for finding empirical papers that provide tests of the effectiveness of health behavior interventions for specific behaviors or populations. Review papers or multi-study papers might be especially useful in guiding intervention selection. For example, Kahn et al.'s (2002) review paper discusses the varying success of physical activity interventions implemented at different levels (i.e., individual level, community level) that might aid practitioners and program managers in selecting an appropriate intervention for a given population. As another illustration, Johnson and Goldstein's (2003) article titled "Do Defaults Save Lives?" discusses potential benefits of a "nudge"-style intervention to increase organ donation, an approach that the authors suggest might be particularly effective in controlled settings, such as a school. Research of this type is particularly useful when it provides evidence about approaches that have been effective and those that have been ineffective in catalyzing health behavior change in different settings or with different populations. Additionally, Michie, van Stralen, and West (2011) have begun to develop an exhaustive classification framework for the full range of techniques that can be applied to change behavior, which may provide broader guidance for those developing novel interventions in domains where prior empirically supported interventions are not available.

Once an intervention modality has been selected that is expected to be effective with the target population, tailoring intervention tone and content to the characteristics of the population can lead to better engagement in the intervention and more substantial intervention effects on behavior. For example, in a sample of juvenile justice-involved adolescents, an intervention including techniques that allowed the participants to assert autonomy, control the intervention discussion, and be characterized as "experts" on their own experience (in contrast to their typical role as passive recipients of rules in an authoritarian context) was more successful in changing risky sexual behavior than was a standard psychosocial intervention style that has previously been successful in college students (Bryan, Schmiede, & Broadbudd, 2009; Schmiede, Broadbudd, Levin, & Bryan, 2009). Cultural and environmental factors relevant to the target population also warrant investigation prior to conducting the intervention, as even the best designed intervention cannot succeed if participants cannot participate in the intervention or engage in the behavior. For example, a psychosocial intervention for physical activity cannot succeed if participants do not have the opportunity to exercise (e.g., working multiple sedentary jobs, lack of access to appropriate facilities), and individuals cannot benefit from the intervention if they are unable to enroll in it (e.g., participants who cannot attend

in-person sessions that occur in locations inaccessible via public transportation). Thus, it is wise to develop an understanding of characteristics of the target population, including a thorough needs assessment and collection of qualitative data about the barriers for integrating the particular health behavior at focus into their daily lives, prior to selecting intervention content and delivery methods. Considering the population and the social, micro- and macroenvironment in which their behavior occurs (cf., Egger & Swinburn, 1997) can offer critical insights that may guide intervention design choices.

How Do We Know if the Intervention Is Successful?

Selection of a very specific health behavior, target population, theoretical approach, and intervention modality/content are the primary considerations for conducting a health behavior intervention. However, whether an intervention is conducted in the context of formal research or in order to inform policy makers and stakeholders as they plan for future interventions, it is wise to develop a plan to collect data that will allow for the assessment of intervention efficacy. Such assessments should be incorporated into the intervention design from the start, so that changes in the determinants of behavior, overt behavior itself, and/or health outcomes expected as a result of behavior change can be tracked. Aiken (2011) describes in detail the phases of intervention evaluation that allow for the determination of (a) whether an intervention led to behavior change and (b) which of the theoretical constructs thought to underlie the behavior were the “active ingredients” that led to behavior change. Typically, this approach is facilitated by the careful implementation of the intervention in, ideally, a randomized experiment with a standard of care control or comparison condition (although see West, Cham, & Liu, 2014; West et al., 2008, for alternatives to the randomized controlled trial). The second step involves the use of statistical mediational analysis to determine whether the intervention led to changes in the theoretical mediators of behavior change (e.g., self-efficacy) and whether that change in the mediator was then associated with observed changes in the behavior (cf., Fairchild & MacKinnon, 2009). The advantage of this approach to intervention implementation and analysis is that it allows for the researcher or program planner to determine not just *whether* an intervention was successful but also *why* it was successful and made changes to the intervention based on that knowledge (cf., West & Aiken, 1997). Imagine that an intervention was conducted to increase fruit and vegetable consumption with adolescents. There is existing literature to support the idea that the constructs of the TPB are associated with fruit and vegetable consumption (e.g., Guillaumie, Godin, & Vézina-Im, 2010). Further imagine that a TPB-based intervention was implemented in a randomized experimental trial in which the intervention was compared with a no-treatment control group. The intervention resulted in very small changes in behavior in the intervention group and no change in the control group. Is the answer to throw out the intervention and start over given the small effects? If the investigators had gone the extra step to conduct a mediational analysis, they might find that while the intervention was successful at changing norms and attitudes, it was unsuccessful at changing perceived behavioral control/self-efficacy, a key determinant of fruit and vegetable consumption. This information would tell intervention designers that they need not start over; rather, they need to bolster the intervention content meant to increase self-efficacy for fruit and vegetable consumption.

The importance of valid and reliable measures of both the behavior and the constructs expected to mediate changes in behavior cannot be overstated. Without these measures at both baseline and after the completion of the intervention, in both the experimental interven-

tion and control groups, an analysis of whether and why changes in behavior occurred as a result of the intervention is impossible (West & Aiken, 1997). As the great methodologist and statistician Ronald Fisher said (*ca.* 1938), "To call in the statistician after the experiment is done may be no more than asking him to perform a postmortem examination: he may be able to say what the experiment died of." Thus, attention to research design and measurement prior to the initiation of the project makes all the difference.

In sum, the development, implementation, and evaluation of interventions to change health behavior warrant substantial forethought in order for the intervention to have its best chance of success and for intervention designers to understand why the intervention worked (or did not) and how it might be improved. This preliminary work can be accomplished in the set of steps we have outlined here. Interventionists should first specify the exact behavior they wish to change and address the characteristics of the population and the sociocultural context of their engagement in the behavior, including understanding the best mode of content delivery for that population. Next is the specification of a theoretical basis of the mechanisms of behavior change and the selection and tailoring or development via intervention mapping of a theory-based intervention. Finally, implementing the intervention using adequate measurement and a research design that permits both an outcome analysis (whether an intervention resulted in behavior change) and a mediational analysis (which theoretical constructs were successfully changed by the intervention and led to behavior change) allows for the evolution of ever-more successful behavior change efforts. Notably, these steps need not be followed in the specific order that we set forth. For example, it is possible to seek out an extant intervention for a particular behavior or population before defining a theoretical orientation, but once the theoretical orientation of such an intervention is determined, it is important to measure constructs that are posited to affect behavior within that framework. Nevertheless, it remains important to address each of these concerns. This initial investment of time and effort can provide great dividends, by enhancing the responsiveness of populations to interventions and ultimately in promoting health behavior changes that lead to decreased morbidity and mortality and better quality of life.

Author Biographies

Casey K. Gardiner, PhD, recently completed graduate training at the University of Colorado Boulder, where she was a National Science Foundation Graduate Research Fellow. Her research examined psychological and neurobiological factors that contribute to health behavior change and maintenance, with particular emphasis on healthy eating behavior and incentive interventions. She is now a management consultant in Boston, MA.

Arielle S. Gillman, PhD, recently completed graduate training in social health psychology at the University of Colorado Boulder and is now pursuing her MPH at Johns Hopkins University as part of her postdoctoral research fellowship with the National Cancer Institute at the National Institutes of Health. Her research broadly focuses on understanding factors that drive the uptake and maintenance of healthy behaviors, including self-regulation, motivation, and decision-making processes, in order to eventually apply these findings through theory-based and empirically supported interventions to increase health behavior.

Courtney J. Stevens, PhD, recently completed graduate training at the University of Colorado Boulder, where she was supported by a National Research Service Award (NRSA) predoctoral training fellowship (NIH/NCI F31 CA180483-01; PI: Stevens). She is currently an NIH T-32 postdoctoral fellow at the Dartmouth Centers for Health and Aging.

Her research is focused on affective/experiential determinants of health behaviors and how health behaviors with high transdiagnostic treatment potential (e.g., exercise, mindfulness practice) are adopted and maintained over time and may be used to augment current best-practice treatment models.

Angela D. Bryan, PhD, is a professor in the Department of Psychology and Neuroscience at the University of Colorado Boulder. She has been continually funded by the National Institutes of Health to study health and risk behavior change for the past 18 years. Her work takes a transdisciplinary approach, integrating genomics, physiology, and neuroscience to the study of the psychological processes that underlie behavior change.

References

- Aiken, L. S. (2011). Advancing health behavior theory: The interplay among theories of health behavior, empirical modeling of health behavior, and behavioral interventions. In H. Friedman (Ed.), *Oxford handbook of health psychology* (pp. 612–636). New York, NY: Oxford University Press.
- Ajzen, I., & Madden, T. J. (1986). Prediction of goal-directed behavior: Attitudes, intentions, and perceived behavioral control. *Journal of Experimental Social Psychology*, 22(5), 453–474. doi:10.1016/0022-1031(86)90045-4
- Bandura, A. (1977). *Social learning theory*. Englewood Cliffs, NJ: Prentice Hall.
- Bartholomew, L. K., Parcel, G. S., Kok, G., Gottlieb, N. H., & Fernandez, M. E. (2011). *Planning health promotion programs: An intervention mapping approach* (3rd ed.). San Francisco, CA: Jossey-Bass.
- Becker, D., Jostmann, N. B., Wiers, R. W., & Holland, R. W. (2015). Approach avoidance training in the eating domain: Testing the effectiveness across three single session studies. *Appetite*, 85, 58–65. doi:10.1016/j.appet.2014.11.017
- Bryan, A. D., Aiken, L. S., & West, S. G. (1996). Increasing condom use: Evaluation of a theory-based intervention to prevent sexually transmitted diseases in young women. *Health Psychology*, 15(5), 371–382. doi:10.1037/0278-6133.15.5.371
- Bryan, A. D., Schmiede, S. J., & Broadus, M. R. (2009). HIV risk reduction among detained adolescents: A randomized, controlled trial. *Pediatrics*, 124(6), e1180–e1188. doi:10.1542/peds.2009-0679
- Centers for Disease Control and Prevention. (2013a). Effective behavioral interventions. Retrieved from <http://www.cdc.gov/hiv/prevention/programs/ebis/>
- Centers for Disease Control and Prevention. (2013b). *State indicator report on fruits and vegetables, 2013*. Atlanta, GA: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services.
- Centers for Disease Control and Prevention. (2015). CLEAR. Retrieved from <https://effectiveinterventions.cdc.gov/en/HighImpactPrevention/Interventions/CLEAR.aspx>
- Davis, R., Campbell, R., Hildon, Z., Hobbs, L., & Michie, S. (2014). Theories of behaviour and behaviour change across the social and behavioural sciences: A scoping review. *Health Psychology Review*, 9(3), 323–344. doi:10.1080/17437199.2014.941722
- Donnelly, J. E., Blair, S. N., Jakicic, J. M., Manore, M. M., Rankin, J. W., & Smith, B. K. (2009). Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine and Science in Sports and Exercise*, 41(7), 1532–1532. doi:10.1249/MSS.0b013e3181949333
- Egger, G., & Swinburn, B. (1997). An ecological approach to the obesity pandemic. *BMJ*, 315(7106), 477–480. doi:10.1136/bmj.315.7106.477
- Fairchild, A. J., & MacKinnon, D. P. (2009). A general model for testing mediation and moderation effects. *Prevention Science*, 10(2), 87–99. doi:10.1007/s11121-008-0109-6

- Gardiner, C., & Bryan, A.D. (2015, April). Can approach motivation training increase fruit and vegetable consumption? Poster presented at the 36th Annual Meeting & Scientific Sessions of the Society of Behavioral Medicine, San Antonio, TX.
- Glanz, K., & Bishop, D. B. (2010). The role of behavioral science theory in development and implementation of public health interventions. *Annual Review of Public Health*, 31, 399–418. doi:10.1146/annurev.publhealth.012809.103604
- Godin, G., & Kok, G. (1996). The theory of planned behavior: A review of its applications to health-related behaviors. *American Journal of Health Promotion*, 11(2), 87–98. doi:10.4278/0890-1171-11.2.87
- Guillaumie, L., Godin, G., & Vézina-Im, L. A. (2010). Psychosocial determinants of fruit and vegetable intake in adult population: A systematic review. *The International Journal of Behavioral Nutrition and Physical Activity*, 7(1), 12. doi:10.1186/1479-5868-7-12
- Johnson, E. J., & Goldstein, D. G. (2003). Do defaults save lives? *Science*, 302, 1338–1339.
- Johnson, N. B., Hayes, L. D., Brown, K., Hoo, E. C., & Ethier, K. A. (2014). CDC National Health Report: Leading causes of morbidity and mortality and associated behavioral risk and protective factors—United States, 2005–2013.
- Johnston, M., & Dixon, D. (2008). Current issues and new directions in psychology and health: What happened to behaviour in the decade of behaviour? *Psychology & Health*, 23(5), 509–513. doi:10.1080/08870440701816728
- Kahn, E. B., Ramsey, L. T., Brownson, R. C., Heath, G. W., Howze, E. H., Powell, K. E., ... Corso, P. (2002). The effectiveness of interventions to increase physical activity. *American Journal of Preventive Medicine*, 22(4S), 73–107. doi:10.1016/S0749-3797(02)00434-8
- McEachan, R. R., Lawton, R. J., & Conner, M. (2010). Classifying health-related behaviours: Exploring similarities and differences amongst behaviours. *British Journal of Health Psychology*, 15(2), 347–366. doi:10.1348/135910709X466487
- Michie, S., & Johnston, M. (2004). Changing clinical behaviour by making guidelines specific. *BMJ: British Medical Journal*, 328(7435), 343–345. doi:10.1136/bmj.328.7435.343
- Michie, S., van Stralen, M. M., & West, R. (2011). The behaviour change wheel: A new method for characterising and designing behaviour change interventions. *Implementation Science*, 6(1), 42–52. doi:10.1186/1748-5908-6-42
- Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist*, 55(1), 68–78. doi:10.1037/0003-066X.55.1.68
- Ryan, R. M., Patrick, H., Deci, E. L., & Williams, G. C. (2008). Facilitating health behaviour change and its maintenance: Interventions based on self-determination theory. *European Health Psychologist*, 10(1), 2–5.
- Schmiege, S. J., Broaddus, M. R., Levin, M., & Bryan, A. D. (2009). Randomized trial of group interventions to reduce HIV/STD risk and change theoretical mediators among detained adolescents. *Journal of Consulting and Clinical Psychology*, 77, 38–50. doi:10.1037/a0014513
- Stevens, C. J., & Bryan, A. D. (2015). A case for leveraging integrated regulation strategies to optimize health benefits from self-determined exercise behavior. *Annals of Behavioral Medicine*, 49(5), 783–784. doi:10.1007/s12160-015-9722-3
- Troiano, R. P., Berrigan, D., Dodd, K. W., Masse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the United States measured by accelerometer. *Medicine and Science in Sports and Exercise*, 40(1), 181–188. doi:10.1249/mss.0b013e31815a51b3
- West, S. G., & Aiken, L. S. (1997). Towards understanding individual effects in multiple component prevention programs: Design and analysis strategies. In K. Bryant, M. Windle, & S. West (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research* (pp. 167–209). Washington, DC: American Psychological Association.
- West, S. G., Cham, H., & Liu, Y. (2014). Causal inference and generalization in field settings: Experimental and quasi-experimental designs. In H. T. Reis & C. M. Judd (Eds.), *Handbook of*

research methods in personality and social psychology (2nd ed., pp. 49–80). New York, NY: Cambridge University Press.

West, S. G., Naihua, D., Pequegnat, W., Gaist, P., Des Jarlais, D. C., Holtgrave, D., ... Mullen, P. (2008). Alternatives to the randomized controlled trial. *American Journal of Public Health*, 98(8), 1359–1366. doi:10.2105/AJPH.2007.124446

Williams, D. M., Dunsiger, S., Miranda, R., Jr., Gwaltney, C. J., Emerson, J. A., Monti, P. M., & Parisi, A. F. (2015). Recommending self-paced exercise among overweight and obese adults: A randomized pilot study. *Annals of Behavioral Medicine*, 49(2), 280–285. doi:10.1007/s12160-014-9642-7

Suggested Reading

Abraham, C., & Michie, S. (2008). A taxonomy of behavior change techniques used in interventions. *Health Psychology*, 27(3), 379–387. doi:10.1037/0278-6133.27.3.379

Bartholomew, L. K., Parcel, G. S., Kok, G., Gottlieb, N. H., & Fernandez, M. E. (2011). *Planning health promotion programs: An intervention mapping approach* (3rd ed.). San Francisco, CA: Jossey-Bass.

Michie, S., van Stralen, M. M., & West, R. (2011). The behaviour change wheel: A new method for characterising and designing behaviour change interventions. *Implementation Science*, 6(1), 42–52. doi:10.1186/1748-5908-6-42

West, S. G., & Aiken, L. S. (1997). Towards understanding individual effects in multiple component prevention programs: Design and analysis strategies. In K. Bryant, M. Windle, & S. West (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research*. Washington, DC: American Psychological Association.

Health Behavior Maintenance

Lisa Auster-Gussman¹, Rachel J. Burns²,
and Alexander J. Rothman¹

¹*Department of Psychology, University of Minnesota, Minneapolis, MN, USA*

²*Douglas Mental Health University Institute, McGill University, Montreal, QC, Canada*

Importance of Behavior Maintenance to Health Outcomes

In the United States, and increasingly around the world, chronic diseases have become the most common cause of death. If healthy patterns of behavior are sustained, the onset of some conditions may be prevented or delayed, and the severity of many of these conditions may be reduced (van Dam, Li, Spiegelman, Franco, & Hu, 2008). For example, compared with continuing smokers, the risk of mortality caused by coronary heart disease is reduced by half after 1 year of abstinence from smoking and drops to a level similar to those who have never smoked after 15 years of abstinence (U.S. Department of Health and Human Services, 1990). However, these health gains are reduced or lost if an individual relapses. Although behavior maintenance is important, maintenance is a challenging phase of the behavior change process, and individuals who are able to change their behaviors often have difficulty sustaining these changes over time.

Describing Behavior Maintenance

Regardless of the specific behavior, maintenance requires sustaining a behavior, but some key aspects of behavior maintenance can vary across a number of dimensions including the number, frequency, time interval, regularity, and consistency of the behavior (Rothman, Baldwin, Hertel, & Fuglestad, 2004). At its core, behavior maintenance arises when the results of a cost–benefit analysis of experienced consequences of a behavior lead that behavior to be sustained over time (Rothman, 2000). Yet, what an individual has to do to maintain a behavior over time can vary dramatically. For instance, health behaviors differ in the number of times they need to be repeated. In some domains, a behavior needs to be repeated only a few times,

whereas in other domains maintenance requires a large number of repetitions. For example, maintenance in the context of human papillomavirus (HPV) vaccination involves attending three vaccination appointments, whereas maintaining a regimen of insulin injections involves administering thousands of shots over time in order to maintain one's health. Health behaviors also differ in how frequently they must be repeated over a given time interval. In the context of breast cancer screening, maintenance may involve getting a mammogram once per year, whereas in the context of kidney disease, dialysis typically involves attending three sessions per week. Health behaviors also differ in the regularity of their timing. Taking a medication is a single behavior that likely occurs at a regular time interval, whereas safe sexual practices, such as condom use, occur on a more variable basis and potentially under differing circumstances. Finally, health behaviors differ in their consistency. Maintaining a healthful pattern of eating likely requires a diverse array of behaviors such as avoiding unhealthy foods, eating and preparing healthy foods, and resisting tempting foods. These behaviors will occur across a variety of contexts and times, whereas regular tooth flossing involves the consistent repetition of the same behavior, often in the same context.

How Is Behavior Maintenance Operationalized in Health Behavior Models?

There are two main categories of health behavior models: process models and stage models (Rothman & Salovey, 2007). Each of these classes of models takes a different approach to operationalizing maintenance and delineating factors that facilitate and inhibit behavior maintenance.

Process Models

In process models, behavior maintenance is operationalized as a continuation of the processes that initiated a given behavior (e.g. theory of planned behavior [Ajzen, 1991]; health belief model [Janz & Becker, 1984]). Process models identify variables that predict a behavior, such as intentions for that behavior, attitude toward the behavior, or past behavior. These variables are then combined into an equation whereby individuals' scores on the predictor variables determine their position a continuum of behavior likelihood. Individuals placed at the lowest end of these continuums are very unlikely to engage in a behavior, whereas individuals at the high end are very likely to engage in the behavior. Importantly, strengthening any one of the predictor variables moves an individual forward on the continuum. For example, having a more positive attitude about a behavior even in the absence of change in intention for that behavior would predict moving forward on the continuum, but strengthening intention and positive attitude would move someone even further along the continuum. Of note, each theory specifies only one prediction equation. Therefore, the model that is hypothesized to predict an initial pattern of behavior is the same as the model that is hypothesized to predict a later, maintained, pattern of behavior.

Stage Models

Stage models assume that behavior change involves movement through a sequence of discrete stages. Individuals categorized as being in the initial stage often lack awareness of the

need for behavior change; they then move through a stage in which they prepare to take action, then one in which they initiate a behavior, and the final stage is often labeled behavior maintenance or resolution (e.g. transtheoretical model [TTM; Prochaska & Velicer, 1997]; precaution adoption process model [PAPM; Weinstein, 1988]). Transitions into and out of each stage are specified by a distinct set of rules that assign individuals to a given stage. Individuals move through these stages at different rates and, in some cases, may not always move forward in sequential order. In addition, setbacks can lead an individual to revert back to a previous stage. Because these models specify different prediction models for each stage transition, they offer a framework that can distinguish between factors that predict an initial change in behavior and factors that predict a later, maintained pattern of behavior. Thus, these models have the potential to explain the observation that people are able to change their behavior initially but have difficulty maintaining that behavior over time.

Factors That Influence Behavior Maintenance: A Brief Overview

Many theoretical models of health behavior regard behavior maintenance as self-reinforcing. Implicit in these models is a positive, reinforcing feedback loop in which one's beliefs facilitate the performance of a behavior, which in turn reinforces one's beliefs, which then again reinforce one's behavior. However, this premise is often at variance with the observation that people who are motivated to and able to make changes in their behavior do not always sustain these changes over time. This has led researchers to search for factors that may determine when these self-reinforcing processes succeed and when they fail. Feeling satisfied with a health behavior, perceiving a health behavior as internally motivated, and adopting a prevention-focused self-regulatory style are three factors that have been proposed to support successful maintenance. We consider each of these perspectives, in turn.

The Role of Satisfaction in Maintenance

Rothman (2000) argued that behavior is sustained as long as an individual is satisfied with a given health behavior and that the hypothesized self-reinforcing system fails, in part, when an individual is no longer satisfied with a given health behavior. When faced with the decision to maintain a pattern of behavior, individuals usually know the actions they need to take to maintain their health. Therefore, when grappling with maintaining a pattern of behavior, people focus their attention on the extent to which they are satisfied with the behavior itself and the outcomes it has produced (e.g., Baldwin et al., 2006; Rothman et al., 2004). Maintenance is expected to continue as long as the advantages of maintaining the behavior (e.g., losing weight or positive comments from friends about your weight loss) outweigh the disadvantages of maintaining the behavior (Rothman, 2000; Rothman et al., 2004). Because the individual has demonstrated that she/he can successfully enact the pattern of behavior, to the extent that the person remains satisfied with the new behavior, she/he will choose to sustain it. This means, in turn, that she/he will continue to reap the benefits of the new pattern of behavior. Framed this way, maintenance should be reinforced as long as one believes the benefits of the behavior outweigh the costs. Importantly, social factors, such as reference group norms, may have a significant impact on how people carry out these cost-benefit and satisfaction assessments (e.g., Baldwin, Rothman, & Jeffery, 2009).

The Application of Self-Determination Theory to Maintenance

Self-determination theory (SDT) (Deci & Ryan, 1985) provides an alternative perspective on promoting behavior maintenance by suggesting that a behavior will be sustained when an individual feels that the behavior is internally motivated and consistent with one's sense of self. Specifically, the extent to which an individual develops an autonomous motivation to perform a behavior (i.e., feels that this motivation originates from the self) and integrates engaging in the behavior into his/her self-concept is thought to be the key to maintenance of that behavior over time (Ng et al., 2012; Ryan & Deci, 2000). Approaches that focus on developing autonomous motivation have been shown to facilitate rates of behavior maintenance across a range of behavioral domains (e.g., Ng et al., 2012; Williams et al., 2006). Conceptualized in terms of self-reinforcing maintenance processes, SDT suggests that when people integrate a behavior into their self-concept, they are much more likely to engage in it, and engaging in it will, in turn, reinforce it as valuable part of their self-concept.

Importantly, this approach also emphasizes the role of the social context in supporting or impeding motivation (Ryan & Deci, 2000). SDT defines three psychological needs critical to supporting the process of internalization: autonomy, competence, and relatedness. Autonomy reflects the idea that one had agency over choosing one's action. Competence involves a person's need to feel efficacious in obtaining a desired outcome. Relatedness reflects the extent to which one feels that they have support from close or important others. Autonomous motivation is thought to be at its highest when all these needs are met, and these needs can all be facilitated or hindered by social processes. For example, autonomy may be facilitated by a healthcare provider, who acknowledges the patient's perspectives and opinions on treatments before prescribing a course of treatment. Competence could be supported by one's friends or family members acknowledging that they believe the person has the capacity to sustain a course of action over time or helping the person reframe past failures as opportunities to learn. Relatedness could be facilitated by close others being empathetic to the difficulties of behavior maintenance.

The Effect of Regulatory Focus on Maintenance

Regulatory focus theory (Higgins, 1996) proposes that promotion focus and prevention focus, two separate motivational systems, regulate how an individual engages in goal-directed behavior; individuals high in promotion focus are engaged in the eager pursuit of desired outcomes, whereas people high in prevention focus are engaged in vigilantly avoiding undesired outcomes. Because behavior maintenance typically involves vigilantly avoiding or minimizing slips or lapses, Fuglestad, Rothman, & Jeffery, (2008) have proposed that these tasks may be a particularly good fit for people who are prevention focused, rendering them better able to maintain a pattern of behavior. Consistent with this perspective, they have shown that people with high prevention focus are more likely to successfully maintain behavior changes over time and respond better after setbacks (Fuglestad et al., 2008; Fuglestad, Rothman, & Jeffery, 2013). A prevention-focused individual's behavior is reinforced as long as she/he is not experiencing negative outcomes, and the absence of negative outcomes will continue to reinforce the behavior. Regardless of someone's inclination toward being promotion or prevention focused, social environments and messages may play an important role in facilitating or inhibiting one's regulatory focus. Therefore, social environments and messages that facilitate focus on prevention may help facilitate behavior maintenance (Cesario, Higgins, & Scholer, 2008).

Emerging Directions in Behavior Maintenance Interventions and Research

Given the importance of sustaining health behavior over time, a number of interventions have sought to promote behavior maintenance. The most effective maintenance interventions employ multiple strategies. These strategies include disseminating information, changing people's attitudes, creating a supportive environment whenever possible, and providing the skills to navigate an unsupportive environment (e.g., Janssen, Gucht, van Exel, & Maes, 2013). These interventions tend to provide individuals with a range of resources such as counseling sessions with a health psychologist, goal setting strategies, and strategies that focus on identifying potential barriers to maintenance. They also help individuals develop self-regulation skills including self-monitoring, developing specific action plans, getting progress-related feedback, developing realistic outcome expectancies, and discussing their strategies with their partner or close others in order to increase social support (e.g., Janssen et al., 2013).

Although there is some evidence that in combination these strategies can bolster successful maintenance, our understanding of which strategies may be particularly effective in promoting maintenance remains limited and is a priority for future research. In particular, consideration should be given to determining whether specific strategies are more effective for specific groups of people, for particular behavioral domains, or in particular contexts given the many effects that social factors and people's environment have on their ability to maintain their health. People are implicitly or explicitly informed about appropriate behavior, including appropriate health behavior, by their peers and social environments, and they look to their social network to assess risk for a given disease, to ways of reducing that risk, or to models of whether and how to engage in health behaviors.

Technological innovations have led and will likely continue to lead to the development of new, innovative ways to promote and research behavioral maintenance. Wearable devices help people track their steps, online communities can provide support and information, and people can track their eating using applications on computer and smartphones. These technologies should help to expand the range of theory-based strategies available for effective behavior maintenance, especially considering that maintenance often requires continued focused efforts that develop and change over time. As this happens, we will also learn more about whether these strategies are effective and hone the strategies that will be most beneficial for each individual.

Author Biographies

Lisa Auster-Gussman is a doctoral student in the social psychology program at University of Minnesota. Her current research focuses on the relationship between beliefs about health conditions, such as their etiology or controllability, and social cognitive and behavioral variables including stigma, self-blame, and self-regulatory processes. Her research focuses on how these relationships affect health behavior change and maintenance.

Rachel J. Burns is a postdoctoral fellow at McGill University. She received her PhD in Psychology from the University of Minnesota. Her research lies at the intersection of social psychology and health psychology and identifies the social, cognitive, and motivational processes that shape how people make health-relevant decisions and manage their health behavior.

Alex J. Rothman is currently associate dean for Research and Graduate Programs in the College of Liberal Arts and professor in the Department of Psychology, University of Minnesota. His program of research concerns the application of social psychological theory to illness prevention and health promotion and is a synthesis of basic research on how people process and respond to health information with the development and evaluation of theory-based interventions to promote sustained changes in healthy behavior.

References

- Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, 50, 179–211. doi:10.1016/0749-5978(91)90020-T
- Baldwin, A. S., Rothman, A. J., Hertel, A. W., Linde, J. A., Jeffery, R. W., Finch, E. A., & Lando, H. A. (2006). Specifying the determinants of the initiation and maintenance of behavior change: An examination of self-efficacy, satisfaction, and smoking cessation. *Health Psychology*, 25, 626–634. doi:10.1037/0278-6133.25.5.626
- Baldwin, A. S., Rothman, A. J., & Jeffery, R. W. (2009). Satisfaction with weight loss: Examining the longitudinal covariation between people's weight-loss-related outcomes and experiences and their satisfaction. *Annals of Behavioral Medicine*, 38, 213–224. doi:10.1007/s12160-009-9148-x
- Cesario, J., Higgins, E. T., & Scholer, A. A. (2008). Regulatory fit and persuasion: Basic principles and remaining questions. *Social and Personality Psychology Compass*, 2, 444–463. doi:10.1111/j.1751-9004.2007.00055.x
- Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior*. New York, NY: Plenum Press.
- Fuglestad, P. T., Rothman, A. J., & Jeffery, R. W. (2008). Getting there and hanging on: The effect of regulatory focus on performance in smoking and weight loss interventions. *Health Psychology*, 27(3, Suppl), S260–S270. doi:10.1037/0278-6133.27.3(Suppl.).S260
- Fuglestad, P. T., Rothman, A. J., & Jeffery, R. W. (2013). The effects of regulatory focus on responding to and avoiding slips in a longitudinal study of smoking cessation. *Basic and Applied Social Psychology*, 35, 426–435.
- Higgins, E. T. (1996). The “self digest”: Self-knowledge serving self-regulatory functions. *Journal of Personality and Social Psychology*, 71, 1062–1083. doi:10.1037/0022-3514.71.6.1062
- Janssen, V., Gucht, V. D., van Exel, H., & Maes, S. (2013). Beyond resolutions? A randomized controlled trial of a self-regulation lifestyle programme for post-cardiac rehabilitation patients. *European Journal of Preventive Cardiology*, 20, 431–441. doi:10.1177/2047487312441728
- Janz, N. K., & Becker, M. H. (1984). The health belief model: A decade later. *Health Education & Behavior*, 11, 1–47. doi:10.1177/109019818401100101
- Ng, J. Y. Y., Ntoumanis, N., Thøgersen-Ntoumani, C., Deci, E. L., Ryan, R. M., Duda, J. L., & Williams, G. C. (2012). Self-determination theory applied to health contexts a meta-analysis. *Perspectives on Psychological Science*, 7, 325–340. doi:10.1177/1745691612447309
- Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion*, 12, 38–48. doi:10.4278/0890-1171-12.1.38
- Rothman, A. J. (2000). Toward a theory-based analysis of behavioral maintenance. *Health Psychology*, 19(1, Suppl), 64–69. doi:10.1037/0278-6133.19.Suppl1.64
- Rothman, A. J., Baldwin, A. S., Hertel, A. W., & Fuglestad, P. T. (2004). Self-regulation and behavior change: Disentangling behavioral initiation and behavioral maintenance. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation* (pp. 130–148). New York, NY: Guilford Press.
- Rothman, A. J., & Salovey, P. (2007). The reciprocal relation between principles and practice: Social psychology and health behavior. In A. Kruglanski & E. T. Higgins (Eds.), *Social psychology: Handbook of basic principles* (2nd ed., pp. 826–849). New York, NY: Guilford Press.

- Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist*, 55, 68–78. doi:10.1037/0003-066X.55.1.68
- U.S. Department of Health and Human Services. (1990). *The health benefits of smoking cessation*. Washington, DC: Department of Health and Human Services.
- van Dam, R. M., Li, T., Spiegelman, D., Franco, O. H., & Hu, F. B. (2008). Combined impact of life-style factors on mortality: Prospective cohort study in US women. *BMJ*, 337, a1440. doi:10.1136/bmj.a1440
- Weinstein, N. D. (1988). The precaution adoption process. *Health Psychology*, 7, 355–386. doi:10.1037/0278-6133.7.4.355
- Williams, G. C., McGregor, H. A., Sharp, D., Levesque, C., Kouides, R. W., Ryan, R. M., & Deci, E. L. (2006). Testing a self-determination theory intervention for motivating tobacco cessation: Supporting autonomy and competence in a clinical trial. *Health Psychology*, 25, 91–101. doi:10.1037/0278-6133.25.1.91

Suggested Reading

- Baldwin, A. S., Rothman, A. J., Hertel, A. W., Linde, J. A., Jeffery, R. W., Finch, E. A., & Lando, H. A. (2006). Specifying the determinants of the initiation and maintenance of behavior change: An examination of self-efficacy, satisfaction, and smoking cessation. *Health Psychology*, 25, 626–634. doi:10.1037/0278-6133.25.5.626
- Ng, J. Y. Y., Ntoumanis, N., Thøgersen-Ntoumani, C., Deci, E. L., Ryan, R. M., Duda, J. L., & Williams, G. C. (2012). Self-determination theory applied to health contexts a meta-analysis. *Perspectives on Psychological Science*, 7, 325–340. doi:10.1177/1745691612447309
- Rothman, A. J. (2000). Toward a theory-based analysis of behavioral maintenance. *Health Psychology*, 19(1, Suppl), 64–69. doi:10.1037/0278-6133.19.Suppl.64
- Rothman, A. J., Baldwin, A. S., Hertel, A. W., & Fuglestad, P. T. (2004). Self-regulation and behavior change: Disentangling behavioral initiation and behavioral maintenance. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation* (pp. 130–148). New York, NY: Guilford Press.

The Health Belief Model

Edward C. Green¹, Elaine M. Murphy²,
and Kristina Gryboski³

¹*Department of Anthropology, The George Washington University, Washington, DC, USA*

²*Population Reference Bureau, Washington, DC, USA*

³*Project Hope, Millwood, VA, USA*

History of the Health Belief Model

Behavioral scientists working for the US Public Health Service developed the health belief model in the 1950s, and it remains today one of the most widely applied conceptual frameworks of health behavior used by health educators, other health professionals, and psychologists (Rosenstock, Strecher, & Becker, 1988). At the time there were medical diagnostic tools such as chest X-rays for tuberculosis (TB) screening that were underused because many people with TB did not recognize their symptoms and did not seek medical care for what they deemed a mere cough. There was a parallel need to increase use of preventive services such as immunization and medical adherence in general, in addition to health screening. At the beginning, the health belief model was rooted in information giving to increase people's awareness of and concern about the serious health risks associated with certain preventable illnesses, including illnesses that could be cured if caught early enough. Health educators also wanted people to understand that they could reduce these health risks by taking certain actions. The (primarily) psychologists theorized that people are afraid of getting serious illnesses and that health-related behaviors reflect both a person's level of fear of perceived health threats and the expected fear-reduction potential of taking a recommended action. People consider whether the outcomes of the behavior change outweigh its practical and psychological obstacles. In short, individuals assess the net benefits of changing their behavior to reduce the threat to their health and decide whether to act (Murphy, 2005).

The model identifies and organizes interventions around four aspects of this assessment: perceived susceptibility to ill health, or risk perception, perceived severity of ill health, perceived benefits of behavior change, and perceived barriers to taking action. Later, health belief theorists added the concept of self-efficacy as a factor in health behavior decision making. Individuals' perceptions of risks, benefits, and obstacles add up to their readiness to act or lack

of readiness. If a person is ready to change behavior to obtain the perceived benefits, health promotion messages—through mass media, peer education, and other interventions—act as cues to action, transforming readiness into overt behavior. These cues are particularly important when unhealthy behaviors are habitual such as not wearing seat belts, overeating, not exercising, or smoking. The health belief model can also help identify leverage points for change. A smoker who may not think he is capable of quitting on his own—that is, who has low self-efficacy—can be coached on proven ways to quit and encouraged to enroll in a supportive smoking cessation program (Rimer & Glanz, 2005).

The health belief model of behavioral change was later categorized as *individual centered*. In addition to the health belief model, the category includes social cognitive theory (in spite of the word “social,” it focuses on individual learning through observation), the theory of reasoned action, the stages of change model, and the AIDS risk reduction model. Although they differ in several ways, all these theories focus on the individual’s perceived susceptibility to a threat to health and benefits that would result from changing behavior, as well as on constraints to such change. They assume that people are rational and will do the right thing once they are provided adequate information and understand that change is in their personal self-interest. The model would be effective unless a person is neither concerned about nor afraid of negative health consequences, or is simply not rational, or not currently in a rational frame of mind (e.g., when intoxicated).

Behavioral researchers came to realize that complex health behaviors such as those involving sexual intercourse or addiction take place in a dynamic (i.e., reciprocally influential) social and cultural context, and an individual’s thinking and decision making reflect this context (Sweat & Denison, 1995). Therefore, social and cultural factors surrounding and going beyond the individual must be considered in designing preventive interventions. A second group of theories takes both individual and social factors into account. This group includes diffusion of innovations theory, social influence or social inoculation model, social network theory, and theory of gender and power (Green, 2003).

Key Constructs in the Health Belief Model

The health belief model was originally based on four cognitive constructs:

- Perceived susceptibility (individual’s assessment of the risk of becoming ill, that is, realizing an unwanted outcome).
- Perceived severity (individual’s assessment of the seriousness of the illness, condition, or unwanted outcome and the potential consequences).
- Perceived barriers (individual’s assessment of the influences that discourage adoption of the promoted action or new behavior).
- Perceived benefits (individual’s assessment of the positive consequences of adopting the promoted action).

These constructs can be expanded to apply to specific areas of behavioral change. To use the example of HIV infection, the thinking might proceed thus:

- 1 Perceived susceptibility to a particular health problem: Am I at risk for HIV? What behaviors put me at risk—having (unprotected) sex with multiple partners? Intravenous drug use?
- 2 Perceived severity of the condition: How serious is AIDS? How hard would my life be if I were infected? Who would take care of me? Could I afford medication?

- 3 Belief in effectiveness of the new behavior: Are condoms effective against HIV transmission? Would reducing the number of sexual partners reduce my risk? Would using clean needles reduce my risk?
- 4 Cues to action: I am hearing frequent radio messages about AIDS and how to avoid it. I notice that free condoms are available at in men's rooms. I watched AIDS weaken the health of a family member. I went to the funeral of a close friend who died of AIDS.
- 5 Perceived benefits of preventive action: If I start using condoms, or become monogamous, I can avoid HIV infection. The needle exchange clinic is a long distance, but clean needles will prevent injecting HIV into my bloodstream.
- 6 Barriers to taking action: I do not like using condoms, and I cannot afford them anyway. Having several sex partners at the same time gives me the variety I like and makes me feel like a real man (UNAIDS, 1999).

As noted earlier in the example of smoking cessation, the health belief model has also been adapted to include the construct of self-efficacy, the belief that one is capable of carrying out the behavior. Consequently, it is sometimes referred to as the expanded health belief model (Strecher & Rosenstock, 1997). Using the expanded model, a seventh question would be added for the AIDS example above: Am I capable of convincing my sexual partners to use condoms every time?

Recognizing that an individual's behavior is affected by a wider context, many organizations implementing public health promotion and prevention strategies employ a multilevel framework often referred to as the social ecological model that includes factors beyond the individual level. For example, UNAIDS researchers concluded that prevention has to operate at four levels: superstructural, structural, environmental, and individual. A superstructural factor might be an international tariff that makes imported condoms too expensive for sexually active youth living in countries where condoms are not manufactured locally. Policies that exclude unmarried youth from receiving condoms from public health clinics or addicts of any age from receiving sterile syringes represent a structural barrier. Environmental influences could include what one's peer group is saying and doing about AIDS prevention, as well as cultural norms and values transmitted through family and religious teachings. Individual factors beyond health risk and benefit analysis include whether a person is a risk taker or timid by nature and whether the person tends to be a leader or a follower.

Predicting Behavior With the Health Belief Model

A review of the effectiveness of the health belief model to predict and explain behavior found that perceived severity was a weak predictor of behavior, perceived susceptibility was not a predictor in most studies, and perceived barriers and perceived benefits were consistently the strongest predictors of behavior (Carpenter, 2010). While the health belief model is still widely used to guide intervention design, a recent meta-analysis concluded there is insufficient evidence to attribute the use of the model in health interventions to effectiveness in changing behaviors (Jones, Smith, & Llewellyn, 2014). The authors report that while the studies they reviewed state that they used the health belief model, only 6 of the 18 studies followed the full model, and only 5 measured model constructs before and after the intervention. Increasingly, behavior change interventions do not rely on a single theory or model. Recognizing that behavior is influenced by many factors, behavior change strategies draw on several theories simultaneously. To advance knowledge about effective behavior change strategies, further

studies should be done to clearly demonstrate how the intervention design is informed by the theories and to measure the theoretical constructs before and after the intervention, as well as the associated changes in behavior.

Author Biographies

Edward C. Green, PhD, is a medical anthropologist who served as senior research scientist at the Harvard Center for Population and Development Studies. In retirement, he is currently a research professor at the Department of Anthropology, the George Washington University, and director of the New Paradigm Fund.

Elaine M. Murphy, PhD, is a visiting scholar at the Population Reference Bureau in Washington, DC, and an international health consultant. She has worked for 30 years in the areas of population and reproductive health (family planning).

Dr. Kristina Gryboski is a medical anthropologist who has worked in global health for over 20 years as a researcher, program manager, technical writer, and trainer in nutrition, maternal and child health, gender, and reproductive health.

References

- Carpenter, C. J. (2010). A meta-analysis of the effectiveness of health belief model variables in predicting behavior. *Health Communication*, 25, 661–669. doi:10.1080/10410236.2010.521906
- Green, E. C. (2003). *Rethinking AIDS prevention: Learning from successes in developing countries*. Westport, CT: Greenwood Publishing Group.
- Jones, C. J., Smith, H., & Llewellyn, C. (2014). Evaluating the effectiveness of health belief model interventions in improving adherence: A systematic review. *Health Psychology Review*, 8, 253–269. doi:10.1080/17437199.2013.802623
- Murphy, E. M. (2005). *Promoting healthy behavior*. Washington, DC: Population Reference Bureau.
- Rimer, B. K., & Glanz, K. (2005). *Theory at a glance: A guide for health promotion practice* (2nd ed.). Washington, DC: National Cancer Institute.
- Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1988). Social learning theory and the health belief model. *Health Education & Behavior*, 15(2), 175–183. doi:10.1177/109019818801500203
- Strecher, V., & Rosenstock, I. (1997). The health belief model. In K. Glanz, F. M. Lewis, & B. K. Rimer (Eds.), *Health behavior and health education: Theory, research, and practice* (pp. 41–58). San Francisco, CA: Jossey-Bass.
- Sweat, M. D., & Denison, J. A. (1995). Reducing HIV incidence in developing countries with structural and environmental interventions. *AIDS*, 9, S251–S257.
- UNAIDS. (1999). *Sexual behaviour change for HIV: Where have theories taken us?* Geneva: Author.

Suggested Reading

- Family Health International/UNAIDS. (2001). *Effective strategies in low HIV prevalence countries*. Geneva: UNAIDS.
- Gryboski, K., Yinger, N. V., Dios, R. H., Worley, H., & Fikree, F. F. (2015). Working with the community for improved health. *Health Bulletin*, 3, 27.
- Obregon, R., & Waisbord, S. (Eds.) (2012). *The handbook of global health communication* (Vol. 29). West Sussex, UK: Wiley.

Health Consequences and Correlates of Life Stories

William L. Dunlop, Tara P. McCoy,
and Grace E. Hanley

Psychology Department, University of California, Riverside, Riverside, CA, USA

*I'm in a chain of memories
It's my will*

John Frusicante (2001)

The life story is somewhat of a curious construct. Most everyone has some idea what a life story *is*, yet few in the field of health psychology actively study it. “Why should they?” the reader may reasonably ask. “There already exist countless more tractable psychological constructs that have proven themselves to be robust indicators – as well as determinants – of physical and psychological health.” Fair point. To this we would respond that a consideration of life stories is only necessary if researchers wish to attain a deeper, more thorough, and psychologically rich understanding of the persons they aim to study (Dunlop, 2015). It is also worth noting that there exists a growing literature recognizing the *unique* predictive ability of aspects of life stories, exhibited in relation to health behaviors and outcomes (e.g., Adler et al., 2015; Dunlop & Tracy, 2013; Dunlop, Walker, & Wiens, 2013).

Before offering a summary of previous research examining life stories in relation to physical and psychological health, it is necessary to provide a more thorough discussion of what exactly a life story is and what it is not. Following this summary, we will provide an overview of previous research that has considered aspects of life stories in relation to health-related outcomes. Although we do not anticipate that this entry will convert each and every reader into a life story “believer,” we do hope that it increases the level of interest among health psychologists in the life story.

The Time of Our Lives

Lives are complicated things. Just when people feel as though they finally have a handle on all of their various tasks, responsibilities, roles, and requirements, something changes. Something *always* changes. Indeed, this fact, the fact that lives are constantly in flux, gets to the very heart of the matter: In large part, lives are complicated because they are always morphing. Can people ever claim to be living the same life they were living 5 years or even 5 months ago? The nature of modern circumstances, as well as common sense, would seem to dictate otherwise.

Yet, despite all the rampant change, people continually lay claim to the fact that they are, numerically speaking, the same person they were in 2005, 1995, and so on (age permitting). This claim of “personal persistence” (Chandler, Lalonde, Sokol, Hallett, & Marcia, 2003) is larger than any one life that falls under its purview. Indeed, the very moral, social, and economic bedrocks of civilization are predicated on the notion that people take ownership of their past and have some invested interest in the manner in which their lives play out in the future (Chandler et al., 2003). In the absence of such a temporal thread, “contracts and debts and promises would all fly out the same window, all prospects for a just and moral world would evaporate, and judgment day would simply go out of business” (Chandler et al., 2003, pp. 7–8).

The life story represents an important psychological resource because it can be used to justify, to the self and others, a connection between the many different temporal “versions” of the self as well as the varied experiences of which it has been a part—that is, it can be used to create a sense of personal persistence or temporal continuity (Chandler et al., 2003; Dunlop & Walker, 2014). The “active ingredient” in this process is autobiographical reasoning (Habermas & Bluck, 2000).

Autobiographical Reasoning: A Primer

Readers looking for some reprieve from the nebulous nature of the life story via autobiographical reasoning will be disappointed. Autobiographical reasoning is just as, if not more, complex than the life stories in which it manifests: this form of reasoning actually is not one thing, but four (at least; Habermas & Bluck, 2000). First, a given life story must make “sense” within a particular social and cultural context; it must accord to the *cultural concept of biography* (Habermas, 2007). This concept includes tacit assumptions regarding the events that are “worthy” of one’s life story (e.g., graduating from college, having a child; Habermas, 2007) as well as the manner in which these specific events, and the life story as a whole, should be told.

Second, one may consider the *temporal coherence* of the life story in question. Some stories contain sufficient detail regarding when a particular experience occurred, as well as a logical (most frequently temporal) progression between the various events that the narrator deems self-defining. When these features are present, the story is said to have a high level of temporal coherence. Third, the *thematic coherence* of life stories is noteworthy. As Habermas and Bluck (2000) observed, narratives have a tendency to espouse certain themes or maxims in the stories they construct. Individuals differ in the degree to which the themes in their stories are consistent and coherent. Finally, life stories vary in their degree of *causal coherence*. Narrators differ with respect to the degree to which they associate changes in the self with accumulations of experience (e.g., “summer school made me more studious”) as well as the accumulation of experiences with properties of the self (e.g., “I was quite shy at the time, so I decided to study by myself in the library”).

Where the Rubber Meets the Road: Measuring Life Stories

How do researchers assess life stories? Before answering this question, it is important to recognize the distinction between the life story as a psychological construct and the life story as a *measurable* psychological construct. One intriguing feature of autobiographical stories is that they are, invariably, “created within a specific situation, by particular individuals, for particular audiences, and to fulfill particular goals” (McLean, Pasupathi, & Pals, 2007, p. 262). In this sense, the elements of life stories that are produced in research contexts do not represent a pure “assessment of internal representations but rather...an emergent product of representations and features in which narratives are told” (p. 264; see also McCoy & Dunlop, 2016).

When researchers set their sights on measuring life stories, they most commonly ask participants to provide a series of key autobiographical scenes from their participants’ lives, such as “high points,” “low points,” and “turning points” (see McCoy & Dunlop, 2016). An example prompt is as follows (McAdams, 2008):

Please describe a scene, episode, or moment in your life story that stands out as an especially positive experience. This might be the high point scene of your entire life, or else an especially happy, joyous, exciting, or wonderful moment in the story. Please describe this high point scene in detail. What happened, when and where, who was involved, and what were you thinking and feeling? Also, please say a word or two about why you think this particular moment was so good and what the scene may say about who you are as a person and your life.

Several aspects of this prompt are worth noting. First, individuals are pushed for a single, definitive event rather than a general period in their lives (see also Baranski, Morse, & Dunlop, 2016). Second, as much detail as possible is requested. Together, these two elements of prompts (specificity and detail) have proven to offer the most psychologically meaningful narratives.

From our experience, we believe that autobiographical narratives represent some of the most interesting and illustrative data that can be extracted from participants. Consider, for example, the following narrative, which was produced by an undergraduate at our institution (University of California, Riverside) during a recent study conducted in our lab:

On [date], I got a tattoo – a compass on my chest. This was obviously a big deal for me, and it remains my pride and joy. My girlfriend and her mother were there with me when I got it, but my parents, who I’m generally close to, still don’t know about it. I got the tattoo because I felt something missing from my body, like the flesh on my chest where the tattoo was supposed to be was just missing. I could feel the loss and see a lighter part of my skin where there should have been ink. When I got the tattoo, it felt right. My body was complete again. With such a positive change of who I am as a person (and I do think that my tattoo helps to define me), it feels weird not to tell my parents. I’m not afraid they’ll be mad at me (which they will) but that they’ll be mad at themselves. They don’t understand that tattoos aren’t just for sailors, gangsters, and convicts and they’ll blame themselves for not raising me properly. I can hear my mom saying, “What would your grandfather say?” I know that they’ll find out about it someday, and I am prepared to take care of them when they do. I don’t want to hurt them with what I have to hide.

Perhaps we could have gotten much of the same information about this particular participant if we had administered questionnaires pertaining to adult attachment, relations with his or her parents, and family environment. Perhaps not. In any case, we contend that considering participants’ narratives serves as a stark reminder that we are studying people—real, living, and breathing people—rather than the cold variables that, as academic psychologists, we tend to focus on.

Researchers have collected autobiographical narratives in a variety of ways. Some, for example, have collected participants' oral narratives via semi-structured interviews (e.g., Dunlop & Walker, 2014; McAdams, 2008), whereas others have collected participants' written responses via paper-and-pencil assessment packages or computer-mediated programs (e.g., Dunlop et al., 2013; McAdams et al., 2006). Despite the current focus on the contextualized, or situated, nature of stories (e.g., McLean et al., 2007), until recently researchers have generally assumed that the narrative material gathered via these various assessment procedures was comparable. Our research group (McCoy & Dunlop, 2016) has just completed a study that challenges this assumption.

In our study, approximately 400 participants were asked to provide 3 autobiographical key scenes from within 1 of 4 assessment contexts, constituting a 2(interviewer present, interviewer absent) $\times$ 2(written responses, spoken responses) study design. The resulting narratives were quantified in terms of 15 linguistic (i.e., word based) categories and 6 more conceptual themes (the specific categories and themes are irrelevant at this time, though they will be explored in detail later in this entry). Of the 15 linguistic categories considered, 14 exhibited a significant main effect for, or interaction with, assessment context. A similar pattern was observed when we considered the conceptual content of participants' narratives. Here, five of the six categories differed significantly across assessment contexts. The take-home message from this research is that assessment contexts substantially influence the content of autobiographical narratives.

Where the Rubber Really Meets the Road: Quantifying Narratives

Unlike other personality characteristics, notably personality traits, there exists no single framework or conceptual paradigm that the majority of narrative researchers endorse. In contrast, it is not uncommon for researchers to create coding systems for various constructs embedded within narratives on the basis of research questions or the nature of the data in question (e.g., Dunlop et al., 2013). Despite this idiosyncrasy, the following three coding systems have developed considerable traction within the field.

Agency and Communion

Agency, represented by themes such as independence and self-assertion, and communion, represented by themes such as togetherness and social unity. What this really means is that the understanding of many topics, including autobiographical narratives, has been aided through the consideration of the fundamental modality represented by conflicting desires to "get ahead" (agency) and "get along" (communion; Hogan, 1982).

Redemption and Contamination

Earlier we noted that life stories and autobiographical narratives need be considered in relation to the social and cultural contexts in which they manifest. A related point is that certain forms of narratives are generally supported by certain cultures, relative to others. Within American contexts, the redemptive story represents one such supported, or master, narrative (McAdams, 2006). A redemptive story manifests when a bad beginning (e.g., getting dumped by a girlfriend) leads to a positive ending (e.g., finding a more compatible match). The inverse of redemption is contamination, which manifests when promising beginnings give way to spoiled endings.

Transformational Processing

Autobiographical narratives have also been noted to differ in terms of affective tone (e.g., positive valence, negative valence) and complexity (e.g., the degree to which the narrator recognizes, and integrates, multiple perspectives in his or her story; see McAdams et al., 2004). Together, these variables constitute the transformational processing framework.

Health Consequences of Life Stories

Life stories represent more than mere conceptions of the past. Some theorists, such as Sarbin (2004), have argued that, once formed, there exists a push to keep the particular story going. This reasoning is suggestive of the possibility that adopting the “right” type of story may actually be associated with certain health trajectories. Recognizing the gravity of this theorizing, our research group (Dunlop & Tracy, 2013) sought to examine differing autobiographical narratives in relation to subsequent health trajectories. We did this by studying recently sober alcoholics (i.e., those sober 6 months or less) enrolled in Alcoholics Anonymous (AA). AA represents a particularly interesting group for narrative psychologists, as stories are central to this recovery program (O’Reilly, 1997). Indeed, as sociological researchers have noted, in AA there exists the expectation that recovery is aided through the adoption of an appropriate narrative, in this case a redemptive narrative.

In our study, we prompted recovering alcoholics for stories of their most recent drink and then distinguished those narratives that contained redemptive imagery from those narratives that did not. Of note, participants who produced redemptive narratives were initially otherwise demographically, psychologically, and health-wise indistinguishable from non-redemptive participants. Over time, however, the redemptive group was much more likely to maintain their sobriety relative to the non-redemptive group. In addition, redemptive participants increased in their self-reported health over time. The sum total of these results remained significant after controlling for the “usual suspects” in addictions research, such as attribution style, optimism, and depression. Far from being merely reflective, the elements of our life stories exhibit heavy bearing on the lives we come to lead (Sarbin, 2004).

The research summarized above represents but the tip of the iceberg. A sizable literature notes a strong relationship between the various conceptual elements of key autobiographical scenes and various markers of physical and psychological health (e.g., Adler, 2012; Dunlop et al., 2013). Among these various research pursuits, the work of Adler (2012; Adler, Turner, et al., 2015; Adler, Lodi-Smith, Philippe, & Houle, 2015) is particular noteworthy. For example, he has shown that changes in the agency of participants’ narratives predict subsequent changes in self-reported health over the course of psychotherapy (Adler, 2012). More recently, Adler, Turner, et al. (2015) have also shown that various aspects of narratives (e.g., redemption) predict subsequent health trajectories over a span of 4 years.

In addition to his empirical contributions in this area, Adler, Lodi-Smith, et al. (2015) has recently published a thorough literature review documenting both the typical themes or aspect narratives researchers consider (the three conceptual systems noted earlier are present on this list) and the numerous studies that have shown that the relationship between aspects of key autobiographical scenes remains significant predictors of psychological health and well-being even after controlling for certain demographic and personological variables. To take one study from this review, Adler, Kissel, and McAdams (2006) noted that the tendency to construct contaminated key autobiographical scenes corresponded negatively with well-being. This

relationship remained significant after controlling for participants' degree of neuroticism. The conclusion to be taken from this illustrative review is that life stories and key scenes hold considerable incremental validity within the realm of psychological adjustment, health, and well-being.

Collectively, the work of our research group (e.g., Dunlop et al., 2013; Dunlop & Tracy, 2013), Adler's research group (Adler, 2012; Adler, Turner, et al., 2015; Adler, Lodi-Smith, et al., 2015), and others (McAdams, 2006) leads to the following conclusions: (a) aspects of life stories often correspond with participants' current levels of physical and psychological health (e.g., McAdams, 2006), (b) changes in the elements of life stories predict changes in psychological health (e.g., Adler, 2012), (c) life stories serve as powerful predictors of subsequent health trajectories (e.g., Adler, Turner, et al., 2015; Dunlop & Tracy, 2013), and (d) both concurrently and longitudinally, life stories have proved themselves to be independent predictors of the aforementioned outcomes, even after controlling for a host of non-narrative psychological and health-based variables (e.g., Adler, Lodi-Smith, et al., 2015; Dunlop & Tracy, 2013).

Conclusion

Life stories matter. For health psychologists, life stories should matter more. These stories have proven themselves to be significant predictors of a host of health-related outcomes and processes. These stories have also been recognized as determinants of subsequent development trajectories and our own "character and personality style" (Sarbin, 2004, p. 7). The implications this construct holds for health psychology, thus, are self-evident.

Author Biographies

William L. Dunlop is an assistant professor of social/personality psychology at the University of California, Riverside. He uses idiographic, nomothetic, and idiographic-nomothetic approaches to examine psychological differences between people, as well as to understand the nature and development of individual lives.

Tara P. McCoy is a graduate student at the University of California, Riverside. She studies the relation between self-defining autobiographical narratives and social contexts. Her most recent work has explored the influence assessment contexts yield on individuals' narratives.

Grace E. Hanley is a doctoral graduate student of social/personality psychology at the University of California, Riverside. Her broad interests include life narratives and self-continuity. She is currently studying the domain specificity on life narratives in relation to certain life outcomes.

References

- Adler, J. M. (2012). Living into the story: Agency and coherence in a longitudinal study of narrative identity development and mental health over the course of psychotherapy. *Journal of Personality and Social Psychology*, 102, 367–389. doi:10.1037/a0025289
- Adler, J. M., Kissel, E. C., & McAdams, D. P. (2006). Emerging from the CAVE: Attributional style and the narrative study of identity in midlife adults. *Cognitive Therapy and Research*, 30, 39–51. doi:10.1007/s10608-006-9005-1

- Adler, J. M., Lodi-Smith, J., Philippe, F. L., & Houle, I. (2015). The incremental validity of narrative identity in predicting well-being: a review of the field and recommendations for the future. *Personality and Social Psychology Review (online ahead of print)*, 1–32. doi:10.1037/a0038601
- Adler, J. M., Turner, A. F., Brookshier, K. M., Monahan, C., Walder-Biesanz, I., Harmeling, L. H., ... Oltmanns, T. F. (2015). Variation in narrative identity is associated with trajectories of mental health over several years. *Journal of Personality and Social Psychology*, 108, 476–496. doi:10.1037/a0038601
- Baranski, E., Morse, P., & Dunlop, W. L. (2016). Lay-conceptions of volitional personality change: From strategies pursued to stories told. *Journal of Personality*, 85(3), 285–299.
- Chandler, M. J., Lalonde, C. E., Sokol, B. W., Hallett, D., & Marcia, J. E. (2003). Personal persistence, identity development, and suicide: A study of Native and Non-native North American adolescents. *Monographs of the Society for Research in Child Development*, 68, 1–138.
- Dunlop, W. L. (2015). Contextualized personality, beyond traits. *European Journal of Personality*, 29, 310–325. doi:10.1002/per.1995
- Dunlop, W. L., & Tracy, J. L. (2013). Sobering stories: Narratives of self-redemption predict behavioral change and improved health among recovering alcoholics. *Journal of Personality and Social Psychology*, 104, 576–590. doi:10.1037/a0031185
- Dunlop, W. L., & Walker, L. J. (2014). Cross-cultural variability in self-continuity warranting strategies. *Journal of Language and Social Psychology*, 34, 300–315. doi:10.1177/0261927X14555873
- Dunlop, W. L., Walker, L. J., & Wiens, T. K. (2013). What do we know when we know a person across contexts? Examining self-concept differentiation at the three levels of personality. *Journal of Personality*, 81(4), 376–389.
- Frusicante, J. (2001). Going inside. On *To record only water for 10 days* [CD]. Burbank, CA: Warner Bros., Records.
- Habermas, T. (2007). How to tell a life: The development of the cultural concept of biography. *Journal of Cognition and Development*, 8, 1–31. doi:10.1080/15248370709336991
- Habermas, T., & Bluck, S. (2000). Getting a life: The emergence of the life story in adolescence. *Psychological Bulletin*, 126, 748–769. doi:10.1037/0033-2909.126.5.748
- Hogan, R. (1982). A socioanalytic theory of personality. In M. Page (Ed.), *Nebraska symposium on motivation* (Vol. 30, pp. 55–89). Lincoln: University of Nebraska Press.
- McAdams, D. P. (2006). The redemptive self: Generativity and the stories Americans live by. *Research in Human Development*, 3, 81–100. doi:10.1080/15427609.2006.9683363
- McAdams, D. P. (2008). *The life story interview*. (Unpublished manuscript). Evanston, IL: Northwestern University.
- McAdams, D. P., Anyidoho, N. A., Brown, C., Huang, Y. T., Kaplan, B., & Machado, M. A. (2004). Traits and stories: Links between dispositional and narrative features of personality. *Journal of Personality*, 72, 761–784. doi:10.1111/j.0022-3506.2004.00279.x
- McAdams, D. P., Bauer, J. J., Sakaeda, A. R., Anyidoho, N. A., Machado, M. A., Magrino-Failla, K., ... & Pals, J. L. (2006). Continuity and change in the life story: A longitudinal study of autobiographical memories in emerging adulthood. *Journal of Personality*, 74(5), 1371–1400.
- McCoy, T. P., & Dunlop, W. L. (2016). Contextualizing narrative identity: A consideration of assessment settings. *Journal of Research in Personality*, 65, 16–21.
- McLean, K. C., Pasupathi, M., & Pals, J. L. (2007). Selves creating stories creating selves: A process model of self-development. *Personality and Social Psychology Review*, 11, 262–278. doi:10.1177/1088868307301034
- O'Reilly, E. B. (1997). *Sobering tales: Narratives of alcoholism and recovery*. Amherst: University of Massachusetts Press.
- Sarbin, T. R. (2004). The role of imagination in narrative construction. In C. Daiute & C. Lightfoot (Eds.), *Narrative analysis: Studying the development of individuals in society* (pp. 5–20). Thousand Oaks, CA: Sage Publications.

Suggested Reading

- Adler, J. M. (2012). Living into the story: Agency and coherence in a longitudinal study of narrative identity development and mental health over the course of psychotherapy. *Journal of Personality and Social Psychology, 102*, 367–389. doi:10.1037/a0025289
- Dunlop, W. L., & Tracy, J. L. (2013). Sobering stories: Narratives of self-redemption predict behavioral change and improved health among recovering alcoholics. *Journal of Personality and Social Psychology, 104*, 576–590. doi:10.1037/a0031185
- McAdams, D. P. (2006). The redemptive self: Generativity and the stories Americans live by. *Research in Human Development, 3*, 81–100. doi:10.1080/15427609.2006.9683363
- McLean, K. C., Pasupathi, M., & Pals, J. L. (2007). Selves creating stories creating selves: A process model of self-development. *Personality and Social Psychology Review, 11*, 262–278. doi:10.1177/1088868307301034

Health Consequences and Correlates of Social Justice

Todd Lucas

C. S. Mott Endowed Professor of Public Health Associate Professor of Epidemiology and Biostatistics Division of Public Health College of Human Medicine Michigan State University, Wayne State University, Detroit, MI, USA

In 1999, the US congress directed the Institute of Medicine to examine the extent to which racial and ethnic minorities receive disparate health services, to explore factors that contribute to these inequities, and to recommend policies and practices to eradicate them. The resulting report, *Unequal Treatment: Confronting Racial and Ethnic Health Disparities in Health Care* (Nelson, Smedley, & Stith, 2002), became an authoritative source for documenting the existence of racial and ethnic healthcare disparities in the United States. Perhaps the most important contribution of *Unequal Treatment* is that it identified and organized a substantial body of evidence to support that significant differences exist in the delivery of healthcare between racial and ethnic minorities and nonminorities in the United States and that these disparities remain even when differences in income, age, insurance status, and illness severity are accounted for. For example, *Unequal Treatment* showed that when minorities in the United States are ill, they are less likely to receive desirable medical procedures such as bypass surgery and organ transplants, and they are more likely to receive less desirable procedures such as lower limb amputations. *Unequal Treatment* thus revealed to clinicians and policy makers that death rates from illnesses such as heart disease and cancer are significantly higher among racial and ethnic minorities due in no trivial part to disparities in healthcare delivery.

In broader terms, *Unequal Treatment* underscores that the concept of social justice is understood by many as the extent to which resources are distributed fairly across a society based on factors such as race, gender, education, and income. That uneven distribution of health resources may contribute to social inequity has been increasingly appreciated and attended to in healthcare training and delivery and in accompanying research and health policy. Although resource inequity comprises a valuable perspective indeed, a less appreciated and psychologically oriented view is that social justice encompasses not only the extent to which health resources are accessible to various segments of a society but also the subjective evaluations of fairness that occur as individuals react to everyday social exchange. A psychologically oriented notion of social justice illuminates that social justice can arise from everyday contexts, environments, and occurrences, and justice-oriented cognitions and emotion can contribute to health and illness. This broader notion emphasizes four key characteristics of social justice that are fundamental to identifying social and psychological antecedents and to understanding both

the readily apparent and more subtle ways in which justice acts as social determinant of health. First, a psychologically oriented view emphasizes that social justice can be connected to health both within and outside of formal healthcare delivery settings and systems. Second, the health consequences of social justice stem not only from the extent to which health resources are equally accessible to members of a society but also from the extent to which individuals are treated fairly and respectfully during everyday social exchange. Third, the social justice framework is exceptionally well suited to considering health disparities, though social justice carries health implications for both the socially advantaged and disadvantaged members of a society. Fourth, a psychological orientation emphasizes that health and illness are affected not only through differential access to health resources and treatments but also through biological stress processes and health behaviors that contribute in fundamental ways to a social justice perspective of health.

Social Justice as Distributive Justice

Clinicians, medical scholars, and policy makers have most often viewed social justice through the lens of *distributive justice*. Generally speaking, distributive justice refers to the fairness of resource allocations and outcomes (for recent review, Jost & Kay, 2010). Within health and medical contexts, such resource allocations most readily encompass access to healthcare goods and services. This can include initial entry into a healthcare system, as well as access to effective or desired medical treatments once inside. As highlighted by distributive justice theory and research, one or more allocative principles may be used by individuals in deciding whether health resources are allocated fairly. For example, people may consider the extent to which health resources are equally versus equitably (i.e., proportionally based on one's own as well as others' social contributions) disbursed. Alternatively, people may consider whether health resources are available to those most in need. Health outcomes associated with resource distributions generally entail differences in both the desirable (e.g., cure and quality of life) and undesirable (e.g., morbidity, mortality, adverse events) outcomes that stem from differential access to health resources, and these may be similarly evaluated according to one or more allocative principles.

Emphasis on distributive justice within the social justice framework has galvanized at least two broad approaches to addressing health inequity. First, health inequity may be addressed through *social engineering*, which is rooted in modifying social and environmental factors to better ensure distributive social justice. Very often, social engineering in the context of health inequity is accomplished through legislation. For example, the Patient Protection and Affordable Care Act may be regarded as a social engineering approach to addressing distributive health inequity to the extent that it attempts to facilitate more equitable access to healthcare. Social engineering also includes attempts to alter other environmental contexts to better promote distributive justice. For example, inequities in primary prevention of illness might be addressed by altering the built environment to facilitate exercise, or to provide better access to healthy food (for review, Blacksher & Lovasi, 2012).

With an eye toward future research, a more recent perspective of health inequity that overlaps with distributive justice arises from emerging research on implicit bias among healthcare providers (for review, Penner, Blair, Albrecht, & Dovidio, 2014). This nascent literature is revealing that healthcare providers may hold subtle biases against patients based on sociodemographic characteristics, such as race or ethnicity. Importantly, these implicit biases are uncontrollable and subconscious, and they exist despite that virtually all clinicians are not

explicitly racially biased. Ultimately, implicit bias among healthcare providers may manifest as distributive social injustice when it alters clinical decision making in ways that result in health inequity. For example, and unbeknownst to the provider, implicit biases may alter the allotment of specific medical treatments to racial minorities, or they may create communication errors or feelings of mistrust during provider–patient interactions that produce inequitable health outcomes (Penner et al., 2014).

Social Justice Beyond Distributive Justice

Social justice describes not only the disbursement of benefits and burdens according to allocative principles but also the rules and norms that govern decisions affecting individuals and groups, and it ensures that people are treated with dignity and respect. Along these lines, the concept of *procedural justice* may be understood as an evaluation of the fairness of decision processes that are used to determine outcomes (for recent review, Jost & Kay, 2010). In the context of health and illness, procedural justice underscores that assessing social justice requires one not only to assess how health resources and outcomes are allocated but also to consider the rules and deliberations that determine such allocations.

A consideration of procedural justice suggests at least two additional connections between the social justice framework and health inequity. First, procedural justice is important to the extent that fair processes can ensure a fair distribution of health resources. For example, health insurers and managed care organizations are required to follow rules and adopt publicly accountable procedures in determining coverage decisions, and these processes may be vital to ensuring that members of a society have fair and adequate access to healthcare (e.g., Chan, 2002). In focusing on such *instrumental functions* of procedural justice, individuals may especially evaluate the extent to which decision processes include *voice*, or the opportunity to provide input into an allocative decision process prior to determining an outcome. Allocative processes may be similarly and simultaneously evaluated according to additional criteria, such as the extent to which decision procedures are consistent, accurate, free of bias, and correctable.

A second and more subtle way in which fair processes contribute to social justice is through the *relational function* of procedural justice. From a relational perspective, fair processes also matter to individuals because they communicate the extent to which one is seen as a valued and respected member of the larger society. For example, having voice may profoundly affect social justice even if it serves no instrumental function in a decision-making process. This is because having a voice can provide individuals with reassurance that they are respected by decision-making authorities. In similar fashion, *fair process effects* suggest that inequitable allocations of health resources and outcomes may be more palatable to members of a society if those members can be sure that allotments were at least arrived at using fair allocative procedures. In the context of healthcare delivery, the relational functions of procedural justice may be an especially crucial precursor to creating trust among healthcare recipients in the overarching healthcare system (Gilson, 2003).

In parallel to procedural justice, at least two additional directions for connecting social justice to health and illness beyond the realm of distributive justice are suggested by ongoing research. First is the closely related concept of *interactional justice*, referring to the fairness of treatment that is accorded to persons by others when implementing allocative procedures or determining outcomes (for recent review, Jost & Kay, 2010). Interactional justice consists of *informational justice*, focusing on the extent to which explanations for why certain procedures

were used or outcomes were determined, and also *interpersonal justice*, reflecting the extent to which people are treated with politeness, dignity, and respect by authorities during allocative decisions and processes. Informational justice highlights that when interacting with healthcare systems and authorities, people may or may not be given an adequate account of the health allocations and outcomes affecting them and also the accompanying deliberative rules and processes. Interpersonal justice suggests that such accounts may be delivered respectfully and politely to individuals, or else disrespectfully and impolitely.

A second and related ongoing direction considers other known psychosocial determinants of health and illness that could reflect procedural justice. For example, psychosocial concepts such as *medical mistrust*, *cultural competency*, and *perceived racism* carry important implications for the health and wellness, and they may partly reflect the extent to which individuals consider rules and interpersonal treatment to be fair. Thus, emerging literature suggests that procedural and interactional justice may carry implications for attending to social justice in a number of related psychosocial contexts. This especially includes encounters with healthcare providers or other healthcare system authorities, who can profoundly affect impressions of procedural and interactional justice through routine social exchange.

Social Justice as Psychological Justice

As highlighted by *Unequal Treatment* (2002), social justice is objective in the sense that the allotment of many health resources across various segments of a society can be quantified, as can the extent to which illness and healthcare-associated hardship disproportionately burdens these segments. Although this constitutes a critically important perspective, justice must also be more broadly construed as a psychological perception of individuals and communities and a psychosocial determinant of individual health and wellness in and of itself. In other words, social justice encompasses both the objective indices of inequity, as well as the subjective impressions of fairness held by individuals and groups. Importantly, both of these sources may be linked to health and illness.

Recognizing that justice also encompasses a psychosocial perception underscores two ways in which justice can be subjectively measured and linked to health within the social justice framework. First, justice may be operationalized as a momentary evaluation of a specific context or occurrence. That is, situational or *state justice* refers to judgments of fairness associated with specific settings and social interactions. State justice evaluations underscore that contextual factors are important to individuals in deciding whether momentary occurrences and social interactions are fair in nature. For example, the concept of *relative deprivation* suggests that perceptions of distributive justice in health contexts may depend on the extent to which one perceives his or her access to health resources to be equal to that of comparable others, irrespective of any objective level of health resource access (for recent review, Pettigrew, 2015). Considering state justice further underscores that perceptions of justice are adaptable and multidimensional and that individuals may simultaneously hold impressions of social justice and injustice. These aspects are perhaps best exemplified by the *organizational justice* measurement framework, which highlights that perceptions of fairness associated with evaluating specific contexts may be assessed in terms of evaluating an individual's impressions of distributive, procedural, and interactional justice criteria (e.g., Pérez-Arechaederra, Briones, Lind, & García-Ortiz, 2014).

In tandem to state justice judgments, a psychosocial consideration of social justice also suggests attending to individual differences tendencies, or *trait justice* (for recent review,

Dalbert, 2009). In health contexts, an individual differences approach underscores that stable and enduring tendencies to perceive justice in the world also carry health implications. Individual differences further highlight the multidimensional nature of justice, and they suggest that some justice tendencies may be more strongly implicated in health and well-being than others. At present, two individual differences distinctions have been especially well considered in health contexts. First is the distinction between personal and general justice beliefs. Whereas *personal justice beliefs* describe a stable propensity to view that the world as fair to one's self, *general justice beliefs* refer to the tendency to view the world in broad terms as fair or to believe in justice for others. A sizable body of research supports that these two justice tendencies are only moderately correlated with one another and that personal justice beliefs are especially associated with health and wellness outcomes (for review, Lucas & Wendorf, 2012). A second and more recent individual differences distinction considers distributive and procedural justice beliefs (Lucas, Zhdanova, & Alexander, 2011). In addition to characterizing impressions of fairness that accompany momentary social occurrences, procedural and distributive justice also encompass stable dispositional tendencies to perceive outcomes and allocations (*distributive justice beliefs*) versus rules and processes (*procedural justice beliefs*) as fair and deserved. Procedural and distributive justice beliefs are similarly moderately correlated with one another, and initial research suggests that each may be uniquely implicated in health. For example, whereas distributive justice may be especially associated with enhanced positive affect, procedural justice may play an especially prominent role in reducing negative affect. Other research suggests that procedural justice beliefs in particular may be associated with healthy behavior, including diet and exercise (Lucas, Alexander, Firestone, & LeBreton, 2008).

At least two future directions are evident for furthering a psychosocial consideration of justice and for deciphering links from psychologically oriented social justice to health and illness. First, and in terms of individual difference approaches, emerging literature suggests that the general–personal and distributive–procedural justice distinctions can be concurrently specified, such that individuals may be described by tendencies to believe in *distributive and procedural justice for self and others* (Lucas et al., 2011). Initial research using this four-factor measurement approach has suggested a potential to refine links between dispositional justice beliefs and health outcomes. For example, distributive justice beliefs for self have been shown to exclusively predict measures of *personal health*, such as self-rated health, whereas procedural justice beliefs for self exclusively predicted *interpersonal health*, such as conflict at work. Such graduated distinctions may carry potential implications for observing specific links to health and illness in a number of contexts.

A second future direction is suggested by growing consideration of the ways in which trait and state sources of justice jointly act as a social determinant of health and illness. One example is *worldview verification theory*, which suggests that individuals are highly motivated to maintain consistency between lived experiences and enduring worldviews and that health outcomes ultimately stem from inconsistencies between justice experiences and beliefs (Major & Townsend, 2012). According to worldview verification theory, health and wellness fundamentally depend on whether state and trait justice judgments are in agreement, even if these sources sway toward injustice. Thus, worldview verification suggests the seemingly ironic notion that perceptions of injustice may protect health better than perceptions of justice, so long as state and trait sources of injustice perceptions are congruent with one another. In support, recent research reveals that for racial minority individuals, justice dispositions and externally imposed justice jointly influence adaptive coping, and the degree of consistency between these two sources is ultimately crucial (Lucas et al., 2016, for recent example).

Health Consequences of Social Justice

Social justice is of interest to many because of its potential to influence the health and well-being of individuals and communities. Not surprisingly then, a psychosocial consideration of justice has revealed that perceptions of fairness affect the health of individuals in numerous ways (for reviews, Elovainio, Kivimäki, & Vahtera, 2002; Jackson, Kubzansky, & Wright, 2006; Lucas & Wendorf, 2012). Perhaps most clearly evident are associations between perceived fairness and mental health, especially including an inverse relationship between fairness and indices of negative emotion and depression. In addition to mental well-being, justice perceptions have been increasingly implicated in physical health. Most notably, research suggests that perceived injustice is prospectively associated with an increased incidence of cardiovascular illness. Strikingly, some research suggests that perceived injustice may be as strongly implicated in cardiovascular illness as traditional risk factors such as cholesterol, body mass index, and physical activity, and the contribution of perceived injustice to cardiovascular illness appears to be unique from that of seemingly related psychosocial variables.

From a mechanistic standpoint, health effects of psychological justice are thought to occur predominantly through links to stress. Stress pathways are empirically supported through studies linking perceived fairness to biological indices of stress reactivity, including activation of the sympathetic–adrenal–medullary axis (Tomaka & Blascovich, 1994) and also the hypothalamic–pituitary–adrenal axis (Lucas et al., 2016; Vermunt, Peeters, & Berggren, 2007). Recent research suggests that justice may also be linked to inflammatory reactivity (Lucas et al., 2016). Accordingly, justice theory has emphasized that perceived injustice may act as a stressor (Vermunt & Steensma, 2001). A justice approach to stress implicates cognitive appraisals of stressors, including evaluations of the threat value of an unfair event (i.e., primary appraisal) and also whether personal resources are adequate to cope with unfairness (i.e., secondary appraisal). Links to psychological and biological stress reactivity seem highly consistent with research suggesting that justice is especially important to heart health and depression, which have also been strongly linked to stress. More recently, research has identified health behaviors such as smoking, diet, and exercise and other forms of health detection and prevention as additional pathways through which justice beliefs may affect health (e.g., Lucas et al., 2008). One additional and particularly intriguing health pathway suggested by available literature is the connection between justice perceptions, insomnia, and metabolic risk (for recent example, Levine, Basu, & Chen, 2015). Other recent research is considering the ways in which perceptions of justice contribute to experiences of pain, which in turn may contribute to physical and mental wellness (McParland & Eccleston, 2013).

At least three future directions are relevant to further understanding the physical and mental health consequences of justice perceptions. First, future research must explore the extent to which perceptions of justice are particularly linked to stress and health among minority groups and individuals. This especially includes racial minorities, for whom justice perceptions may be intertwined with other known psychosocial determinants of stress and health, such as perceived discrimination and racial identity. Second and related, although a handful of studies have shown the ways in which justice may be related to acute biological stress reactivity, future research will be needed to better understand the ways in which justice perceptions are related to chronic stress. Critically, this includes the better understanding of the contributions of justice to chronically deregulated stress system functioning, which may be an important antecedent to broader health disparities. Third, future research must continue to explore the ways in which justice perceptions are linked to specific and impactful health behaviors. This includes further exploring how justice plays a role in the use of protective health behaviors such as diet

and exercise and also health-compromising behaviors such as smoking and substance use. Attending to health disparities that are underscored by the social justice framework may prove particularly useful in linking justice to health behavior. For example, the extent to which perceived justice affects adherence to prescribed medical regimens remains virtually unexplored, despite evidence that adherence among racial minorities is affected by perceived racism. Related efforts to encourage preventative health behavior often include the use of health communication strategies. The extent to which justice can be used as the basis of health messaging also holds considerable promise for future intervention research.

Author Biography

Todd Lucas is an associate professor in the Department of Family Medicine and Public Health Sciences and the Department of Psychology at Wayne State University (Detroit, MI). Dr. Lucas is a social and health psychologist. His research considers self and social regulation processes in stress and health. He is especially focused on the role of perceived fairness in health and social disparities.

References

- Blacksher, E., & Lovasi, G. S. (2012). Place-focused physical activity research, human agency, and social justice in public health: Taking agency seriously in studies of the built environment. *Health & Place*, 18, 172–179. doi:10.1016/j.healthplace.2011.08.019
- Chan, M. (2002). Violations of service fairness and legal ramifications: The case of the managed care industry. *Journal of Business Ethics*, 36, 315–336.
- Dalbert, C. (2009). Belief in a just world. In M. R. Leary & R. H. Hoyle (Eds.), *Handbook of individual differences in social behavior* (pp. 288–297). New York, NY: Guilford Publications.
- Elovainio, M., Kivimäki, M., & Vahtera, J. (2002). Organizational justice: Evidence of a new psychosocial predictor of health. *American Journal of Public Health*, 92, 105–108. doi:10.2105/AJPH.92.1.105
- Gilson, L. (2003). Trust and the development of health care as a social institution. *Social Science & Medicine*, 56, 1453–1468. doi:10.1016/S0277-9536(02)00142-9
- Jackson, B., Kubzansky, L. D., & Wright, R. J. (2006). Linking perceived unfairness to physical health: The perceived unfairness model. *Review of General Psychology*, 10, 21–40. doi:10.1037/1089-2680.10.1.21
- Jost, J. T., & Kay, A. C. (2010). Social justice: History, theory, and research. In S. T. Fiske, D. Gilbert, & G. Lindzey (Eds.), *Handbook of social psychology* (5th ed., Vol. 2, pp. 1122–1165). Hoboken, NJ: Wiley.
- Levine, C. S., Basu, D., & Chen, E. (2015). Just world beliefs are associated with lower levels of metabolic risk and inflammation and better sleep after an unfair event. *Journal of Personality*. doi:10.1111/jopy.12236
- Lucas, T., Alexander, S., Firestone, I. J., & LeBreton, J. M. (2008). Just world beliefs, perceived stress, and health behavior: The impact of a procedurally just world. *Psychology & Health*, 23, 849–866. doi:10.1080/08870440701456020
- Lucas, T., Lumley, M. A., Flack, J. M., Wegner, R., Pierce, J., & Goetz, S. M. M. (2016). A preliminary examination of worldview verification, perceived racism, and stress reactivity in African Americans. *Health Psychology*, 35, 366–375.
- Lucas, T., & Wendorf, C. A. (2012). Perceived justice and worker well-being. How and for whom do unethical work practices matter? In R. A. Giacalone & M. D. Promislo (Eds.), *Handbook of unethical work behavior: Implications for individual well-being* (pp. 237–252). New York, NY: ME Sharpe.

- Lucas, T., Zhdanova, L., & Alexander, S. (2011). Procedural and distributive justice beliefs for self and others: Assessment of a four-factor individual differences model. *Journal of Individual Differences*, 32, 14–25. doi:10.1027/1614-0001/a000032
- Major, B., & Townsend, S. S. (2012). Meaning making in response to unfairness. *Psychological Inquiry*, 23, 361–366. doi:10.1080/1047840X.2012.722785.
- McParland, J., & Eccleston, C. (2013). “It’s not fair”: Social justice appraisals in the context of chronic pain. *Current Directions in Psychological Science*, 22, 484–489. doi:10.1177/0963721413496811
- Nelson, A. (2002). Unequal treatment: confronting racial and ethnic disparities in health care. *Journal of the National Medical Association*, 94(8), 666.
- Penner, L. A., Blair, I. V., Albrecht, T. L., & Dovidio, J. F. (2014). Reducing racial health care disparities: A social psychological analysis. *Policy Insights From the Behavioral and Brain Sciences*, 1, 204–212.
- Pérez-Arechaederra, D., Briones, E., Lind, A., & García-Ortiz, L. (2014). Perceived organizational justice in care services: Creation and multi-sample validation of a measure. *Social Science & Medicine*, 102, 26–32. doi:10.1016/j.socscimed.2013.11.045
- Pettigrew, T. F. (2015). Samuel Stouffer and relative deprivation. *Social Psychology Quarterly*, 78, 7–24. doi:10.1177/0190272514566793
- Tomaka, J., & Blascovich, J. (1994). Effects of justice beliefs on cognitive appraisal of and subjective physiological, and behavioral responses to potential stress. *Journal of personality and social psychology*, 67(4), 732.
- Vermunt, R., Peeters, Y., & Berggren, K. (2007). How fair treatment affects saliva cortisol release in stressed low and high type-A behavior individuals. *Scandinavian Journal of Psychology*, 48, 547–555. doi:10.1111/j.1467-9450.2007.00593.x.
- Vermunt, R., & Steensma, H. (2001). Stress and justice in organizations: An exploration into justice processes with the aim to find mechanisms to reduce stress. In R. Cropanzano (Ed.), *Justice in the workplace* (Vol. 2, pp. 27–48). Mahwah, NJ: Lawrence Erlbaum Associates.

Suggested Reading

- Brondolo, E., Ver Halen, N. B., Pencille, M., Beatty, D., & Contrada, R. J. (2009). Coping with racism: A selective review of the literature and a theoretical and methodological critique. *Journal of Behavioral Medicine*, 32, 64–88. doi:10.1007/s10865-008-9193-0.
- Burgess, D., van Ryn, M., Dovidio, J., & Saha, S. (2007). Reducing racial bias among health care providers: Lessons from social-cognitive psychology. *Journal of General Internal Medicine*, 22, 882–887. doi:10.1007/s11606-007-0160-1.
- Major, B., Mendes, W. B., & Dovidio, J. F. (2013). Intergroup relations and health disparities: A social psychological perspective. *Health Psychology*, 32, 514. doi:10.1037/a0030358.

Health Consequences of Prejudice and Discrimination

Tessa L. Dover, Jeffrey M. Hunger, and Brenda Major

*Department of Psychological and Brain Sciences, University of California
Santa Barbara, Santa Barbara, CA, USA*

Prejudice and discrimination feature prominently in psychological research over the past 60 years. Most of this research focused on understanding the *causes* of prejudice and discrimination. It was not until the 1980s that research began in earnest to consider the *consequences* of prejudice and discrimination for those who are their targets. Since then a wealth of research has addressed the mental and physical health effects of perceived prejudice and discrimination (for reviews, see Lewis, Cogburn, & Williams, 2015; Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009). This research has shown that prejudice and discrimination can compromise the physical health of low-status, disadvantaged, and stigmatized groups in multiple ways (Lewis et al., 2015; Major, Mendes, & Dovidio, 2013; Williams & Mohammed, 2013). Below, we describe the different pathways through which prejudice and discrimination can get “under the skin” to undermine physical health. We also highlight critical moderators of how individuals cope with prejudice and discrimination that have implications for health.

Defining Terms

Discrimination and prejudice are related but distinct constructs and can take several different forms. *Prejudice* refers to negative attitudes toward a group. Prejudice can be explicit (acknowledged by the prejudiced individual) or implicit (based on underlying associations that may not be known or acknowledged by the prejudiced individual). Prejudice can also be “old-fashioned” (hostile or overt) or more “modern” (subtle or disguised). Prejudice may or may not be accompanied by poor treatment of the target group. *Discrimination*, in contrast, is behavioral. It refers to poor or unequal treatment of individuals based on their group membership. Many consider prejudice to be a precursor to discrimination, though discrimination can occur at both structural and interpersonal levels. For example, a policy may be discriminatory because

it favors one group over another, even though the policy cannot have “prejudiced attitudes” about a group.

Discrimination also takes several forms. *Acute discrimination* refers to a specific incidence of discrimination, with discrete start and end points—such as being refused service because of group membership. *Pervasive discrimination* refers to more chronic experiences of group-based mistreatment or the extent to which discrimination is experienced in day-to-day life. *Experienced* and *anticipated* discrimination can also be differentiated. A large body of work has assessed the health consequences of having experienced discrimination. A smaller but growing body of research also assesses the health consequences of anticipating being the target of prejudice and discrimination. Overall, this work suggests that the health consequences of prejudice and discrimination stem not only from direct experiences of overt maltreatment but also from the extra cognitive and emotional demands that anticipating prejudice places on targets as they navigate the social world.

A large literature has demonstrated the negative impact of discrimination and prejudice on indices of mental health, including stress, depression, anxiety, negative affect, lower self-esteem, and lower life satisfaction (see Pascoe & Smart Richman, 2009). Mental health is a primary component of overall health and well-being as well as a strong predictor of physical health. Due to space concerns, the current entry focuses primarily on the consequences of discrimination and prejudice for *physical* health. Indices of physical health reviewed here include self-reported health; mortality; cardiovascular, endocrine, and immunological functioning and reactivity; health behaviors; treatment outcomes; and several other biomarkers.

Pathways to Poor Health: Mechanisms of Discrimination and Prejudice Affecting Health

Although the association between discrimination and poor health is well documented, research understanding the mechanisms underlying this relationship is still in its infancy. Below we differentiate between two types of mechanisms (see Pascoe & Smart Richman, 2009, for a discussion of this framework). *Direct effects* refer to the ways in which systemic inequality and biased treatment erode health without any action or psychological input from the target (e.g., receiving poorer treatment by healthcare providers). *Indirect effects* refer to health-eroding behaviors or psychological states that occur in *response* to perceived or anticipated discrimination (e.g., the physiological stress response that accompanies exposure to discrimination, increased alcohol use in order to cope with perceived prejudice).

Direct Effects: Systemic and Interpersonal Mistreatment

The first pathway through which prejudice and discrimination undermine health is direct. By exposing targets to health-compromising environments and limiting access to resources important to promoting health (e.g., education, low-cost healthy foods), both interpersonal and institutionalized prejudice/discrimination can erode health for targets. Below, we briefly discuss four domains in which discrimination and prejudice directly compromise health: housing, education, employment, and healthcare.

In the housing domain, both institutionalized discrimination (e.g., “redlining” policies or using race/ethnicity as a consideration for mortgage accessibility) and interpersonal discrimination (e.g., landlords idiosyncratically favoring White tenants over tenants of color) have limited and continue to limit access to housing for targeted groups. Such segregation and housing disparities directly affect the health of targets by determining access to good schools, safe envi-

ronments, healthy food, outdoor spaces, low pollution, police protection, and economic mobility. In fact, Williams and Collins (2001) suggested that segregation and racialized housing policy are primary contributors to Black/White health disparities in the United States.

Well-documented racial and ethnic disparities also exist in access to quality private and public education. Moreover, teachers and administrators unequally allocate disciplinary policies such as expulsions, suspensions, and other punishments that can hinder performance in school to Black and Latino school children. Race-based school “tracking,” in which White students are encouraged to take more advanced classes than their non-White counterparts, is also common. These often-subtle disparities can directly affect health outcomes over time as academic success and educational achievement are strongly linked to health outcomes.

As with housing and educational discrimination, employment discrimination has shaped and continues to shape the health of targets. Prejudice on the part of hiring managers related to race, ethnicity, age, sexuality, and gender not only affects the ability to acquire the financial resources for a healthy lifestyle but also erects a substantial barrier to obtaining employer-subsidized health insurance.

The healthcare setting is another domain in which prejudice and discrimination directly undermine health. Several large-scale studies have demonstrated that Black, Latino, and poor individuals have less access to healthcare services than their White, Asian, and rich counterparts (e.g., Smedley, Stith, & Nelson, 2002). Moreover, once targets of discrimination and prejudice do gain access to the healthcare system, the quality of treatment, the nature of patient-provider interactions, and the resulting treatment recommendations they receive are often poorer than those of more advantaged groups. Medical conditions tend to be more accurately diagnosed and aggressively treated among White, young, and male patients compared with equivalent Black, older, and female patients—even with identical symptomology (e.g., Schulman et al., 1999).

Healthcare providers also evince strong negative implicit attitudes toward racial minority patients, overweight patients, and patients in other targeted groups, which may compromise the care these groups receive. Levels of implicit bias among healthcare providers have been shown to predict (a) their own behavior in clinical interactions, (b) treatment recommendations, and (c) their patients’ treatment outcomes. As such, health disparities likely result partially from implicit and explicit prejudice among healthcare providers that translates into poorer treatment (for a review, see Dovidio et al., 2008).

Indirect Effects: Stress

A second pathway by which experiencing or anticipating prejudice and discrimination can undermine health is by heightening the experience of stress, precipitating a cascade of neuroendocrine, immunological, and cardiovascular responses that can undermine health over time (Major et al., 2013; Pascoe & Smart Richman, 2009).

A large literature has examined the effects of perceived prejudice and discrimination on cardiovascular reactivity. Exposing individuals to discrimination or to potential discrimination against themselves or members of their group in the laboratory has been shown to lead to increased cardiovascular reactivity among racial/ethnic minorities, women, and overweight individuals. Discrimination has also been shown to activate the stress-responsive hypothalamic–pituitary–adrenal (HPA) axis, the primary output of which is cortisol. This relationship is complex, however, because both heightened cortisol reactivity and blunted reactivity have been shown in response to discrimination, and under certain circumstances, both can be maladaptive. Emerging research is also examining other biological systems that may be implicated

in the development of physical illnesses in response to discrimination. For example, both weight- and race-based discrimination correlate with oxidative stress, a pathogenic process implicated in chronic conditions such as diabetes and hypertension (e.g., Szanton et al., 2012). Limited research also suggests that experiencing prejudice and discrimination is associated with markers of systemic inflammation such as C-reactive protein and interleukin-6 (e.g., Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010). Inflammation can precipitate the development of age-related diseases such as cardiovascular disease and arthritis.

Indirect Effects: Health Behaviors

Experiencing or anticipating being the target of prejudice and discrimination can also alter health behaviors that are essential to maintaining physical well-being. Contending with the stress of prejudice and discrimination is effortful and can tax cognitive resources necessary for self-regulation. As a result, targets of prejudice and discrimination may experience diminished capacity to engage in some health-promoting behaviors. In addition, people may engage in behaviors that have negative health implications as a way of coping with the stress that accompanies experienced or anticipated discrimination. Below we discuss four types of health-related behavior that have been examined in response to prejudice and discrimination: eating behavior, substance use, exercise/physical activity, and healthcare behavior.

Perceived weight-based, gender-based, and race-based discrimination are associated with emotional eating and a host of disordered eating attitudes and behaviors (e.g., Moradi, Dirks, & Matteson, 2005). Likewise, perceiving race-based discrimination during adolescence is prospectively associated with poorer eating habits (e.g., greater fast-food consumption) as an adult. Experiencing or anticipating prejudice and discrimination may contribute to unhealthy eating behaviors by increasing cortisol production, which can spur a drive for high-fat and high-sugar foods; by decreasing the ability to engage in self-control; and by leading individuals to consume highly palatable (but unhealthy) foods to cope.

Adults reporting discrimination on the basis of race/ethnicity, gender, weight, and sexual orientation are more likely to meet diagnostic criteria for a substance use disorder such as nicotine, alcohol, or drug dependence. This relationship also emerges among adolescents. For example, in a longitudinal study of African American youth, greater reports of race-based discrimination at baseline were associated with more alcohol and marijuana use over time (Gibbons et al., 2010). Likewise, lesbian, gay, and bisexual (LGB) youth also report higher levels of substance use than their heterosexual counterparts; the odds of LGB youth engaging in substance use are 190% higher than heterosexual youth, presumably a result of minority stress (Lick, Durso, & Johnson, 2013).

There is also evidence that individuals alter their physical activity in response to perceived prejudice and discrimination, although this may depend on the target group. Research in the weight domain shows that experiencing weight-related criticism among adolescents and weight-based discrimination among adults is related to a greater motivation to avoid exercising in public as well as less physical activity. In the race domain, however, this relationship is more inconsistent. Some researchers found no relationship between perceived discrimination and physical activity; others found a negative relationship, while others still found a *positive* relationship. Whereas racial and ethnic minorities may engage in *increased* physical activity to cope with stress associated with perceived discrimination, overweight individuals likely avoid physical activity in an attempt to limit their exposure to the stigma prevalent in this domain (see Bastos, Celeste, Silva, Priest, & Paradies, 2015; Borrell, Kiefe, Diez-Roux, Williams, & Gordon-Larsen, 2013).

In the healthcare domain, patients from targeted groups often report low levels of trust in their healthcare providers and in the healthcare system in general. This lack of trust, perhaps in response to the healthcare biases evident among healthcare providers, can in turn impede patient adherence to treatment plans and reduce the likelihood that disadvantaged individuals seek medical care. Targets of discrimination and prejudice perceive greater levels of discrimination in the healthcare system compared with their nontargeted counterparts, and perceptions of discrimination are associated with greater healthcare avoidance (Burgess, Warren, Phelan, Dovidio, & Van Ryn, 2010).

Beyond any actual bias held by their healthcare provider, members of targeted groups may experience discomfort in intergroup interactions where discrimination or prejudice is deemed possible. As such, targets of prejudice and discrimination may experience stress, discomfort, and cognitive impairment in their clinical settings—all of which may have negative implications for physical health.

Moderators of the Effect of Discrimination and Prejudice on Health

Not all potential targets of discrimination and prejudice experience, anticipate, and perceive the same amount of unfair treatment. Nor do discrimination and prejudice affect all members of disadvantaged groups equally. Several key moderators of the discrimination–health relationship have been identified, many of which exacerbate or ameliorate the health consequences of discrimination and prejudice by affecting an individual's *perceptions of* or ability to *cope with* the stress of discrimination and prejudice. As such, most of the moderators identified by researchers have their effects on the indirect pathways—by moderating how much stress discrimination and prejudice elicit or by moderating how individuals behaviorally cope with that stress.

As is typical of research on stressors in general, people who have more *social support* (i.e., networks of friends or family that can lend tangible or emotional resources to the target) tend to have more healthful responses to discrimination than those with less social support. This is theorized to occur because those with wider and more helpful social connections have additional coping resources to draw on when experiencing discrimination and prejudice, thus lessening the stress of experiencing discrimination or prejudice.

An individual's *coping style* can also moderate the effect of discrimination and prejudice on health. Those who engage in active, problem-focused coping strategies (i.e., dealing with the stressor “head-on”) may experience fewer negative consequences of discrimination than those who engage in more passive coping strategies (i.e., avoiding or ignoring the stressor). However, some recent work has pointed to the limits of active coping styles as well (see, e.g., Brody et al., 2013).

Group identification (the extent to which one feels strongly connected to one's group) also influences responses to discrimination and prejudice. Several correlational studies point to group identification as a buffering factor for mental health. This research suggests that feeling connected to one's group makes discrimination less distressing and less likely to lead to depression (e.g., Mossakowski, 2003). However, experimental work suggests that group identification can also exacerbate the extent to which group-based discrimination is stressful. For example, Eliezer, Major, and Mendes (2010) found that for women high in gender identification, reading about sexism resulted in a more sustained cardiovascular threat response and anxiety compared with women low in gender identification.

Beliefs about fairness also shape how individuals perceive and cope with discrimination and prejudice. Specifically, the negative effects of discrimination on physical health tend to be more pronounced for Black Americans and women who believe the world was fair compared with those who believed the world was generally unfair. Dover, Major, Kunstman, and Sawyer (2015) found that Latino individuals who were treated unfairly by a White peer exhibited a maladaptive cardiovascular profile only when they generally saw the system as fair (vs. unfair). When Latinos believed the world was unfair, they responded to discrimination with an adaptive cardiovascular response. Research in this area suggests that believing that the social system is unfair can potentially buffer individuals from the stress of acute experiences of discrimination and prejudice.

Conclusion

The health implications of prejudice and discrimination are clear: a large body of work has documented the numerous direct and indirect pathways through which discrimination and prejudice undermine the health of targets. While important strides have been made over the past decade to unpack the mechanisms through which discrimination can lead to poor physical health, added research is clearly warranted. In particular there is a need for theoretically based research that integrates multiple methods (e.g., experimental and intensive longitudinal designs) and additional dimensions of prejudice and discrimination (e.g., discrimination-related vigilance) with indicators of physical health that span levels of analysis (e.g., biological/physiological, behavioral) to more accurately and adequately capture the health effects of prejudice and discrimination. Moreover, additional emphasis should be placed on understanding the individual and system-level variables that moderate how individuals cope with discrimination and prejudice. Ultimately, this will foster a better understanding of these processes and allow researchers, policy makers, and public health practitioners to create theoretically and empirically supported interventions to ameliorate the undue health burden associated with prejudice and discrimination.

Author Biographies

Tessa L. Dover is a social psychology PhD candidate in UC Santa Barbara's Department of Psychological and Brain Sciences. She researches the mental and physical health consequences of group-based unfairness among high-status and low-status groups. Her main focus involves the interplay between group status and fairness beliefs in predicting responses to identity-relevant social cues.

Jeffrey M. Hunger is a doctoral candidate in social psychology at the University of California, Santa Barbara. Broadly speaking, he is interested in using insights from social, personality, and health psychology to understand and improve the well-being of stigmatized populations. His current research focuses primarily on the mental and physical health consequences of weight stigma.

Dr. Brenda Major is a distinguished professor in the Department of Psychological and Brain Sciences at the University of California, Santa Barbara. She is an international expert in the psychology of stigma and how people perceive and cope with stigma and discrimination. A core theme of her work is psychological resilience—how people maintain their sense of self-esteem, psychological well-being, and physical health despite exposure to discrimination, negative life events, and adversity.

References

- Bastos, J. L., Celeste, R. K., Silva, D. A. S., Priest, N., & Paradies, Y. C. (2015). Assessing mediators between discrimination, health behaviours and physical health outcomes: A representative cross-sectional study. *Social Psychiatry and Psychiatric Epidemiology*, 50(11), 1731–1742. doi:10.1007/s00127-015-1108-0
- Borrell, L. N., Kiefe, C. I., Diez-Roux, A. V., Williams, D. R., & Gordon-Larsen, P. (2013). Racial discrimination, racial/ethnic segregation, and health behaviors in the CARDIA study. *Ethnicity & Health*, 18(3), 227–243. doi:10.1080/13557858.2012.713092
- Brody, G. H., Yu, T., Chen, E., Miller, G. E., Kogan, S. M., & Beach, S. R. (2013). Is resilience only skin deep? Rural African Americans' socioeconomic status-related risk and competence in preadolescence and psychological adjustment and allostatic load at age 19. *Psychological Science*, 24(7), 1285–1293. doi:10.1177/0956797612471954
- Burgess, D. J., Warren, J., Phelan, S., Dovidio, J., & Van Ryn, M. (2010). Stereotype threat and health disparities: What medical educators and future physicians need to know. *Journal of General Internal Medicine*, 25(2), 169–177. doi:10.1007/s11606-009-1221-4
- Dover, T. L., Major, B., Kunstman, J. W., & Sawyer, P. J. (2015). Does unfairness feel different if it can be linked to group membership? Cognitive, affective, behavioral and physiological implications of discrimination and unfairness. *Journal of Experimental Social Psychology*, 56(1), 96–103. doi:10.1016/j.jesp.2014.09.003
- Dovidio, J. F., Penner, L. A., Albrecht, T. L., Norton, W. E., Gaertner, S. L., & Shelton, J. N. (2008). Disparities and distrust: The implications of psychological processes for understanding racial disparities in health and health care. *Social Science & Medicine*, 67(3), 478–486. doi:10.1016/j.socscimed.2008.03.019
- Eliezer, D., Major, B., & Mendes, W. B. (2010). The costs of caring: Gender identification increases threat following exposure to sexism. *Journal of Experimental Social Psychology*, 46(1), 159–165. doi:10.1016/j.jesp.2009.09.015
- Gibbons, F. X., Etcheverry, P. E., Stock, M. L., Gerrard, M., Weng, C. Y., Kiviniemi, M., & O'Hara, R. E. (2010). Exploring the link between racial discrimination and substance use: What mediates? What buffers? *Journal of Personality and Social Psychology*, 99(5), 785–801. doi:10.1037/a0019880
- Lewis, T. T., Aiello, A. E., Leurgans, S., Kelly, J., & Barnes, L. L. (2010). Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. *Brain, Behavior, and Immunity*, 24(3), 438–443. doi:10.1016/j.bbi.2009.11.011
- Lewis, T. T., Cogburn, C. D., & Williams, D. R. (2015). Self-reported experiences of discrimination and health: Scientific advances, ongoing controversies, and emerging issues. *Annual Review of Clinical Psychology*, 11, 407–440. doi:10.1146/annurev-clinpsy-032814-112728
- Lick, D. J., Durso, L. E., & Johnson, K. L. (2013). Minority stress and physical health among sexual minorities. *Perspectives on Psychological Science*, 8(5), 521–548. doi:10.1177/1745691613497965
- Major, B., Mendes, W. B., & Dovidio, J. F. (2013). Intergroup relations and health disparities: A social psychological perspective. *Health Psychology*, 32(5), 514–524. doi:10.1037/a0030358
- Moradi, B., Dirks, D., & Matteson, A. V. (2005). Roles of sexual objectification experiences and internalization of standards of beauty in eating disorder symptomatology: A test and extension of Objectification Theory. *Journal of Counseling Psychology*, 52(3), 420–428. doi:10.1037/0022-0167.52.3.420
- Mossakowski, K. N. (2003). Coping with perceived discrimination: Does ethnic identity protect mental health? *Journal of Health and Social Behavior*, 44(3), 318–331. doi:10.2307/1519782
- Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin*, 135(4), 531–554. doi:10.1037/a0016059
- Schulman, K. A., Berlin, J. A., Harless, W., Kerner, J. F., Sistrunk, S., Gersh, B. J., ... Eisenberg, J. M. (1999). The effect of race and sex on physicians' recommendations for cardiac catheterization. *New England Journal of Medicine*, 340(8), 618–626.

- Smedley, B. D., Stith, A. Y., & Nelson, A. R. (Eds.) (2002). *Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington, DC: National Academies Press.
- Szanton, S. L., Rifkind, J. M., Mohanty, J. G., Miller, E. R., III, Thorpe, R. J., Nagababu, E., ... Evans, M. K. (2012). Racial discrimination is associated with a measure of red blood cell oxidative stress: A potential pathway for racial health disparities. *International Journal of Behavioral Medicine*, 19(4), 489–495. doi:10.1007/s12529-011-9188-z
- Williams, D. R., & Collins, C. (2001). Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Reports*, 116, 404–416. doi:doi.org/10.1093/phr/116.5.404
- Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine*, 32(1), 20–47. doi:10.1007/s10865-008-9185-0
- Williams, D. R., & Mohammed, S. A. (2013). Racism and health I: Pathways and scientific evidence. *American Behavioral Scientist*, 57(8), 1152–1173. doi:10.1177/0002764213487340

Suggested Reading

- Hatzenbuehler, M. L., Phelan, J. C., & Link, B. G. (2013). Stigma as a fundamental cause of population health inequalities. *American Journal of Public Health*, 103(5), 813–821.
- Lewis, T. T., Cogburn, C. D., & Williams, D. R. (2015). Self-reported experiences of discrimination and health: Scientific advances, ongoing controversies, and emerging issues. *Annual Review of Clinical Psychology*, 11, 407–440.
- Major, B., Mendes, W. B., & Dovidio, J. F. (2013). Intergroup relations and health disparities: A social psychological perspective. *Health Psychology*, 32(5), 514–524.
- Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin*, 135(4), 531–554.
- Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine*, 32(1), 20–47.

The Health Correlates and Consequences of Friendship

Robert G. Kent de Grey and Bert N. Uchino

Department of Psychology, University of Utah, Salt Lake City, UT, USA

In his *Nicomachean Ethics*, Aristotle is credited as stating, “a friend is a second self.” Friendships, however, entail importance beyond the pleasant affective experience of having close others. Social ties are critical to cognitive and affective development across the lifespan and have benefits beyond this as well. In a recent meta-analysis involving more than 308,000 participants, the quality of one’s social relationships was linked with reduced risk of mortality (Holt-Lunstad, Smith, & Layton, 2010). The magnitude of this association was comparable with smoking cessation and exceeded many other conventional risk factors (e.g., physical inactivity, obesity).

Although most of the prior epidemiological work has not separated out specific relationship types (e.g., family, friends), there are strong reasons to believe that friendships in particular might be important to health. In contrast to relationships with family members, friendships are voluntary (nonbinding) relationships and thus are an important source of support across a variety of situations. Of course, this does not mean that friends have entirely positive influences. Under some circumstances friendships may adversely relate to health. In the present entry, we thus provide a general overview on links between friendships and health, including potential mechanisms, individual differences, intervention opportunities, and important future directions.

Epidemiological Evidence Linking Friendships to Health

Research suggests friendships may decrease risk for health problems and early mortality. These health benefits are evident across the lifespan and can be seen in early life friendships. In a long-term study, researchers assessed the number of friendships reported by 5,814 Scottish schoolchildren and linked them to self-rated health 36–39 years later (Almqvist, 2012). For women, a greater number of childhood friends correlated with higher ratings of health. Among male participants, those who listed no friends at the time of the initial survey reported lower adult health

quality. Finally, another large-scale study of 11,617 adolescents reported on their experiences with close friends. Fourteen years later, analysis of HbA1c (glycohemoglobin, an index of blood glucose levels associated with, and used in the monitoring of, diabetes mellitus) showed that, for female participants, having close male friendships was associated with reduced odds of elevated metabolic risk marker levels (Ehrlich, Hoyt, Sumner, McDade, & Adam, 2015).

It is also important to note that the health correlates and consequences of friendship can sometimes approach or exceed the effect size for social support from family members. For instance, in an analysis using data from the longitudinal Health and Retirement Study, 7,514 US adults were surveyed in 2006 regarding social environmental variables. These responses were used to predict the onset of six conditions (high blood pressure, heart condition, cancer, stroke, lung disease, and arthritis) in 2010. Factors related to friends were more predictive of these health outcomes than factors related to family (Hill, Weston, & Jackson, 2014). In summary, although more work is needed, the available epidemiological evidence suggests that friendships are consequential for health.

Mechanisms Linking Friendships to Health

Mechanisms underlying the association between friendships and health are made salient by two major perspectives: the stress-buffering and direct effects models. Of these theoretical approaches, the more well known is the stress-buffering hypothesis, which holds that social support is healthy primarily because it buffers against the damaging effects of stress. Stress arises in part from the appraisal process—the evaluation of our coping abilities and the demands placed on them. Social support may decrease the perceived and objective coping burdens relative to available resources, as support has been shown to lessen the negative effects of many types of stressors.

The stress-buffering model has been tested with regard to friends in well-controlled laboratory studies. For instance, Christenfeld et al. (1997) examined this by stressing participants with a speech task and assigning them to receive support from a friend or stranger (confederate), or to receive no support. Social support from a friend resulted in the largest reduction in cardiovascular reactivity during stress. Compellingly, supportive behaviors were standardized across conditions, suggesting that differences were not due to friends merely knowing participants more intimately and providing better support on the basis of this knowledge. Although support from friends in such studies is generally beneficial, some studies have revealed more complex results and led to the suggestion that future work should pay closer attention to the support context, for example preferences for support.

In addition to the stress-buffering hypothesis, another major perspective is the direct effects model. This perspective broadly emphasizes the overall benefits of integration into a social network. Social control is one important aspect of this model and suggests that embedment in a social network is healthy because it creates social roles that provide self-esteem and meaning in life. Friends and others may also exert direct social control by attempting to influence individuals to change their health behaviors. Consistent with the social control hypothesis, an analysis of 469 elderly members of a Thai community found friends (but not family) had a direct link to health promoting behaviors such as exercise, healthful eating, and stress management (Thanakwang & Soonthornthada, 2011).

Friendships may also be related to health behaviors that are not completely voluntary. A relatively large literature suggests aspects of sleep, such as quality and duration, are related to health outcomes, including mortality rates. In a community sample of 175 US middle-aged to older adults, participants reported how many friends/family ties they had and characterized

the quality of those friendships. The number of supportive friendships (but not supportive family members) was a significant predictor of better self-reported sleep quality (Kent, Uchino, Cribbet, Bowen, & Smith, 2015).

While the overall findings for the direct effects model suggest that friendships can contribute to positive health behaviors, relationships can have negative consequences in some circumstances (e.g., risk taking and substance abuse). In addition, overt social control may arouse resentment or serve as a source of significant stress if the individual is unwilling or unable to engage in healthy behavioral change. For instance, one study found that perceptions of direct social network control (including friends) was related to greater aging at the cellular level (Uchino et al., 2015). Similarly, research suggests that receiving highly visible social support from friends or others may harm self-esteem or contribute to depression, both of which may be detrimental to health. Thus, high levels of direct social control and highly visible or unsolicited social support appear to relate to negative health outcomes.

Friendships may also have negative influences on health via normative influences (Rook & Pietromonaco, 1987). For example, the social interconnection of 12,067 adults in the Framingham Heart Study was repeatedly assessed along with body mass index (BMI) from 1971 to 2003. Longitudinal models showed that the chances of becoming obese increased 37% if one's spouse became obese and rose by 40% if one's adult sibling became obese, but the largest increase was a 57% greater risk of obesity if a participant had a friend who became obese during the study (Christakis & Fowler, 2007). This social contagion of bad health habits via friends is consistent with findings indicating that friends are surprisingly similar on measures such as frequency and duration of participation in organized physical activity, fast-food habits, body image, and alcohol use (see, e.g., Fletcher, Bonell, & Sorhaindo, 2011). This phenomenon of friend similarity in alcohol use is well documented in university settings, where social norms have been found to exert large influence on drinking behaviors, even when such norms do not reflect the beliefs of most individual students.

Individual Differences, Friendships, and Health

A number of factors may influence the relationship between friendship and health. Men and women differ in important ways that may relate to friendship. One possibility is that females may be more sensitive to the quality of their relationships due to more complex relational schemata. For instance, women may be more affected by adverse interpersonal processes because their self-esteem is more contingent on interpersonal functioning. Women may thus be more sensitive to or expend greater effort in response to relationship concerns. This may translate to health-relevant physiological processes as well; a study of 303 normotensive adults found that conflict within close relationships predicted nocturnal blood pressure dipping for women, but not for men (Holt-Lunstad, Jones, & Birmingham, 2009). Absence of such nightly decreases in blood pressure has been shown to predict both cardiovascular and all-cause mortality.

Exploration of cultural differences is also an area of increasing interest in relationships and health. Importantly, friendship appears to have value and health relevance across many nations, though its specific relation to aspects of well-being may vary. Most of this work has focused on differences between individualistic and collective (especially Asian) cultures. In such work, there is a significant distinction between explicit (i.e., seeking and using support) and implicit (i.e., the comfort of simply having close relationships) support. Due to relational concerns (e.g., worries about placing a burden on friends who are obligated to be helpful), individuals

from more collective cultures may experience greater benefit during stress from implicit forms of support, in which they simply feel more connected with others.

Interventions Utilizing Friends and their Influence on Health

The research reviewed thus far suggests that interventions targeting friendships might be useful in improving health outcomes. Although more work is certainly needed in this area, several intervention approaches are consistent with this possibility. “Befriending” specifically focuses on the friendship formation processes by recruiting outside relationships to serve as the basis for affirming social support over time. A meta-analysis suggests a modest effect for such interventions in improving outcomes in different patient populations (e.g., caregivers, prostate cancer patients; Mead, Lester, Chew-Graham, Gask, & Bower, 2010).

Another important intervention related to building friendships involves support groups. Interventions examining peer support groups suggest beneficial effects on adjustment and well-being (Hogan, Linden, & Najarian, 2002). Researchers have also found that participation in support groups seems to influence the size and composition of one’s friendship network. For instance, studies of individuals with substance abuse problems suggest that self-help groups result in decreased contact with drug-using network members and increased contact with support group friends (Humphreys & Noke, 1997). These network members then become crucial sources of support in offering advice and guidance to help individuals remain abstinent.

Success has also been shown for relationship interventions that teach individuals the skills necessary to acquire responsive support (Hogan et al., 2002). In one intervention, adolescents were assigned to enroll in a semester-long course that focused on friendship development as well as positive peer and teacher relationships (Eggert, Thompson, Herting, Nicholas, & Dicker, 1994). Students also learned how to elicit support regarding personal problems. Results of this study showed that individuals in the intervention had an increase in their friendships and in self-esteem. Moreover, compared with adolescents not provided with the intervention, individuals with social skills training showed a trend toward less drug use, a decrease in drug-related problems, and an increase in grade point average.

More generally, friends may be an important source of support for individuals attempting healthy behavioral change. In one study, researchers combined a support intervention with standard behavioral treatment (SBT) for weight loss (Wing & Jeffery, 1999). Participants either came alone or were asked to bring three friends who were interested in weight loss. Importantly, individuals who participated in the support intervention with friends were more likely to complete the program and maintain their weight loss over the 10-month period. One interesting aspect of this study was the finding that individuals who recruited three friends to join them for the protocol were less likely to have participated in prior organized weight-loss programs. These data suggest an additional benefit of recruitment with friends as it may increase participation for individuals who may not wish to go alone. In summary, although more work is needed, the existing literature highlights the promise of friendship-based interventions to improving health outcomes.

Future Directions

As reviewed in this entry, friendships can have both positive and negative influences on health. Of course, this is true of family relationships as well, and future work that directly contrasts

different types of relationships and the functions they serve will be important to advance theory and highlight unique intervention opportunities. Future work should also examine the utility of considering positive and negative aspects of relationships in combination. Corresponding to the colloquial concept of the “frenemy,” an ambivalent friend is one characterized by both high levels of positivity and negativity. Although one might ask why we would continue to be friends with such ambivalent ties, research indicates that individuals have a sense of commitment to them based on a long history of interactions. Importantly, existing research indicates that friendship ambivalence is quite common and related to negative health-relevant biological outcomes (Holt-Lunstad, Uchino, Smith, & Hicks, 2007).

It is also impossible to ignore the fact that an increasing proportion of social interaction takes place online. Scientific examination of differences between online and offline friendships is still in its infancy. People may be more willing to self-disclose when getting to know someone online vs. in person, which could facilitate friendship formation. The Internet may also preserve friendships that would otherwise deteriorate over time. In one study, the number of Facebook friends was a stronger predictor (vs. offline interpersonal network size) of perceived social support, less physical illness, and greater well-being among participants with a high number of objective life stressors (Nabi, Prestin, & So, 2013). While causality in such research presently remains largely unknown, research indicates that Facebook members who use the site multiple times daily score eight percentage points higher than average Americans on a measure of social support (Hampton, Goulet, Rainie, & Purcell, 2011). The same researchers interpret their findings to mean that “someone who uses Facebook multiple times per day gets about half the boost in total support that someone receives from being married or living with a partner” (Hampton et al., 2011, p. 35). While this claim requires a great deal of additional research before substantiation, such a bold statement hints at the potential importance of this emerging research area.

As mentioned above, much existing epidemiological research does not separate specific relationship types (e.g. friends, family, coworkers). Future work should distinguish between relationship categories to show how friendships may differ from other social ties in terms of health relevance. When relationship types are aggregated, researchers may not be able to detect effects that pertain to specific kinds of social ties (see, for example, Kent et al., 2015).

Conclusions

Friendship may serve as an important source of social support during stress, promote healthy behaviors, and contribute to healthful social norms, all of which may link to positive health outcomes. However, unsolicited, intrusive, or highly visible support attempts, as well as direct social control, may be mechanisms by which friendships can adversely influence health. Additional research is needed to more fully understand the health consequences and mechanisms associated with friendship, which can rival family relationships in terms of health relevance.

Author Biographies

Robert G. Kent de Grey, MS, is a PhD candidate in social and health psychology at the University of Utah studying relationships and health. He also researches in the Cancer Control and Population Sciences Program at the Huntsman Cancer Institute. Specific subareas of

interest include online social networks and virtual interactions, as well as proenvironmental attitudes and behaviors. He serves as a consulting environmental and social psychologist to the University of Utah Sustainability Office.

Bert N. Uchino, PhD, is a professor of social and health psychology at the University of Utah. His graduate studies at The Ohio State University were supervised by Dr. John T. Cacioppo. Uchino also researches the influence of social factors on aging processes as part of the Interdepartmental Program in Neuroscience at the University of Utah. In 2004, Yale University Press published his book *Social Support and Physical Health: Understanding the Health Consequences of Relationships*.

References

- Almquist, Y. M. (2012). Childhood friendships and adult health: Findings from the Aberdeen children of the 1950s cohort study. *The European Journal of Public Health*, 22(3), 378–383. doi:10.1093/eurpub/ckr045
- Christakis, N. A., & Fowler, J. H. (2007). The spread of obesity in a large social network over 32 years. *New England Journal of Medicine*, 357(4), 370–379. doi:10.1056/NEJMsa066082
- Christenfeld, N., Gerin, W., Linden, W., Sanders, M., Mathur, J., Deich, J. D., & Pickering, T. G. (1997). Social support effects on cardiovascular reactivity: Is a stranger as effective as a friend? *Psychosomatic Medicine*, 59(4), 388–398.
- Eggert, L. L., Thompson, E. A., Herting, J. R., Nicholas, L. J., & Dicker, B. G. (1994). Preventing adolescent drug abuse and high school dropout through an intensive school-based social network development program. *American Journal of Health Promotion*, 8(3), 202–215. doi:10.4278/0890-1171-8.3.202
- Ehrlich, K. B., Hoyt, L. T., Sumner, J. A., McDade, T. W., & Adam, E. K. (2015). Quality of relationships with parents and friends in adolescence predicts metabolic risk in young adulthood. *Health Psychology*, 34(9), 896–904. doi:10.1037/hea0000213
- Fletcher, A., Bonell, C., & Sorhaindo, A. (2011). You are what your friends eat: Systematic review of social network analyses of young people's eating behaviours and bodyweight. *Journal of Epidemiology and Community Health*, 65(6), 548–555. doi:10.1136/jech.2010.113936
- Hampton, K. N., Goulet, L. S., Rainie, H., & Purcell, K. (2011). *Social networking sites and our lives: How people's trust, personal relationships, and civic and political involvement are connected to their use of social networking sites and other technologies*. Washington, DC: Pew Internet & American Life Project.
- Hill, P. L., Weston, S. J., & Jackson, J. J. (2014). Connecting social environment variables to the onset of major specific health outcomes. *Psychology & Health*, 29(7), 753–767. doi:10.1080/08870446.2014.884221
- Hogan, B. E., Linden, W., & Najarian, B. (2002). Social support interventions: Do they work? *Clinical Psychology Review*, 22(3), 381–440. doi:10.1016/s0272-7358(01)00102-7
- Holt-Lunstad, J., Jones, B. Q., & Birmingham, W. C. (2009). The influence of close relationships on nocturnal blood pressure dipping. *International Journal of Psychophysiology*, 71(3), 211–217. doi:10.1016/j.ijpsycho.2008.09.008
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7(7), e1000316. doi:10.1371/journal.pmed.1000316
- Holt-Lunstad, J., Uchino, B. N., Smith, T. W., & Hicks, A. (2007). On the importance of relationship quality: The impact of ambivalence in friendships on cardiovascular functioning. *Annals of Behavioral Medicine*, 33(3), 278–290. doi:10.1080/08836610701359795
- Humphreys, K., & Noke, J. M. (1997). The influence of posttreatment mutual help group participation on the friendship networks of substance abuse patients. *American Journal of Community Psychology*, 25(1), 1–16. doi:10.1023/A:1024613507082

- Kent, R. G., Uchino, B. N., Cribbet, M. R., Bowen, K., & Smith, T. W. (2015). Social relationships and sleep quality. *Annals of Behavioral Medicine*, 49(6), 912–917. doi:10.1007/s12160-015-9711-6
- Mead, N., Lester, H., Chew-Graham, C., Gask, L., & Bower, P. (2010). Effects of befriending on depressive symptoms and distress: Systematic review and meta-analysis. *The British Journal of Psychiatry*, 196(2), 96–101. doi:10.1192/bjp.bp.109.064089
- Nabi, R. L., Prestin, A., & So, J. (2013). Facebook friends with (health) benefits? Exploring social network site use and perceptions of social support, stress, and well-being. *Cyberpsychology, Behavior and Social Networking*, 16(10), 721–727. doi:10.1089/cyber.2012.0521
- Rook, K. S., & Pietromonaco, P. (1987). Close relationships: Ties that heal or ties that bind. *Advances in Personal Relationships*, 1, 1–35.
- Thanakwang, K., & Soonthorndhada, K. (2011). Mechanisms by which social support networks influence healthy aging among Thai community-dwelling elderly. *Journal of Aging and Health*, 23(8), 1352–1378. doi:10.1177/0898264311418503
- Uchino, B. N., Cawthon, R., Smith, T. W., Kent, R. G., Bowen, K., & Light, K. (2015). A cross-sectional analysis of the association between perceived network social control and telomere length. *Health Psychology*, 34(5), 531–538. doi:10.1037/hea0000148
- Wing, R. R., & Jeffery, R. W. (1999). Benefits of recruiting participants with friends and increasing social support for weight loss and maintenance. *Journal of Consulting and Clinical Psychology*, 67(1), 132–138. doi:10.1037/0022-006X.67.1.132

Suggested Reading

- Holt-Lunstad, J. (2016). Friendship and physical health. In M. Hojjat & A. Moyer (Eds.), *The psychology of friendship* (pp. 233–249). New York, NY, US: Oxford University Press.
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7(7), e1000316. doi:10.1371/journal.pmed.1000316
- Mendes de Leon, C. F. (2005). Why do friendships matter for survival? *Journal of Epidemiology and Community Health*, 59, 537.
- Sneed, R. S., & Cohen, S. (2014). Negative social interactions and incident hypertension among older adults. *Health Psychology*, 33, 554–565.
- Uchino, B. N. (2004). *Social support and physical health: Understanding the health consequences of relationships*. New Haven, CT: Yale University Press.

Health Correlates and Consequences of Social Comparison

Dana M. Litt¹, Anne M. Fairlie¹, Melissa A. Lewis¹, and Michelle L. Stock²

¹*Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA*

²*Department of Psychological & Brain Sciences, The George Washington University, Washington, DC, USA*

Based on the rapid growth of health psychology over the past few decades, it is clear that issues surrounding health and disease have been major areas of study in modern psychological research. Although there are many psychological approaches and theories that have been applied to health psychology, social comparison theory (Festinger, 1954) has proven to be one of the most important for applications to health-related cognitions and behaviors (Buunk & Gibbons, 1997). Social comparison was originally conceived as a way for people to evaluate themselves in the absence of objective standards (Festinger, 1954) and has been further suggested to be a process through which people search for self-relevant information and how to gain important self-knowledge (e.g., Buunk & Gibbons, 1997). Although Festinger and other early social comparison researchers did not directly discuss social comparison in relation to health behaviors, researchers have argued that the theory is directly relevant to many health-related issues (Buunk & Gibbons, 1997; Litt, Stock, & Gibbons, 2014). As such, a more recent literature has examined the role that social comparison plays in health risk behaviors (Gibbons, Stock, Gerrard, & Finneran, 2015). This is not surprising given that past research has shown that people's risk judgments and decisions are highly affected by comparative reference points (van der Pligt, 1996). Additionally, in a volume that summarized applications of social comparison theory to health (Buunk & Gibbons, 1997), research indicated that people compare themselves with risk prototypes as well as other people who currently engage in risk behaviors when considering whether or not to engage in risky behavior (Gibbons et al., 2015). Combined, this research suggests that social comparisons play an important role in decisions to engage in health risk behaviors.

As this chapter will show, social comparison theory has contributed significantly to the theoretical foundation of health psychology. The primary goal of this entry is to provide a brief

overview of social comparison theory, highlight specific scientific developments related to health behavior, summarize the role that social comparison plays on health and illness, describe the role that social comparison plays in both the etiology and intervention of health and health risk behaviors, and describe important future research directions.

Social Comparison and Illness/Disease

The role of social comparison in mental and physical health has received considerable attention across a broad range of areas including cancer (Brakel, Dijkstra, & Buunk, 2012), diabetes (Schokker et al., 2010), and HIV (Siegel, Lekas, Schrimshaw, & Brown-Bradley, 2011). People dealing with illness and disease symptoms may evaluate their own attitudes, behaviors, and circumstances as a way to understand themselves, process their emotions, and guide future behavior (Suls, Martin, & Leventhal, 1997). As part of the self-evaluation process, people compare themselves with other individuals who are also dealing with the illness, particularly in cases where there is uncertainty regarding their own health or treatment (Suls et al., 1997).

Social comparison can play a role in how individuals manage and cope with chronic illness in a variety of ways. Direction of the comparison refers to the standing of the target in relation to that of the person making the comparison. In the case of illness, upward comparisons are in reference to someone who is perceived as better off (e.g., more responsive to treatment, fewer symptoms), and downward comparisons are in reference to someone who is perceived as worse off (e.g., less likely to fully recover). Perceived similarity to the target may be based on a range of attributes, including illness severity, the type of treatment being received, and even basic demographics (e.g., age, gender). Mode of comparison refers to the manner in which the person learns about the comparison target. Information about a target can be obtained proximally through contact or affiliation with the target (e.g., other patients), distally whereby a person may receive information about the target from another source (e.g., media, another person without the illness) or even through fabricated targets (Buunk & Gibbons, 1997). People may either identify with the target by perceiving themselves as similar to or likely to become similar to the target, or they may make contrasts between themselves and the target such that differences between themselves and the target are highlighted (Mussweiler, Ruter, & Epstude, 2004). Arigo, Suls, and Smyth (2014) found that identifying with or interacting with someone who is perceived to be coping better (upward identification) as well as contrasting oneself to or being exposed to information about someone whose illness is more severe (downward contrast) can be beneficial to patients. In addition, people who are high in neuroticism may frequently engage in social comparison without receiving its beneficial effects and may instead reinforce negative self-perceptions (Buunk & Gibbons, 1997).

People may also compare their attitudes and situation to that of others in order to gain information about the course of the disease, treatment/care, or their likelihood of recovery as well as to gauge how well they are coping with the disease and managing their symptoms. A recent review by Arigo, Suls, et al. (2014) summarized social comparison research that focused on individuals with chronic illnesses. Of the 37 studies included in the review, almost half of the studies included samples of patients currently diagnosed with cancer or in remission, while the remaining studies included samples of patients diagnosed with a variety of diseases including arthritis and cardiovascular disease. Despite some ambiguous results, several consistent trends emerged as a result of this review. For one, identifying or having actual contact with someone who is coping better tends to be beneficial for patients. However, being exposed to information about someone with more severe illness tends to be less beneficial. In contrast,

downward comparisons about coping and upward comparisons about illness severity seem to be unhelpful.

Social comparison processes may operate at different points during the course of an illness, ranging from the individual's initial evaluation of symptoms to the individual's decision about treatment planning once the illness has become severe. In order to receive healthcare, a person must first decide to seek out medical care in order to obtain an evaluation of his or her symptoms, which can result in a diagnosis and a treatment plan. Research has shown that social comparison processes can enhance or impede the decision to seek medical treatment (Suls et al., 1997). Mojtabai (2008) demonstrated that, among adults in the United States, individuals who perceived themselves as more worried or anxious than others were more likely to seek care from a doctor or mental health professional compared with those who did not perceive themselves as more worried or anxious. Therefore, social comparison processes come into play early on as individuals consider their symptoms and decide whether or not treatment, or even consultation with a health professional, is needed. The occurrence of seeking delayed care, after the onset of symptoms such that the illness has had a chance to progress, also has implications for the severity of symptoms among those being treated. Delayed onset of care has further implications for the role of social comparison processes once a diagnosis has been made and individuals are seeking treatment. For example, if for a particular type of illness or symptom, people tend to delay the onset of healthcare, the "typical" patient may be more sick than an individual who chooses to receive medical attention early.

As noted by Arigo, Sulz, et al. (2014), studies on illness and disease have varied greatly with respect to the type of disease (e.g., arthritis, HIV, sickle cell disease) or even the subtype of the disease (e.g., lung cancer, breast cancer). Moreover, certain diseases may be perceived as being more directly tied to an individual's behaviors, which may suggest specific behavioral changes that may aid in controlling or managing the illness in as far as the behaviors can reduce symptom severity and slow disease progression. In addition to the type of illness or disease, social comparison processes may operate differently depending on other characteristics or features of the disease (e.g., severity, clinical prognosis, treatment options, perceived control over managing symptoms). Social comparisons may also serve different functions and result in different outcomes depending on the stage of diagnosis or treatment (e.g., time of initial diagnosis, ten years since diagnosis) and the patient's current health status. For example, research has shown that individuals who were previously treated for cancer responded differently to social comparison information on coping strategies and negative emotions, such that self-reported quality of life varied by the patient's current physical health and the individual's sensitivity to social comparison (Brakel et al., 2012).

Much work on social comparison has evaluated the roles of upward and downward comparisons in relation to individuals' attitudes, perceptions of health or illness, and behaviors. However, comparative optimism has been given particular attention in the literature given its role in illness and risk perception. Comparative optimism, which is well documented in the literature on social comparison, refers to an individual's belief of being at lower risk for a health-related issue or other negative event or the belief of being better at something than his or her peers (Harris, Griffin, & Murray, 2008). In some ways this relates to the lay notion that adolescents tend to believe that a negative outcome (e.g., contracting a sexually transmitted infection from unprotected sex) will not happen to them; instead adolescents tend to perceive that others, who are presumably unlike them, are more at risk for experiencing the negative outcome. Comparative optimism does not account for the degree of accuracy in the perception of risk. At times, the perception of being at lower risk may be accurate; however, when the perception of being at lower risk is inaccurate, this results in bias or unrealistic optimism (Suls et al., 1997).

Social Comparison and Risky Behaviors

Social Comparison as a Predictor of Risk

People may compare themselves to others with respect to virtually any attribute or behavior. Comparisons can be made with varying levels of human interaction such that the target of the comparison may be identified in the media or it may be the person sitting next to someone on the bus. Given the far-reaching applications of social comparison theory, a large body of research has evaluated social comparison processes in the areas of health and risk behaviors. Notably, social comparison is implicated in specific models of health risk behavior such as the prototype willingness model (see Gibbons et al., 2015 for a review). At the heart of this theory is the notion that prototypes, or social images of the typical person who engages in a health risk behavior, influence behavior through a social comparison process (Gibbons et al., 2015).

Although too vast to review in detail, there has been extensive work examining social comparison processes in the areas of unhealthy eating behaviors, body image, and body dissatisfaction (Arigo, Schumacher, & Martin, 2014; Fitzsimmons-Craft et al., 2015). Other health and risk behaviors for which social comparison processes have been evaluated are sexual behavior (Fielder, Walsh, Carey, & Carey, 2013) and substance use (Litt et al., 2014).

Social Comparison Orientation as a Moderator of Risk Cognitions

People vary in the extent to which they compare themselves to others as a way to evaluate their own attitudes and behaviors, and studies have found that links between risk cognitions and behaviors vary according to the degree to which individuals engage in social comparison (Litt et al., 2014). Social comparison orientation pertains to the extent to which individuals compare their own attitudes and behaviors to that of their family and/or peers (Litt et al., 2014; Litt, Lewis, Stahlbrandt, Firth, & Neighbors, 2012).

Social comparison orientation has been shown to moderate the link between same-sex descriptive drinking norms (i.e., perceived quantity and frequency of alcohol consumption of the typical male/female) and alcohol-related consequences, but not alcohol use, among college students (Litt et al., 2012). Among those higher in social comparison, descriptive norms for drinking were positively associated with alcohol-related negative consequences; among those lower in social comparison, the association between descriptive norms for drinking and consequences was not significant. More recent research found that social comparison orientation moderated the effects of perceived friend use and sibling use on change in alcohol use over three years such that adolescents who reported engaging in social comparison more often reported greater increases in alcohol use when perceived friend use or sibling use was high (Litt et al., 2014). Future research is warranted on the manner in which social comparison processes may modify the effects of other known risk factors, including but not limited to perceived social norms.

Social Comparison and Intervention

Relatively little research has *formally* evaluated social comparison theory in the context of health behavior interventions. It should be noted that many constructs such as social norms or unrealistic optimism are directly implied and implicitly connected to social comparison theory but are not central to social comparison theory per se. Moreover, most conceptualizations of peer influence require social comparisons as an integral part of the influence process. In order

for others to have indirect influence on us (as opposed to direct requests to engage in a particular behavior), we must consider their expectations. Thus, while there are a few formal studies focusing directly on social comparison theory-based interventions, it has been suggested that there are few studies related to social influence and health behavior that are not related to social comparison theory. For this chapter we focused on interventions in which social comparison interventions were formally evaluated or in which social comparison orientation was evaluated as a moderator of intervention efficacy.

In another study, Mahler, Kulik, Gerrard, and Gibbons (2010) examined the impact of adding upward or downward social comparison information on the efficacy of an appearance-based sun protection intervention (UV photos and photoaging information). College students were randomly assigned to one of four conditions: control, intervention, intervention plus upward social comparison, and intervention plus downward social comparison. The results demonstrated that all those who received the basic UV photo/photoaging intervention reported greater perceived susceptibility to photoaging, less favorable tanning cognitions, and greater intentions to protect themselves from the sun relative to controls. Of specific interest to social comparison, while the basic intervention increased sun protective behavior during the subsequent 5 weeks relative to controls, the addition of downward comparison information negated this benefit. Moreover, upward comparison information produced sun protection levels that were only slightly (and nonsignificantly) greater than in the basic intervention condition. Thus, these findings did not support an addition of a social comparison component.

As mentioned above, social comparison orientation has been evaluated as a moderator of an intervention aimed to reduce high-risk drinking and negative consequences (Carey, Henson, Carey, & Maisto, 2007). Findings indicated that the personalized feedback intervention that included a normative comparison component was less efficacious for individuals who were higher in social comparison, which suggests that individuals who are higher in social comparison are strongly influenced by the perceived behavior of others. In addition, as Carey et al. (2007) found, the influence of perceived behavior may be more difficult to change for those higher in social comparison. One reason this may be is that individuals who are higher in social comparison may be more aware of the health and/or behaviors around them. Thus, when taking into consideration the normative comparison information designed to initiate social comparison in an intervention study, those higher in social comparison orientation may also have more instances to rely on. Having more sources of information to draw from may lead those higher in social comparison to be defensive or to disregard the normative information presented in an intervention.

Based on the few studies in which social comparison theory has been formally evaluated with intervention studies, findings provide mixed support. Of importance, these studies are across various health behaviors. Future research is needed to examine multiple social comparison-based interventions for one health behavior.

Future Directions

Clearly, social comparison theory has greatly enhanced our knowledge about a number of important health-related processes and outcomes. Through the years, social comparison theory has grown from a relatively narrowly defined theory into one that sheds light on a broad cross section of human behavior including health behaviors. The application of social comparison theory to health is continuously expanding, with more and more issues being explored from a social comparison perspective. Despite the huge growths in this area of inquiry, there are still several

issues that remain to be fully resolved and as such should be the focus of future research. One promising area of inquiry comes out of the dual-processing literature (Gibbons et al., 2015), specifically examining the extent to which social comparisons are the result of more heuristic and affective processing versus reasoned and thoughtful processing. Although there is preliminary work indicating that social comparison moderates health cognitions (e.g., descriptive norms, prototypes, willingness) within the more socially reactive, heuristic, and affective processing pathways of dual-processing models such as the prototype willingness model (Gibbons et al., 2015; Litt et al., 2014; Stock, Gibbons, Beekman, & Gerrard, 2015), the process through which social comparison operates in the greater scheme of decisions regarding health behaviors and health risk behaviors still deserves attention. It is also important that research bridging social psychology and neuroscience approaches be conducted. We anticipate work in the future that will examine brain activity during different types of comparison processes and opportunities (e.g., can reactions to upward versus downward comparison be detected at a neural level?). Are different parts of the brain activated depending on who the comparison target is or what health dimension is being compared, or whether the comparison is made in relation to health risk or health protective behaviors. An additional important avenue for future research is directly related to the functionality of social comparison as a means to effect positive changes for health-related behavior. Specifically, there is a need for research that looks at the motivational effects of induced comparison with certain targets, for example, healthy eaters or light drinkers, to determine if social comparison can be harnessed in such a way to directly impact health-related outcomes. Finally, social comparison has implications for individual-level interventions that include both personal and comparison-based risk information (Stock et al., 2015). In particular, interventions that offer personalized feedback, including social norms, may be more effective for individuals who tend to compare their own attitudes and behaviors to that of others. Future research should continue to examine the ways in which social comparison is integrated into interventions as well as whether interventions should be targeted to those most likely to make social comparisons on health dimensions. Overall, we remain optimistic that more light will be shed on many unresolved issues in the coming years, and we see an active future ahead for social comparison theory and its applications to health behavior.

Author Biographies

Dana M. Litt, PhD, is an assistant professor at the University of Washington in the Department of Psychiatry and Behavioral Sciences. Dr. Litt's primary research interests lie in examining social psychological principles as applied to the etiology and prevention of health-related risk behaviors. Her research addresses questions related to the utility of including socially based variables in prevention programming for substance use, particularly with respect to social comparison, social images, and social norms.

Anne M. Fairlie, PhD, is an acting assistant professor in the Department of Psychiatry and Behavioral Sciences at the University of Washington. Dr. Fairlie's research focuses on psychosocial factors influencing substance use among young adults. In particular, she is interested in how occasion-specific psychological and contextual factors predict day-to-day variations in alcohol use, including extreme drinking and negative consequences.

Melissa A. Lewis, PhD, is a full professor in the Department of Psychiatry and Behavioral Sciences at the University of Washington. Dr. Lewis' research interests lie in examining social psychological principles in broadly defined health-related behaviors. She studies social and motivational mechanisms involved in etiology and prevention of addictive and high-risk

behaviors (e.g., drinking, risky sexual behavior, hooking up). She has expertise in personalized feedback interventions aimed at reducing drinking and related risky sexual behavior.

Michelle L. Stock, PhD, is an associate professor in the Department of Psychology at The George Washington University. She received her PhD in psychology from Iowa State University in 2007. Dr. Stock's program of research includes a focus on the application of the prototype willingness model and social comparison processes to provide a framework for understanding cognitive (heuristic and reasoned), affective, and situational factors that influence risky health decisions.

References

- Arigo, D., Schumacher, L., & Martin, L. M. (2014). Upward appearance comparison and the development of eating pathology in college women. *International Journal of Eating Disorders*, 47, 467–470.
- Arigo, D., Suls, J. M., & Smyth, J. M. (2014). Social comparisons and chronic illness: Research synthesis and clinical implications. *Health Psychology Review*, 8, 154–214.
- Brakel, T. M., Dijkstra, A., & Buunk, A. P. (2012). Effects of the source of social comparison information on former cancer patients' quality of life. *British Journal of Health Psychology*, 16, 667–681.
- Buunk, B. P., & Gibbons, F. X. (Eds.) (1997). *Health, coping, and well-being: Perspectives from social comparison theory*. Mahwah, NJ: Lawrence Erlbaum.
- Carey, K. B., Henson, J. M., Carey, M. P., & Maisto, S. A. (2007). Which heavy drinking college students benefit from a brief motivational intervention? *Journal of Consulting and Clinical Psychology*, 75, 663–669.
- Festinger, L. (1954). A theory of social comparison processes. *Human Relations*, 7, 117–140.
- Fielder, R. L., Walsh, J. L., Carey, K. B., & Carey, M. P. (2013). Predictors of sexual hookups: A theory-based, prospective study of first-year college women. *Archives of Sexual Behavior*, 42, 1425–1441.
- Fitzsimmons-Craft, E. E., Bardone-Cone, A. M., Wonderlich, S. A., Crosby, R. D., Engel, S. G., & Bulik, C. M. (2015). The relationships among social comparisons, body surveillance, and body dissatisfaction in the natural environment. *Behavior Therapy*, 46, 257–271.
- Gibbons, F. X., Stock, M. L., Gerrard, M., & Finneran, S. (2015). The prototype-willingness model. In M. Conner & P. Norman (Eds.), *Predicting and changing health behaviour: Research and practice with social cognition models* (3rd ed., pp. 189–224). Berkshire, UK: Open University Press.
- Harris, P. R., Griffin, D. W., & Murray, S. (2008). Testing the limits of optimistic bias: Event and person moderators in a multilevel framework. *Journal of Personality and Social Psychology*, 95, 1225–1237.
- Litt, D. M., Lewis, M. A., Stahlbrandt, H., Firth, P., & Neighbors, C. (2012). Social comparison as a moderator of the association between perceived norms and alcohol use and negative consequences among college students. *Journal of Studies on Alcohol and Drugs*, 73, 961–967.
- Litt, D. M., Stock, M. L., & Gibbons, F. X. (2014). Adolescent alcohol use: Social comparison orientation moderates the impact of friend and sibling behaviour. *British Journal of Health Psychology*. doi:10.1111/bjhp.12118
- Mahler, H. I., Kulik, J. A., Gerrard, M., & Gibbons, F. X. (2010). Effects of upward and downward social comparison information on the efficacy of an appearance-based sun protection intervention: A randomized, controlled experiment. *Journal of Behavioral Medicine*, 33, 496–507.
- Mojitabai, R. (2008). Social comparison of distress and mental health help-seeking in the US general population. *Social Science and Medicine*, 67, 1944–1950.
- Mussweiler, T., Rüter, K., & Epstude, K. (2004). The ups and downs of social comparison: Mechanisms of assimilation and contrast. *Journal of Personality and Social Psychology*, 87, 832–844.
- Schokker, M. C., Keers, J. C., Bouma, J., Links, T. P., Sanderman, R., Wolffenbuttel, B. H. R., & Hagedoorn, M. (2010). The impact of social comparison information on motivation in patients with diabetes as a function of regulatory focus and self-efficacy. *Health Psychology*, 29, 438–445.

- Siegel, K., Lekas, H.-M., Schrimshaw, E. W., & Brown-Bradley, C. J. (2011). Strategies adopted by late middle-age and older adults with HIV/AIDS to explain their physical symptoms. *Psychology and Health*, 26, 41–62.
- Stock, M. L., Gibbons, F. X., Beekman, J. B., & Gerrard, M. (2015). It only takes once: The absent-exempt heuristic and reactions to comparison-based sexual risk information. *Journal of Personality and Social Psychology*, 109, 39–52.
- Suls, J., Martin, R., & Leventhal, H. (1997). Social comparison, lay referral, and the decision to seek medical care. In B. Buunk & F. Gibbons (Eds.), *Health and coping: Perspectives from social comparison theory* (pp. 195–226). Mahwah, NJ: Lawrence Erlbaum.
- van der Pligt, J. (1996). Judgment and decision making. In G. Semin & K. Fiedler (Eds.), *Applied social psychology* (pp. 30–64). London, UK: Sage.

Suggested Reading

- Arigo, D., Suls, J. M., & Smyth, J. M. (2014). Social comparisons and chronic illness: Research synthesis and clinical implications. *Health Psychology Review*, 8, 154–214.
- Gibbons, F. X., Stock, M. L., Gerrard, M., & Finneran, S. (2015). The prototype-willingness model. In M. Conner & P. Norman (Eds.), *Predicting and changing health behaviour: Research and practice with social cognition models* (3rd ed., pp. 189–224). Berkshire, UK: Open University Press.
- Mussweiler, T., Rüter, K., & Epstude, K. (2004). The ups and downs of social comparison: Mechanisms of assimilation and contrast. *Journal of Personality and Social Psychology*, 87, 832–844.

Health Effects of Traumatic Events

Judith Andersen¹ and Roxane Cohen Silver²

¹*Department of Psychology, University of Toronto, Mississauga, ON, Canada*

²*Department of Psychological Science, University of California, Irvine, Irvine, CA, USA*

Globally, traumatic stress is quite common. Each day, events such as war, natural disasters, accidents, and murders claim the lives of hundreds of people. Traumatic events are defined as those that threaten serious injury or threat of death to oneself or others or witnessing the death or serious injury of another person. When an individual's personal physical integrity is compromised, such as in the case of sexual assault, it is thought to be traumatic. Traumas can have specific characteristics and may impact individuals in different ways. For example, traumas can be experienced in childhood, adolescence, adulthood, or old age. Traumas can be individually or collectively experienced, and they can be acute (i.e., short term) or chronic.

There are different types of traumas to which people can be exposed. Individual traumas can include sudden and unexpected losses of persons or property. Interpersonal traumas are those that are perpetrated by humans against one another. There are traumas shared by communities, such as gang violence and school shootings. Natural disasters like hurricanes and earthquakes displace thousands of people and can bring suffering, injury, homelessness, and even death to many individuals. Mass traumas such as terrorist attacks are becoming more common around the world. Researchers have posited that traumas may be organized into the following categories: injury and illness (e.g., witnessing or experiencing an accident, serious illness or injury), loss (e.g., death of close other), social environmental events (e.g., financial hardship, homelessness, discrimination, exposure to chemical or biological agents), relationship events (e.g., forced separation from family, marital discord, unwanted pregnancy), natural disaster (e.g., flood, fire, earthquake), interpersonal violence (e.g., rape, sexual or physical abuse), and collective violence (e.g., loss of loved one to homicide or suicide, witnessed violence between parents or adults, war exposure) (see e.g., Blum, Silver, & Poulin, 2014).

Within the last 50 years, healthcare providers working with individuals who previously experienced trauma, such as war veterans and survivors of childhood maltreatment, began to notice a link between trauma experiences and physical health problems. Researchers began to investigate how traumatic experiences may lead to chronic disease and health ailments over time.

We now understand that there are a number of direct and indirect pathways by which traumatic experiences may lead to medical ailments and chronic disease conditions.

For example, trauma may directly impact health through injury or death from the experience itself, such as in the case of physical abuse or war wounds. Alternatively, the experience of trauma can stimulate stress processes that, over time, are maladaptive to physical health. Developments in neuroscience and neurobiology have shown that the internal, physical pathways by which traumatic experiences can lead to physical health problems include the possibility that an individual's stress response may become more sensitive, resulting in problems regulating blood pressure, glucose metabolism, the immune system, and even gene expression. It is also possible that ongoing disruption in core biological processes such as stress hormones and cardiovascular function can lead to chronic disease conditions over time, especially if the individual does not seek medical care.

McEwen (2002) reviewed the process by which the experience of severe stress impacts the body. Specifically, when encountering severe stress, the body responds by activating a cascade of neurochemical reactions involving the sympathetic branch of the autonomic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis. These activating chemicals stimulate the liver to produce high amounts of glucose (high blood sugar) and directly speed up heart rate and respiration. While these processes are adaptive when stressors are short term and resolved quickly, it is clear that if the stress response is chronically activated or unable to shut off, individuals are at risk of diabetes (high blood sugar), as well as respiratory, immune, digestive, and cardiovascular problems. Long-term cardiovascular activation is associated with high blood pressure and the possible development of atherosclerosis. Further, these core physiological systems work synergistically, and their disruption is thought to contribute to many comorbid diseases and even early mortality.

There are also indirect pathways by which trauma exposure has been shown to impact physical health. The most common are behavioral and psychological mechanisms. For example, often when an individual experiences a traumatic stressor, it is unexpected and overwhelming. Many people do not have the coping resources necessary to manage the emotional distress or life disruption associated with the event. Therefore, as a coping strategy, individuals may engage in negative health behaviors such as poor sleep habits, poor diet, excessive alcohol or drug use, or smoking. Researchers have demonstrated that negative health behaviors are related to many physical health problems and chronic conditions, including obesity, cancer, heart disease, pain, and other health conditions.

Following a traumatic experience, many individuals may also experience mental or emotional distress such as anxiety or depressive symptoms. While most individuals recover their equilibrium in the months following the experience, some individuals develop more severe symptoms of clinically diagnosable mental health conditions. Anxiety disorders such as post-traumatic stress disorder (PTSD) and mood disorders are common mental health conditions associated with traumatic exposure. Decades of research have demonstrated that the symptoms of anxiety and depressive disorders are associated with disruptions in the sympathetic nervous system and the HPA axis (Schnurr & Green, 2004). Specifically, PTSD is associated with high cardiovascular reactivity to stress and disrupted levels of cortisol, a central stress hormone in the HPA axis, which is implicated in many organic and somatic disease conditions. Specific symptoms of PTSD, such as hyperarousal, are associated with repeated activation of the cardiovascular system, which is associated with high blood pressure, atherosclerosis, hypercoagulation, and cardiac arrhythmias that may result in myocardial infarction. Depressive symptoms such as apathy and exhaustion are related to disruption in HPA functioning, specifically cortisol and the corticotrophin-releasing hormone (CRH). Further, depression is

associated with disruptions in the immune system due to HPA activity and maladaptive levels of cortisol. Specifically, researchers posit that specific immune chemicals, called cytokines, are more active in depressed individuals (Loftis, Huckans, & Morasco, 2010). Cytokines signal “sickness behavior” by causing the skin and body to feel pain and exhaustion in order to stimulate the individual to slow down and let the immune system identify and manage the invader. However, in the case of depression, it is not a bacterial or viral infection but rather emotional distress that is related to disruptions in cortisol that further stimulate cytokine activation. The resulting pain and tiredness are symptoms that further exacerbate feelings of depression.

Health problems stemming from traumatic exposures may differ depending on the type and length of exposure. For example, researchers studying the impact of acute trauma, such as exposure to the September 11th terrorist attacks, report a rise in risk of cardiovascular ailments in the 3 years following the attacks, with the greatest risk experienced by individuals who reported ongoing worry and anxiety about future attacks (Holman et al., 2008). Adolescents who have been bullied and harassed at school are more likely to report somatic complaints with no known cause (e.g., headaches, pain, gastrointestinal distress) and poorer overall general health. In the above examples, the mechanisms that seem to best explain both cardiovascular ailments and somatic complaints are the repeated activation of the stress response, colloquially known as the “fight-or-flight” response. This response is accompanied by emotions such as anger and fear and physical reactions such as an increase in breathing and heart rate. Further, digestion and immune processes are halted during fight or flight. Therefore, general gastrointestinal distress, pain, somatic complaints such as frequent colds and flus, and cardiovascular ailments like high blood pressure can result from frequent stress responses.

Some types of traumatic experiences are associated with severe health problems that occur in multiple systems of the body. For example, experiencing violence or childhood maltreatment such as physical and sexual abuse or neglect is considered particularly damaging for long-term health. The health effects of exposure to violence have also been studied in depth by researchers examining the experiences of combat veterans. Pizarro, Silver, and Prause (2006) found that among combat soldiers, exposure to trauma (e.g., witnessing death or dismemberment, losing friends or family in battle, fearing for one’s life, killing other people) was directly related to physician-diagnosed cardiovascular disease and gastrointestinal ailments in later life. The age at which the soldiers were exposed to these traumatic events was also important to consider. The youngest soldiers who witnessed the most deadly combat had shorter lifespans, despite surviving the war itself.

Age at exposure to trauma is an important variable for understanding why childhood maltreatment is particularly detrimental for health. Childhood trauma has been linked to autoimmune conditions, cancer, heart disease, and sexual and reproductive health problems in later life. Scientists who study childhood development explain how the effects of childhood trauma may be so pernicious based on a theory of “biological imbedding.” During childhood and adolescence, trauma may alter the shape and function of biological systems as they develop. For example, trauma may sensitize the HPA axis that impacts stress hormone function, stress response reactivity, and emotional competence. The development of brain areas associated with anxiety and depression, memory and attention, and attachment and social skills may also be negatively impacted by childhood trauma. Taken together, the adaptations the body is forced into making in response to childhood trauma exposure become maladaptive for health and functioning over the life course. Researchers conducting a study called the Adverse Childhood Experiences (ACE) research project (e.g., Felitti et al., 1998) interviewed thousands of adult survivors of childhood maltreatment and found a dose–response relationship between the number of adverse childhood experiences and both health risk behaviors and chronic disease.

These researchers report that individuals who experienced four or more adverse childhood experiences were two times more likely to report ischemic heart disease, any cancer, and stroke.

While experiencing early, violent, or chronic trauma has clear implications for health, researchers have found that exposure to trauma does not always negatively impact health in a linear relationship. There are indications that there may be a curvilinear effect of trauma exposure on health (Seery, Holman, & Silver, 2010). Researchers uncovered nonlinear patterns by examining the health of individuals exposed to a collective trauma in adulthood. Results of this study revealed that individuals who had experienced one or two events, such as the death of a loved one or divorce, before the mass trauma fared better than individuals who had never experienced any negative events and those who had previously experienced frequent or severe traumas. The authors explain these findings in light of a human's propensity to seek coping resources and support in order to survive during times of stress. Those who had prior exposure to one or few traumas had the opportunity to garner social support and may have enhanced their confidence in their ability to cope during adversity. Those who had never experienced any trauma or who had experienced chronic or severe trauma may have been less able to garner the needed supports and coping resources.

The good news is that because clinicians and researchers have discovered both the indirect and direct links between trauma and physical health, there are treatments that help people recover from traumatic experiences and avoid chronic disease conditions and negative physical health symptoms. Health psychologists have worked to design interventions that reduce the likelihood that someone will become ill following trauma and approaches within a therapeutic context that improve health when an individual has already begun to experience physical health problems. Health interventions target psychosocial processes, such as negative mood, catastrophic thinking, excessive anxiety, depression, and worry. Research has shown that psychosocial processes and cognitions are directly linked to an individual's neuroendocrine and immune system and can exert negative and positive responses. Therapies can be divided into several categories and may be recommended to a patient based on his or her symptoms and preferred method of treatment. For example, there are cognitive behavioral stress management techniques and techniques that directly target maladaptive physiology. The National Center for PTSD is a repository of information about trauma and treatment. Recommended treatments for trauma include cognitive processing therapies and prolonged exposure therapy. Other therapeutic treatment options for trauma include approaches that directly target symptoms of stress, such as relaxation, meditation, emotional disclosure, and biofeedback. Sleep hygiene and substance abuse counseling may also be necessary if an individual is struggling in these domains. It is thought that effective therapies for trauma also improve health because they target both maladaptive coping behaviors and exaggerated stress reactivity that, over time, contributes to disease processes.

Social support and positive social interactions have been shown to be extremely helpful in preventing health problems following trauma and as a treatment intervention after health symptoms arise. For example, social support group therapy has been shown to improve the quality of life of cancer patients and even extend their lives. Researchers examining genetic factors in aging and illness have found that individuals in high stress environments and those experiencing traumatic stress are more likely to suffer from premature aging and health problems. The premature aging is demonstrated by the rapid shortening of their telomeres, the caps at the end of a person's chromosomes, which fray as a person ages. A therapeutic intervention to slow the process of premature aging includes social support groups specifically targeted to help individuals with the stress they are experiencing. Positive support during and following trauma also helps individuals to cope with traumatic stress without engaging in

negative health behaviors that increase the risk of disease, such as drug and alcohol use or overeating.

In conclusion, researchers have demonstrated that trauma exposure is associated with an increased risk of physical health problems across the lifespan. Trauma exposure may strike individuals or may be experienced collectively by a population. Researchers have shown multiple pathways by which trauma exposure can raise health risks, such as through increased stress responses or immune disruption. Fortunately, factors such as social support and healthy living may protect individuals against the deleterious effects of trauma on health.

Author Biographies

Judith Andersen is an assistant professor in the Department of Psychology and Affiliated Faculty of Medicine at the University of Toronto, Mississauga. She is a health psychologist who specializes in the psychophysiology of stress and stress-related mental and physical health issues.

Roxane Cohen Silver is a professor of psychology and social behavior, public health, and medicine at the University of California, Irvine. Trained as a social psychologist, her research examines individual and community responses to stressful life experiences, including collective traumas such as terrorist attacks, war, and natural disasters.

References

- Blum, S., Silver, R. C., & Poulin, M. J. (2014). Perceiving risk in a dangerous world: Associations between life experiences and risk perceptions. *Social Cognition*, 32, 297–314.
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245–258.
- Holman, E. A., Silver, R. C., Poulin, M., Andersen, J., Gil-Rivas, V., & McIntosh, D. N. (2008). Terrorism, acute stress, and cardiovascular health: A 3-year national study following the September 11th attacks. *Archives of General Psychiatry*, 65, 73–80.
- Loftis, J. M., Huckans, M., & Morasco, B. J. (2010). Neuroimmune mechanisms of cytokine-induced depression: Current theories and novel treatment strategies. *Neurobiology of Disease*, 37, 519–533.
- McEwen, B. S. (2002). The neurobiology and neuroendocrinology of stress: Implications for post-traumatic stress disorder from a basic science perspective. *Psychiatric Clinics of North America*, 25, 469–494.
- Pizarro, J., Silver, R. C., & Prause, J. (2006). Physical and mental health costs of traumatic war experiences among Civil War veterans. *Archives of General Psychiatry*, 63, 193–200.
- Schnurr, P. P., & Green, B. L. (2004). *Trauma and health: Physical health consequences of exposure to extreme stress*. Washington, DC: American Psychological Association.
- Seery, M. D., Holman, E. A., & Silver, R. C. (2010). Whatever does not kill us: Cumulative lifetime adversity, vulnerability, and resilience. *Journal of Personality and Social Psychology*, 99, 1025–1041.

Suggested Reading

- Charney, D. S. (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *American Journal of Psychiatry*, 161, 195–216.

- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *The New England Journal of Medicine*, 338, 171–179.
- Schnurr, P. P., & Green, B. L. (2004). *Trauma and health: Physical health consequences of exposure to extreme stress*. Washington, DC: American Psychological Association.

Health Implications of Gratitude

Kristin Layous

Department of Psychology, California State University East Bay, Hayward, CA, USA

Most of the world's cultural, religious, and philosophical traditions celebrate gratitude as a virtue. Supporting the age-old wisdom about the importance of gratitude, accumulating empirical evidence demonstrates that gratitude is related to many social and psychological benefits. Specifically, cross-sectional, longitudinal, and experimental evidence reveals that a habit of gratitude is associated with greater well-being, more prosocial behavior, and better relationships (Wood, Froh, & Geraghty, 2010). In addition to these social psychological benefits, many researchers also suggest a link between gratitude and physical health. This entry reviews the literature on gratitude and psychological, social, and physical health, noting some complications and limitations as well as some promising findings.

Defining Gratitude

One confusing aspect of the gratitude literature is the multiple ways in which gratitude can be defined. Gratitude is a habit or trait (i.e., one's general tendency to be grateful), a state or emotion (i.e., a momentary feeling of gratitude), or an expression (i.e., one actively expresses gratitude verbally or in writing). Furthermore, some research focuses on gratitude as a response to a benefit received from another person (i.e., benefit-triggered gratitude; McCullough, Kilpatrick, Emmons, & Larson, 2001) or nonperson entity (e.g., God or nature; Emmons & McCullough, 2003), and other research uses the umbrella term "appreciation" to represent feelings of gratitude that do not have a clear source (e.g., a good night's rest). This entry covers all of these types of gratitude but specifies which type of gratitude is targeted in each study described.

Defining Health

Health can also be defined and measured in myriad ways. The World Health Organization (WHO) defines health as consisting of physical, mental (or psychological/subjective), and

social well-being. This broad conception of health is helpful when considering the potential role of gratitude in people's lives. Indeed, gratitude is related to all three aspects of health specified by the WHO, and many posit that although gratitude may act directly on physical health, it more likely acts indirectly by strengthening social and psychological well-being or by stimulating health-promoting behaviors (e.g., Hill, Allemand, & Roberts, 2013; Wood et al., 2010).

Physical health too may be defined in many ways—as the absence of disease, the absence of risk factors for disease, the ability to perform necessary daily activities, or the longevity of one's life. Consequently, researchers use many different methods to measure physical health, with some relying on self-reported physical health, which is highly related to psychological or subjective well-being (Friedman & Kern, 2014). Given the different ways in which physical health can be assessed, I try to be clear about which studies include what measures, and, even when a correlation between gratitude and physical health is reported, I allow for the possibility that another intermediary mechanism (e.g., psychological well-being) could be at play.

Gratitude and Positive Affect

Gratitude is itself a positive emotion, which could mean that the entire body of literature linking positive emotions to health can be applied to the relationship between gratitude and health (e.g., Pressman & Cohen, 2005). Indeed, the state of gratitude is often found among the many emotions included in positive emotion composites, and gratitude may behave similarly to those other positive emotions and has the same effect on health and health behaviors. However, gratitude may also have a distinct effect on health that is not explained by general positive affect. Truthfully, gratitude and general positive affect are not easily disentangled, but this chapter will consider those instances in which gratitude was uniquely studied and related to health in some way. For more information on the link between positive affect and health, see Jenkins, Acevedo, and Pressman in this volume.

Gratitude and Psychological Well-Being

The frequency with which people experience positive emotions is just one part of their psychological well-being. Other parts include the infrequency with which they experience negative emotions and their global sense that their life is satisfying and meaningful (Diener, Suh, Lucas, & Smith, 1999; Ryff, 1989). Importantly, trait gratitude is positively related to positive affect, global well-being, vitality, optimism, and hope and negatively related to anxiety, depression, envy, and neuroticism (Froh, Yurkewicz, & Kashdan, 2009; McCullough, Emmons, & Tsang, 2002). Among college students, gratitude at the beginning of the semester predicts lower likelihood of depression and stress at the end of the semester, controlling for baseline levels (Wood, Maltby, Gillett, Linley, & Joseph, 2008). The reverse direction of baseline stress and depression predicting gratitude at the end of the semester was not supported. Thus, these findings suggest that trait gratitude is not only related to well-being but also predictive of future well-being.

In a stronger test of the causal influence of gratitude on well-being, multiple randomized controlled experiments have now found that prompting people to express gratitude over time—either by counting blessings or writing gratitude letters—can shift reports of trait gratitude (e.g., Emmons & McCullough, 2003; Kerr, O'Donovan, & Pepping, 2014) and boost psychological well-being (e.g., Davis et al., 2016; Emmons & McCullough, 2003).

Boosting psychological well-being could also have an impact on physical health. Indeed, meta-analyses and reviews have found that psychological well-being is predictive of short-term outcomes like immune system response (Howell, Kern, & Lyubomirsky, 2007) and long-term outcomes like longevity (Chida & Steptoe, 2008; Howell et al., 2007, but see Friedman & Kern, 2014). In addition, gratitude could also affect physical health by decreasing stress, which has an adverse relationship with physical health.

Gratitude and Social Relationships

Trait gratitude is also related to qualities that promote healthy relationships such as empathy, perspective taking, and prosocial behavior (McCullough et al., 2002). Theory suggests that gratitude helps people recognize the good things people have done for them and motivates them to either repay their benefactor or pay the kind act forward, which strengthens relationships (McCullough et al., 2001). Indeed, trait gratitude is related to general perceptions of social support (Froh et al., 2009; Wood et al., 2008), and even momentary instances of gratitude seem important for relationships, as people who report feeling grateful toward their partner on one day also feel more satisfied with their relationship and more connected to their partner on the following day (Algoe, Gable, & Maisel, 2010). Furthermore, feelings of gratitude on one day also predict the degree to which people are responsive to their partner's needs on the following day and committed to their relationship the following day and 9 months later (Gordon, Impett, Kogan, Oveis, & Keltner, 2012).

Experimental evidence suggests that the link between gratitude and healthy relationships may be causal. For example, in one experiment, people randomly assigned to write gratitude letters felt more globally connected to others than those assigned to a control group that did not complete the gratitude activity (Kerr et al., 2014). In another experiment, people assigned to express gratitude to a close friend reported more positive perceptions of their friends than those assigned to neutral or positive comparison groups (Lambert & Fincham, 2011).

That gratitude relates to, predicts, and causes beneficial effects on relationships and perceptions of social support has implications for understanding the link between gratitude and physical health. The association between social support and psychological and physical well-being has been well documented, with prospective and experimental evidence suggesting a causal relationship (e.g., Cohen & Wills, 1985). Theory suggests that social support may buffer people from the deleterious effects of stress on psychological and physical well-being and that being socially integrated (i.e., having relationships and social roles in one's life) may promote positive states including mental health and health-promoting physiological responses. Accordingly, gratitude could also affect physical health via improvements in perceptions of social support and relationship quality.

Gratitude and Physical Health and Health Behaviors

Although the exact pathways by which gratitude may affect physical health and health-promoting behaviors are still largely theoretical, accumulating evidence has demonstrated a relationship between gratitude and physical health. One study found that trait gratitude and self-reported physical health were positively correlated in adults, even when controlling for key personality traits, and suggestive cross-sectional evidence indicated that psychological health, healthy activities, and willingness to seek help for health concerns mediates the pathway

between gratitude and self-reported physical health (Hill et al., 2013). Similarly, a study in adolescents found a correlation between trait gratitude and reduced physical symptoms, which cross-sectional evidence suggested was explained by relational fulfillment (Froh et al., 2009). In one of the few studies that included objective measures of health, researchers found that feelings of appreciation are related to “coherence” or entrainment among the oscillatory systems in the autonomic nervous system, that is, when people feel appreciative, the body’s heart, respiratory, and blood pressure oscillations synchronize, which is associated with efficiency in bodily processes (McCraty & Childre, 2004).

As mentioned before, gratitude could improve physical health by inhibiting negative attitudes and emotions, by promoting psychological well-being and health-promoting behaviors, or by strengthening perceived social support (Wood et al., 2010). Another way that gratitude could improve physical health is by improving sleep quality and duration. Indeed, in a community sample, trait gratitude was related to greater subjective sleep quality and duration, less time to fall asleep, and less daytime dysfunction, even after controlling for personality (Wood, Joseph, Lloyd, & Atkins, 2009). Cross-sectional evidence suggested that the relationship between gratitude and better sleep was explained by more positive and less negative thoughts at bedtime (Wood et al., 2009).

The link between gratitude and better sleep is important in understanding the relationship between gratitude and physical health because a vast body of research links sleep duration and quality to psychological and physical well-being and even mortality (Cappuccio, D’Elia, Strazzullo, & Miller, 2010). As one example, in a prospective study of college students, sleep duration and quality predicted better subjective psychological and physical well-being, as well as better grades, even after controlling for demographics and daytime functioning at baseline (Wong et al., 2013). Of course, many factors are related to sleep quality and duration, such as age, socioeconomic status, alcohol consumption, and weight, but if increasing grateful feelings before bed could improve sleep relative to one’s baseline levels, gratitude could indirectly improve physical health. Indeed, one experiment found that patients with neuromuscular disorders who were prompted to list what they were thankful for once per day for 3 weeks saw improvements in sleep duration and felt more refreshed upon waking than patients in a control group who did not complete the gratitude activity (Emmons & McCullough, 2003, Study 3).

Experimental evidence linking gratitude directly to physical health and health behaviors is currently thin, but two other studies are relevant (in addition to Emmons & McCullough, 2003, Study 3 just listed). In one experiment, college students were randomly assigned to either list their blessings (the gratitude condition), list their hassles, or list life events once per week for 10 weeks. Those assigned to the gratitude group reported fewer physical health symptoms than either comparison group and more hours of exercise than the hassles group (Emmons & McCullough, 2003, Study 1). Additionally, hypertensive African Americans who were randomly assigned to list their blessings for 10 weeks (the gratitude condition) decreased in systolic blood pressure over time (Shipon, 2007).

All three of the abovementioned experiments showed significant increases in trait gratitude among people in gratitude versus comparison conditions; however, the first two experiments did not test whether these increases in gratitude mediated the relationship between the expression of gratitude and the changes in physical health (Emmons & McCullough, 2003, Study 1 & Study 3), and the second experiment tested the relationship but found that changes in gratitude were not related to changes in blood pressure (Shipon, 2007), leaving the mechanisms of the change up for debate. Future research would do well to test the mechanisms through which gratitude interventions improve physical health to inform a causal model. Furthermore, to address the overlap between self-reported health and psychological well-being, future

studies of gratitude and gratitude interventions should strive to include objective measures of physical health that could disentangle gratitude's influence on psychological health from its influence on physical health.

Moderators of the Link Between Gratitude and Health

An important caveat is that trait gratitude may show different patterns in different people, and similarly, gratitude interventions may have different effects on different people (see Lyubomirsky & Layous, 2013). For example, the relationship between gratitude and self-reported physical health increases with age (Hill et al., 2013), and evidence suggests that the effects of psychological well-being interventions (including gratitude) are stronger in older adults (Sin & Lyubomirsky, 2009). Regarding gender differences, one study found that men were less likely to feel and express gratitude and received fewer benefits from gratitude (Kashdan, Mishra, Breen, & Froh, 2009). Another study in adolescents also found that girls report marginally more trait gratitude than boys, but largely did not find gender differences in how much girls or boys benefit from gratitude (Froh et al., 2009). Thus, some evidence suggests that women may feel and express gratitude more than men and may receive more benefits from gratitude, but further investigation is necessary. Some evidence suggests that gratitude interventions are particularly successful in raising psychological well-being among people low in trait gratitude (e.g., Harbaugh & Vasey, 2014), suggesting that if men are lower in trait gratitude, they may be particularly primed to benefit from a gratitude intervention.

Lastly, one quasi-experiment found that writing gratitude letters once per week for 6 weeks (versus engaging in a neutral writing activity) raised psychological well-being in a sample of US college students but trended toward decreasing well-being in a sample of South Korean college students (Layous, Lee, Choi, & Lyubomirsky, 2013). The authors thought the difference between cultures may be due to South Koreans' feeling less comfortable receiving social support than their US counterparts. Another possibility is that Asian cultures already emphasize gratitude and therefore may be approaching an upper limit, such that expressing yet more gratitude does not confer additional benefits. This and other potential moderators of the effect of gratitude on various types of health should continue to be explored.

Conclusion

In sum, the research linking gratitude to psychological well-being and positive social relationships is strong and even causal, whereas the research linking gratitude and physical health and health behaviors is thus far limited but suggestive. A review of all articles published in the three leading health psychology journals found that only 3% of all articles published included "positive psychological constructs" such as optimism, happiness, and emotion-focused coping (Schmidt, Raque-Bogdan, Piontkowski, & Schaefer, 2011). That percentage seems small in and of itself, but another telling fact is that gratitude was not even on their list of searched terms, speaking to its relative lack of consideration thus far in the health psychology field.

Future research should include prospective studies of trait gratitude and physical health, as well as randomized controlled studies of gratitude interventions that explore the mechanisms that drive changes in physical health. Because of gratitude's well-documented effects on psychological well-being and relationships, and because of the well-documented association between psychological well-being, relationships, and physical health, increasing gratitude has

the potential to affect physical health, once again supporting Cicero's contention that "gratitude is not only the greatest of virtues, but the parent of all others."

Author Biography

Kristin Layous (PhD, University of California, Riverside; MA, Ohio State University; BA, University of California, Santa Barbara) is an assistant professor of psychology at California State University, East Bay. Her research explores the processes by which people can become happier and healthier through practicing simple positive activities (e.g., expressing gratitude or performing kind acts). During a postdoc at Stanford University, she also investigated how psychological interventions can enhance weight-loss efforts and reduce negative stereotypes surrounding obesity.

References

- Algoe, S. B., Gable, S. L., & Maisel, N. C. (2010). It's the little things: Everyday gratitude as a booster shot for romantic relationships. *Personal Relationships, 17*(2), 217–233. doi:10.1111/j.1475-6811.2010.01273.x
- Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. *Sleep, 33*(5), 585–592.
- Chida, Y., & Steptoe, A. (2008). Positive psychological well-being and mortality: A quantitative review of prospective observational studies. *Psychosomatic Medicine, 70*, 741–756. doi:0033-3174/08/7007-0741
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*(2), 310–357. doi:10.1037/0033-2909.98.2.310
- Davis, D. E., Choe, E., Meyers, J., Wade, N., Varjas, K., Gifford, A., ... Worthington, E. L., Jr. (2016). Thankful for the little things: A meta-analysis of gratitude interventions. *Journal of Counseling Psychology, 63*, 20–31.
- Diener, E., Suh, E. M., Lucas, R. E., & Smith, H. L. (1999). Subjective well-being: Three decades of progress. *Psychological Bulletin, 125*, 276–302.
- Emmons, R. A., & McCullough, M. E. (2003). Counting blessings versus burdens: An experimental investigation of gratitude and subjective well-being in daily life. *Journal of Personality and Social Psychology, 84*(2), 377–389. doi:10.1037/0022-3514.84.2.377
- Friedman, H. S., & Kern, M. L. (2014). Personality, well-being, and health. *Annual Review of Psychology, 65*, 719–742. doi:10.1146/annurev-psych-010213-115123
- Froh, J. J., Yurkewicz, C., & Kashdan, T. B. (2009). Gratitude and subjective well-being in early adolescence: Examining gender differences. *Journal of Adolescence, 32*, 633–650. doi:10.1016/j.adolescence.2008.06.006
- Gordon, A. M., Impett, E. A., Kogan, A., Oveis, C., & Keltner, D. (2012). To have and to hold: Gratitude promotes relationship maintenance in intimate bonds. *Journal of Personality and Social Psychology, 103*(2), 257–274. doi:10.1037/a0028723
- Harbaugh, C. N., & Vasey, M. W. (2014). When do people benefit from gratitude practice? *The Journal of Positive Psychology, 9*(6), 535–546. doi:10.1080/17439760.2014.927905
- Hill, P. L., Allemand, M., & Roberts, B. W. (2013). Examining the pathways between gratitude and self-rated physical health across adulthood. *Personality and Individual Differences, 54*(1), 92–96. doi:10.1016/j.paid.2012.08.011
- Howell, R. T., Kern, M. L., & Lyubomirsky, S. (2007). Health benefits: Meta-analytically determining the impact of well-being on objective health outcomes. *Health Psychology Review, 1*(1), 83–136. doi:10.1080/17437190701492486

- Kashdan, T. B., Mishra, A., Breen, W. E., & Froh, J. J. (2009). Gender differences in gratitude: Examining appraisals, narratives, the willingness to express emotions, and changes in psychological needs. *Journal of Personality*, 77(3), 1–40. doi:10.1111/j.1467-6494.2009.00562.x
- Kerr, S. L., O'Donovan, A., & Pepping, C. A. (2014). Can gratitude and kindness interventions enhance well-being in a clinical sample? *Journal of Happiness Studies*. doi:10.1007/s10902-013-9492-1
- Lambert, N. M., & Fincham, F. D. (2011). Expressing gratitude to a partner leads to more relationship maintenance behavior. *Emotion*, 11(1), 52–60. doi:10.1037/a0021557
- Layous, K., Lee, H., Choi, I., & Lyubomirsky, S. (2013). Culture matters when designing a successful happiness-increasing activity: A comparison of the United States and South Korea. *Journal of Cross-Cultural Psychology*, 44(8), 1294–1303. doi:10.1177/0022022113487591
- Lyubomirsky, S., & Layous, K. (2013). How do simple positive activities increase well-being? *Current Directions in Psychological Science*, 22(1), 57–62. doi:10.1177/0963721412469809
- McCraty, R., & Childre, D. (2004). The grateful heart: The psychophysiology of appreciation. In R. A. Emmons & M. E. McCullough (Eds.), *The psychology of gratitude* (pp. 230–255). New York, NY: Oxford University Press.
- McCullough, M. E., Emmons, R. A., & Tsang, J. (2002). The grateful disposition: A conceptual and empirical topography. *Journal of Personality and Social Psychology*, 82(1), 112–127. doi:10.1037//0022-3514.82.1.112
- McCullough, M. E., Kilpatrick, S. D., Emmons, R. A., & Larson, D. B. (2001). Is gratitude a moral affect? *Psychological Bulletin*, 127(2), 249–266. doi:10.1037/0033-2909.127.2.249
- Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin*, 131, 925–971.
- Ryff, C. D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology*, 57, 1069–1081.
- Schmidt, C. K., Raque-Bogdan, T., Piontkowski, S., & Schaefer, K. L. (2011). Putting the positive in health psychology: A content analysis of three journals. *Journal of Health Psychology*, 16(4), 607–620. doi:10.1177/1359105310384296
- Shipon, R. W. (2007). Gratitude: Effect on perspectives and blood pressure of inner-city African-American hypertensive patients. *Dissertation Abstracts International: Section B. Sciences and Engineering*, 68(3–B), 1977.
- Sin, N. L., & Lyubomirsky, S. (2009). Enhancing well-being and alleviating depressive symptoms with positive psychology interventions: A practice-friendly meta-analysis. *Journal of Clinical Psychology*, 65(5), 467–487. doi:10.1002/jclp.20593
- Wong, M. W., Lau, E. Y. Y., Wan, J. H. Y., Cheung, S. F., Hui, C. H., & MOK, D. S. Y. (2013). The interplay between sleep and mood in predicting academic functioning, physical health and psychological health: A longitudinal study. *Journal of Psychosomatic Research*, 74, 271–277. doi:10.1016/j.jpsychores.2012.08.014
- Wood, A. M., Froh, J. J., & Geraghty, A. W. (2010). Gratitude and well-being: A review and theoretical integration. *Clinical Psychology Review*, 30(7), 890–905. doi:10.1016/j.cpr.2010.03.005
- Wood, A. M., Joseph, S., Lloyd, J., & Atkins, S. (2009). Gratitude influences sleep through the mechanism of pre-sleep cognitions. *Journal of Psychosomatic Research*, 66(1), 43–48. doi:10.1016/j.jpsychores.2008.09.002
- Wood, A. M., Maltby, J., Gillett, R., Linley, P. A., & Joseph, S. (2008). The role of gratitude in the development of social support, stress, and depression: Two longitudinal studies. *Journal of Research in Personality*, 42(4), 854–871. doi:10.1016/j.jrp.2007.11.003

Suggested Reading

- Emmons, R. A., & McCullough, M. E. (2003). Counting blessings versus burdens: An experimental investigation of gratitude and subjective well-being in daily life. *Journal of Personality and Social Psychology*, 84(2), 377–389. doi:10.1037/0022-3514.84.2.377

- Hill, P. L., Allemand, M., & Roberts, B. W. (2013). Examining the pathways between gratitude and self-rated physical health across adulthood. *Personality and Individual Differences*, 54(1), 92–96. doi:10.1016/j.paid.2012.08.011
- Wood, A. M., Froh, J. J., & Geraghty, A. W. (2010). Gratitude and well-being: A review and theoretical integration. *Clinical Psychology Review*, 30(7), 890–905. doi:10.1016/j.cpr.2010.03.005
- Wood, A. M., Joseph, S., Lloyd, J., & Atkins, S. (2009). Gratitude influences sleep through the mechanism of pre-sleep cognitions. *Journal of Psychosomatic Research*, 66(1), 43–48. doi:10.1016/j.jpsychores.2008.09.002
- Wood, A. M., Maltby, J., Gillett, R., Linley, P. A., & Joseph, S. (2008). The role of gratitude in the development of social support, stress, and depression: Two longitudinal studies. *Journal of Research in Personality*, 42(4), 854–871. doi:10.1016/j.jrp.2007.11.003

The Health Implications of Resilience

Kristen L. Rudd and Tuppett M. Yates

Department of Psychology, University of California, Riverside, , CA, USA

Introduction

Resilience is a developmental process that supports positive or better-than-expected adjustment despite exposure to incontrovertible adversity (Masten, 2014). Two pieces of information are necessary to “diagnose” resilience: (a) the individual must have encountered a clear threat to typical development, and (b) the individual must evidence competent adaptation as evaluated with respect to developmental and cultural expectations. In addition to *resilience* (i.e., positive adjustment in the context of high adversity), development can be characterized by *maladaptation* (i.e., negative adjustment in the context of high adversity), *competence* (i.e., positive adjustment in the context of low adversity), or, somewhat less commonly, *vulnerability* (i.e., negative adjustment in the context of low adversity). However, given that an estimated 64% of adults report exposure to at least one clear and pronounced adverse experience prior to age 18 (Anda et al., 2006), efforts to identify processes that promote positive adaptation in contexts of adversity (i.e., resilience) have significant implications for supporting human health and well-being in childhood and beyond.

Once thought to be an individual capacity for “invincibility” (Werner & Smith, 1982), current theories conceptualize resilience as a process of “ordinary magic” (Masten, 2014). In this view, normative developmental support systems enable individuals to function in developmentally and culturally expected ways despite the disruptive influences of adverse life experiences. Thus, resilience is no longer viewed as an extraordinary capacity of an individual, but rather as the natural expression of ordinary adaptive systems despite extraordinary experiences. Capacities for self-regulation, attachment, and cognitive reasoning comprise core systems that influence human development and adaptation. When these basic adaptive systems fail, negative adjustment will follow (i.e., *maladaptation* in the context of adversity and *vulnerability* in the absence of adversity). Thus, the quest to understand and promote resilience must advance beyond individual traits and characteristics, to encompass the entire system of dynamic developmental influences (Luthar & Cicchetti, 2000).

This entry provides an introduction to the construct of resilience with an explicit emphasis on its relevance for understanding and promoting health and wellness. First, we conceptualize resilience within a developmental framework wherein culturally and developmentally appropriate adaptation in the context of prior or ongoing adversity reflects a developmental process that may vary over time and contexts. Second, we explicate factors that contribute to resilience generally (i.e., main effects of risks and assets), as well as those that take on disproportionate salience in contexts of adversity exposure (i.e., moderating effects of vulnerability and protective factors). Third, we review mechanisms at biological, psychological, and social levels of analysis that may influence individual differences in the expression of resilience over time and across contexts. Finally, we consider the implications of resilience theory for health psychology and for specific efforts to bolster positive health outcomes, particularly in contexts of adversity.

Resilience as a Developmental Process

Although resilience can characterize a range of living systems, including schools, neighborhoods, and ecosystems, it is most often used to describe a pattern of individual development that proceeds in a (culturally) desired fashion despite encountering challenges and threats to positive development that go beyond the bounds of typical human experience (i.e., adversity). Contemporary models recognize that resilience is a process that may characterize the development of an individual but nevertheless reflects a range of influences beyond the individual. In this view, human resilience is not an expression of a singular trait or characteristic, but rather reflects a process of transactional exchanges among multiple systems within and beyond the individual (Luthar & Cicchetti, 2000). Together, these systems enable the individual to adapt effectively despite the presence of clear and present threats to their doing so (i.e., adversity).

Resilience is not about those who *have* or do not *have* resilience, or those who *are* or *are not* resilient; it is a description, rather than a causal agent. Resilience describes a developmental process by which core adaptive systems continue to operate in a healthy and desirable manner despite threats to their doing so. As described in the next section, resilience may be supported or thwarted by a range of factors. As these influences change over time, so, too, will the expression of resilience wax or wane across development. Likewise, expressions of resilience may vary over contexts within time, and individuals may have the resources to navigate one type of adverse experience, but not another. Resilience embodies a dynamic developmental process that rests neither in the individual nor in the context but rather in the transactional relation between individuals and their contexts.

Influences on the Process of Resilience

Resilience is a dynamic developmental process that emerges out of broader contexts of risk and protection. Across multiple levels of developmental influence, there are factors that are universally good or bad for everyone (i.e., main effects), and there are factors that change the impact of specific events and experiences for better and/or for worse (i.e., moderating effects). Resilience is multiply determined by the influence of main and moderating effects on the individual's capacity to adapt effectively despite adversity exposure.

Asset and *risk* factors exert main or uniform effects on development for better or worse, respectively. Indeed, the expression of resilience is, in part, a reflection of cumulative or net

risk, namely, the compilation of factors that are known to threaten positive adaptation (i.e., risks) as they operate concurrently with factors that are known to support positive adaptation (i.e., assets). Some examples of common assets or resources that support health and wellness for *all* individuals include perinatal care, high-quality schools, regular health checkups, a safe and cohesive family environment, and strong cognitive and problem-solving skills. Risk factors that are statistically associated with negative human health outcomes include premature birth, family history of heritable illness, obesity, and smoking. Importantly, assets and risks rarely occur in isolation and tend to accumulate such that strength engenders strength and risk engenders risk. Unfortunately, as risks accumulate in number and severity, assets typically wane yielding high *net risk* and an increased probability of maladaptive outcomes (Appleyard, Egeland, Van Dulmen, & Sroufe, 2005).

Whereas assets and risks affect all individuals to a similar degree regardless of individual differences in adversity exposure, other developmental influences take on special significance when they occur in contexts of adversity. *Protective* and *vulnerability* factors are characterized by neutral or modest effects in contexts of low adversity but evidence stronger developmental effects as risks increase, assets decrease, and adversity becomes prominent. Protective factors moderate the impact of adversity on development by reducing its negative effects. For example, recent efforts to develop disaster-proof hospitals in high-risk areas will have neutral effects on patient health, except in the case of a natural disaster when such efforts will take on special significance to promote resilience to the negative impact of disaster on health service provisions. Much like an airbag takes its protective function from its ability to buffer the blow of an accident, protective factors are particularly important for understanding when and how individuals adapt effectively in the context of concomitant adversity. In contrast to the buffering effect of protective factors, vulnerability factors moderate the impact of adversity on development by magnifying its negative effects. For example, a person with an immune deficiency is vulnerable to negative health outcomes when challenged by toxins in the environment or disease processes in the body, but this vulnerability factor has a relatively neutral effect on health in the absence of such exposures.

Protective and vulnerability processes influence adaptation in many different ways. They may open or close avenues of opportunity, stop or initiate cascades of negative events, or interact with other developmental influences to foment or mitigate negative adjustment in contexts of adversity (i.e., maladaptation). Importantly, some factors may serve multiple moderating functions, thereby influencing the impact of adversity for better or worse. A classic example of this differential effect can be seen in research on how individuals' genes can interact with the environment to influence emotional adjustment. The serotonin transporter gene is responsible for helping the brain regulate the amount of the serotonin neurotransmitter, which is strongly implicated in various forms of psychopathology, including depression. The gene comes in two primary forms, short and long. On the one hand, a short serotonin transporter genotype is a vulnerability factor, which has the potential to magnify risks for depression in the context of clear adversity exposure, such as child maltreatment (Caspi et al., 2003). On the other hand, the long allele genotype serves as a protective factor that mitigates against this same negative outcome but only in the context of maltreatment. Operating as a vulnerability and protective process, the serotonin transporter genotype does not influence adjustment in all situations uniformly, but rather the meaning of this genotype takes its significance from the surrounding context of relative adversity, serving to moderate the impact of child maltreatment on emotional health outcomes for better and for worse.

Just as the short and long serotonin transporter genotypes may magnify or mitigate the impact of adversity on emotional health, respectively, other factors may moderate the

contributions of both positive and negative environmental factors on human health and well-being. For example, contemporary research on physiologic stress reactivity points to a biological sensitivity to context effect, wherein the organism is rendered more sensitive to both positive and negative environmental influences. In a now-classic study of psychobiologic stress reactivity and illness, Boyce et al. (1995) demonstrated that children with sensitive stress response systems (i.e., children who exhibit higher cardiac reactivity in response to challenge) developed more respiratory health problems in the context of high environmental stress (i.e., family and childcare setting stressors) but actually developed fewer respiratory illnesses in the absence of such exposure.

Taken together, the main effects of assets and risks, the moderating effects of protective and vulnerability factors, and the differential effects illustrated in recent research on biological models of contextual sensitivity and impact illustrate that resilience is multiply determined. The impact of a given factor on adjustment may vary across time, across contexts, and in tandem with other factors to engender or undermine resilience processes in development. In addition to identifying these factors, efforts to clarify the mechanisms by which they influence resilience have significant implications for developing the most efficacious interventions to prevent maladaptation and promote resilience in contexts of adversity.

Explanatory Mechanisms

Multiple mechanisms underlie the main, moderating, and differential effects of various influences on resilience. Although classical theories of risk and protection favored psychosocial conceptualizations, recent years have witnessed a complementary rise in the consideration of resilience (and the factors that promote or thwart it) in biobehavioral models of health and wellness. Clarifying *how* processes of resilience emerge and diverge at multiple levels of analysis will reveal opportune moments and sites for interventions to promote human health and well-being (Cicchetti, 2010). A range of biological, psychological, and social processes may explain the impact of varied risks and assets, or protective and vulnerability factors on health and health behaviors in contexts of adversity.

The primary biological mechanism by which organisms adjust to environmental variation and stress is allostasis. *Allostasis* is the process by which individuals mobilize physiologic responses to manage challenge and return to a homeostatic condition that promotes health and restoration (McEwen, 1998). Multiple systems mediate this process, particularly the neuroendocrine hypothalamic–pituitary–adrenal (HPA) axis and the parasympathetic and sympathetic branches of the autonomic nervous system. With exposure to chronic stress (e.g., disease, malnutrition, violence), allostatic capacities may become overloaded. *Allostatic load* refers to the excessive demand placed on psychobiologic systems in contexts of chronic stress and adversity, which, in turn, may contribute to negative health outcomes (McEwen, 1998). At the level of biology, assets and risks, as well as protective and vulnerability factors, may operate to increase or decrease the allostatic demand on the organism. For example, assets may reduce the need for allostatic adjustment directly and/or provide additional support if allostatic adjustment is needed (e.g., medication to reduce the physiologic demands of illness; food, blankets, and shelter to offset the physiologic demands of homelessness).

Influences on resilience may also operate via psychological mechanisms. For example, a sense of personal coherence supports one's ability to understand the world and one's experiences in it (particularly difficult life experiences) and is thought to be an important process for

coping with stress and managing health (Amirkhan & Greaves, 2003). Various factors may influence the capacity for coherence and meaning making and, by extension, for resilience. For example, individuals living in contexts of increased mortality risk (e.g., life-threatening illness, active military service) may have a foreshortened sense of time such that they struggle to project themselves into the future. By undermining the psychological sense of continuity across past, present, and future that defines a person's sense of coherence, these factors may negatively impact the individual's use of healthcare and/or health-related activities, such as healthy eating, exercise, or risk-taking. In a recent study of natural disaster preparation, investigators showed that a sense of personal agency (i.e., the belief that one's action will have an effect on one's outcome) facilitated positive and preventive action (Paton, Parkes, Daly, & Smith, 2008). In contrast, influences that confer a sense of helplessness, fatalism, and limited opportunity (e.g., poverty, racism) can jeopardize a sense of personal coherence and, by extension, undermine the capacity to express resilient adaptation.

Finally, main and interactive influences on resilience can operate via social mechanisms. For example, social capital refers to knowledge and resources that derive from the interdependence of social networks (Caughy, O'Campo, & Muntaner, 2003). In times of need, access to and inclusion in social networks can provide necessary resources to navigate life's difficulties. Influences on resilience may operate by providing connections with positive networks and/or blocking engagement with negative networks. With regard to health, assets and protective factors on the one hand and risks and vulnerability factors on the other hand may provide or hinder the social capital (e.g., relevant connections) to successfully navigate the healthcare system. For example, in the context of a life-threatening illness, these factors may influence the patient's ability to advocate for a second opinion, locate an expert physician, and/or martial support for care and recovery.

Multiple mechanisms underlie resilience and may work together at any given time to produce various outcomes. For example, studies of post-traumatic stress disorder (PTSD) show that the presence of a supportive other can reduce the *biological* hyperarousal that characterizes the trauma response and other features prominent in the onset of PTSD (Ozbay, Fitterling, Charney, & Southwick, 2008). Through conversation and empathic exchange, a supportive other may also buffer, engender, or restore the individual's *psychological* sense of coherence in the wake of a traumatizing experience. Finally, a supportive *social* connection may provide access to tangible resources, such as a therapist referral or a safe place to stay, that may foster resilience to the impact of traumatic experiences.

The fundamental adaptive systems thought to most strongly support the "ordinary magic" of resilience likely operate across biological, psychological, *and* social levels of analysis and action. As yet another example, attachment processes promote adaptive stress modulation at the biological level (Gunnar & Quevedo, 2007), a sense of coherence at the psychological level (Sroufe, 1979), and connections to support networks at the social level (Kawachi & Berkman, 2001). Additional core systems of adaptation, such as self-regulation and cognitive processing, likely operate across multiple levels of developmental influence as well. In a complementary fashion, the most potent risks to development also undermine adjustment across multiple systems. For example, poverty likely taxes allostatic capacities (Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011), distorts an individual's sense of coherence (Amirkhan & Greaves, 2003), and limits social capital (Caughy et al., 2003). In future research, it will be important to elucidate whether and how specific mechanisms take on differential salience as a function of the kind of adversity exposure (e.g., illness, poverty, maltreatment), the specific type of adjustment outcome (e.g., immune function, behavior, mood), and the period of development in question (e.g., infancy, adulthood, old age).

Implications for Health and Health Resilience

A developmental process framework adopts a systems view in which resilience is conceptualized as a pattern of individual adaptation in a given context and over time. Moreover, this process may be engendered or compromised by the operation of multiple factors working at biological, psychological, and social levels of action. This view has significant implications for how we understand and promote health and health resilience.

Time is of central importance in a developmental model of resilience. Development is cumulative such that early adaptations provide a foundation for subsequent adjustment. In some instances, the health consequences of adversity exposure may be readily apparent (e.g., if you bump your head, a bruise will appear immediately), yet in others the effects may be quite delayed (e.g., maintaining homeostasis despite childhood stress exposure may lead to a range of physical ailments in middle adulthood; Brody et al., 2013). Moreover, the impact of these influences may vary over time (e.g., breaking a growth plate in childhood may result in a shortened limb, whereas the same break would not stymie growth in later development). Likewise, some traumatic experiences are especially pernicious when they occur early in life, whereas the same events may have less impact in later life if previous experiences have provided necessary skills to better navigate that adversity. For example, being in a rare natural disaster in childhood may lead to increased anxiety of another occurrence, while the same experience in adulthood would be accompanied with the knowledge of its rarity. In other ways, such events may be less consequential for early development relative to later development. For example, experiencing a hurricane and losing one's possessions in infancy is perhaps less detrimental than in adolescence when youth place greater importance on material markers of status and identity. Finally, the expression of resilience itself may vary over time amidst shifts in the balance of assets and risks, of protective and vulnerability factors.

Greater consideration of the individual's developmental status and the timing of factors influencing resilience will inform future prevention and intervention efforts. Identifying periods of marked vulnerability to subsequent maladaptation may provide information about the most opportune times to intervene. For example, because smoking behaviors most often begin in adolescence, prevention efforts should target this developmental epoch. In addition, a developmental perspective emphasizes the cumulative nature of both adversity and resilience. For example, individuals who have had positive health encounters in the past will be more likely to negotiate future health challenges in an effective fashion, both because the developmental system itself is healthier and because the individual is able to mobilize adaptive resources within and outside the self to engage positive health change.

A comprehensive model of health resilience must consider the simultaneous operation of multiple influences at multiple levels of action over time. The roots of individual health and health resilience extend well beyond the individual. In a study of patients with diabetes, Yi, Vitaliano, Smith, Yi, and Weinger (2008) found that stable glycemic control and fewer diabetes-related health problems reflected the simultaneous operation of both individual processes (e.g., self-efficacy and self-esteem) and systems external to the patient (e.g., social support and health education resources). Extending to the community, access to medical facilities, professional guidance, and effective treatments can increase the likelihood that a person may resist or recover from the negative impact of adverse health events. At the broadest level, health welfare and insurance policies influence individuals' access to affordable, reliable, and immediate healthcare. Together, these factors transact to influence individuals' relative risk or resilience to negative health outcomes.

The emphases on time and multiple levels of influence in a developmental model of resilience suggest that efforts to promote health may operate in various periods of development

and at different levels (see Yates & Masten, 2004 for discussion). When risks are identifiable and modifiable, risk-focused interventions can eradicate threats to development. For example, given that adolescence is a period of heightened risk for smoking, we can strive to decrease risk by preventing youth from purchasing nicotine products prior to age 18. However, in cases where the source of risk is unclear or intractable, asset-focused interventions can combat threats to development. For example, genetic vulnerabilities to illness resist modification, but we can ensure access to basic healthcare and preventive screening to increase the possibility of a positive health outcome in the event of disease expression. Preventing risks and increasing assets are important intervention initiatives in their own right, but their impact will be magnified by process-focused approaches that aim to increase individuals' capacities to make use of a new resource context and/or to navigate ongoing risks. Such process-focused interventions can target basic systems known to promote resilience (e.g., attachment, self-regulation) to protect them from harm and/or to promote or reactivate their optimal functioning.

Just as the most pernicious threats to development undermine multiple systems, the most efficacious interventions incorporate multiple avenues to health and wellness (Wyman, Sandler, Wolchik, & Nelson, 2000). For example, obesity is a prominent health concern with negative and enduring health ramifications. Soda bans in various states constitute a *risk-focused* intervention that decreases exposure to a known risk for obesity, namely, excess sugar consumption. At the same time, *asset-focused* interventions may provide nutritional education or low-cost opportunities for health and fitness to combat obesity. Finally, a *process-focused* model could target core systems of self-worth and self-efficacy to activate individuals' motivation to take responsibility for their health and promote their agency to enact a healthy lifestyle. In combination, these approaches have the potential to combat cumulative sources of risk and vulnerability associated with obesity.

Closing Comments

Resilience is a prominent area of interest for psychologists and health researchers alike. Amidst contemporary debates about vaccinations and struggles with infectious diseases, meeting the need to understand why and how people are differentially susceptible to risk and achieving the challenge to elucidate mechanisms by which we can bolster health and wellness are of increasing importance. Resilience theory and research have the potential to guide these efforts. However, opportunities for translation from resilience science to practice may be limited by barriers among research, practice, and policy. Moving resilience ideas forward into the lived experiences of individuals and communities will require open minds and new collaborations. In this chapter, we explicate the multitude of factors and processes that support or undermine positive adaptation in contexts of adversity. We encourage and guide the application of a developmental view of resilience to the fields of health and health psychology with the hope of advancing and informing ongoing and collaborative efforts to promote positive health trajectories for all people.

Author Biographies

Kristen L. Rudd, MA, is a doctoral student at the University of California, Riverside. Her research focuses on early childhood experiences of stress and adversity and the influence of the physiologic stress response on adjustment outcomes.

Tuppett M. Yates, PhD, is an associate professor of psychology and director of the Adversity and Adaptation Laboratory (www.adlab.ucr.edu) at the University of California, Riverside. Her research examines the impact of childhood adversity on developmental pathways toward psychopathology and competence with particular emphases on relational and regulatory developmental processes.

References

- Amirkhan, J.H. and Greaves, H. (2003). Sense of coherence and stress: The mechanics of a healthy disposition. *Psychology and Health* 18 (1): 31–62. <https://doi.org/10.1080/0887044021000044233>.
- Anda, R.F., Felitti, V.J., Bremner, J.D. et al. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience* 256 (3): 174–186. <https://doi.org/10.1007/s00406-005-0624-4>.
- Appleyard, K., Egeland, B., Van Dulmen, M.H.M., and Sroufe, L.A. (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology & Psychiatry* 46 (3): 235–245. <https://doi.org/10.1111/j.1469-7610.2004.00351.x>.
- Blair, C., Raver, C.C., Granger, D. et al. (2011). Allostatic and allostatic load in the context of poverty in early childhood. *Development and Psychopathology* 23 (03): 845–857. <https://doi.org/10.1017/S0954579411000344>.
- Boyce, W.T., Chesney, M., Alkon, A. et al. (1995). Psychobiologic reactivity to stress and childhood respiratory illnesses: Results of two prospective studies. *Psychosomatic Medicine* 57 (5): 411–422.
- Brody, G.H., Yu, T., Chen, E. et al. (2013). Is resilience only skin deep? Rural African Americans' socio-economic status-related risk and competence in preadolescence and psychological adjustment and allostatic load at age 19. *Psychological Science* 24 (7): 1285–1293. <https://doi.org/10.1177/0956797612471954>.
- Caspi, A., Sugden, K., Moffitt, T.E. et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science* 301 (5631): 386–389. <https://doi.org/10.1126/science.1082968>.
- Caughy, M.O.B., O'Campo, P.J., and Muntaner, C. (2003). When being alone might be better: Neighborhood poverty, social capital, and child mental health. *Social Science & Medicine* 57 (2): 227–237. [https://doi.org/10.1016/S0277-9536\(02\)00342-8](https://doi.org/10.1016/S0277-9536(02)00342-8).
- Cicchetti, D. (2010). Resilience under conditions of extreme stress: A multilevel perspective. *World Psychiatry* 9 (3): 145–154.
- Gunnar, M.R. and Quevedo, K.M. (2007). Early care experiences and HPA axis regulation in children: A mechanism for later trauma vulnerability. *Progress in Brain Research* 167: 137–149. [https://doi.org/10.1016/s0079-6123\(07\)67010-1](https://doi.org/10.1016/s0079-6123(07)67010-1).
- Kawachi, I. and Berkman, L.F. (2001). Social ties and mental health. *Journal of Urban Health* 78 (3): 458–467. <https://doi.org/10.1093/jurban/78.3.458>.
- Luthar, S.S. and Cicchetti, D. (2000). The construct of resilience: Implications for interventions and social policies. *Development and Psychopathology* 12 (04): 857–885.
- Masten, A.S. (2014). *Ordinary magic: Resilience in development*. New York, NY: Guilford Publications <https://doi.org/10.1037/0003-066X.56.3.227>.
- McEwen, B.S. (1998). Stress, adaptation, and disease: Allostatic and allostatic load. *Annals of the New York Academy of Sciences* 840 (1): 33–44. <https://doi.org/10.1111/j.1749-6632.1998.tb09546.x>.
- Ozday, F., Fitterling, H., Charney, D., and Southwick, S. (2008). Social support and resilience to stress across the life span: A neurobiologic framework. *Current Psychiatry Reports* 10 (4): 304–310. <https://doi.org/10.1007/s11920-008>.
- Paton, D., Parkes, B., Daly, M., and Smith, L. (2008). Fighting the flu: Developing sustained community resilience and preparedness. *Health Promotion Practice* 9 (4 suppl): 45S–53S. <https://doi.org/10.1177/1524839908319088>.

- Sroufe, L.A. (1979). The coherence of individual development: Early care, attachment, and subsequent developmental issues. *American Psychologist* 34 (10): 834–841. <https://doi.org/10.1037/0003-066X.34.10.834>.
- Werner, E.E. and Smith, R.S. (1982). *Vulnerable but invincible: A longitudinal study of resilient children and youth*. New York, NY: McGraw Hill.
- Wyman, P.A., Sandler, I., Wolchik, S., and Nelson, K. (2000). Resilience as cumulative competence promotion and stress protection: Theory and intervention. In: *The promotion of wellness in children and adolescents* (eds. D. Cicchetti, J. Rappaport, I. Sandler and R.P. Weissberg), 133–184. Washington, DC: Child Welfare League of America.
- Yates, T.M. and Masten, A.S. (2004). Fostering the future: Resilience theory and the practice of positive psychology. In: *Positive psychology in practice* (eds. P.A. Linley and S. Joseph), 521–539. Hoboken, NJ: Wiley.
- Yi, J.P., Vitaliano, P.P., Smith, R.E. et al. (2008). The role of resilience on psychological adjustment and physical health in patients with diabetes. *British Journal of Health Psychology* 13 (2): 311–325. <https://doi.org/10.1348/135910707x186994>.

Suggested Reading

- Cicchetti, D. (2010). Resilience under conditions of extreme stress: A multilevel perspective. *World Psychiatry* 9 (3): 145–154.
- Masten, A.S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist* 56 (3): 227.
- Paton, D., Parkes, B., Daly, M., and Smith, L. (2008). Fighting the flu: Developing sustained community resilience and preparedness. *Health Promotion Practice* 9 (4 suppl): 45S–53S.
- Yates, T.M. and Masten, A.S. (2004). *Fostering the future: Resilience theory and the practice of positive psychology*. Hoboken, NJ: Wiley.

Health Information Avoidance

Jennifer L. Howell¹, Nikolette P. Lipsey²,
and James A. Shepperd²

¹*Psychology Department, Ohio University, Athens, OH, USA*

²*Psychology Department, University of Florida, Gainesville, FL, USA*

In the modern era, people can learn their disease risk, receive diagnoses, track their physical activity, share health information with their physician, and receive direct-to-consumer genetic testing all from the comfort of home. Nevertheless, people are sometimes reticent to learn information about themselves (Shepperd & Howell, 2015; Sweeny, Melnyk, Miller, & Shepperd, 2010).

We review research on *health information avoidance*, the motivated decision to avoid learning information about one's physical health (Shepperd & Howell, 2015). In this entry, we define health information avoidance and discuss its prevalence. We also discuss individual differences in health information avoidance, explore its causes, and examine interventions that can reduce it.

Defining Health Information Avoidance

Information avoidance is “any behavior designed to prevent or delay the acquisition of available but potentially unwanted information” (Sweeny et al., 2010, p. 314). Information avoidance is distinct from failing to seek information in that the former represents a motivated decision to avoid information that threatens how one wishes to think, feel, or behave. For example, a woman who does not seek articles about oral cancer because she has never heard of oral cancer or because she simply does not care is not actively avoiding that information.

Health information avoidance encompasses a variety of behaviors. Moreover, it subsumes several other information-related psychological phenomena including selective exposure (avoiding attitude inconsistent information), blunting (ignoring information about a disease after diagnosis with that disease), preferences for information (patients' desire to know about their medical status and care), and other forms of defensiveness and avoidance (e.g., ignoring

stimuli that might indicate risk; see Howell & Shepperd, 2016). These other psychological phenomena represent special cases of information avoidance (Sweeny et al., 2010).

The Problem of Health Information Avoidance

Although information avoidance can protect people from threatening information, it can also be problematic. For instance, delaying diagnosis can have dire health consequences for diseases such as cancer or HIV, for which early initiation of treatment can substantially prolong life. Thus, to the extent that late diagnosis reflects health information avoidance, such avoidance can prove deadly.

Prevalence of Health Information Avoidance

How prevalent is health information avoidance? In one study, 37% of university students and 31% of general-population adults indicated that they had at some point done something to “avoid getting information about a health topic” (Barbour, Rintamaki, Ramsey, & Brashers, 2012, p. 215). In another study, 25% of a nationally representative sample of US adults agreed or strongly agreed with the statement “I can think of situations in which I would rather not know everything about my health” (Howell, Crosier, & Shepperd, 2014).

Evidence suggests that the prevalence of health information varies widely based on the type of information avoidance examined and the population evaluated. For instance, 39% of a nationally representative sample of US adults agreed with the statement “I would rather not know my risk for cancer” (Emanuel et al., 2015). Nevertheless, only 13% of a sample of Black adults living in rural north Florida agreed with the statement “I would rather not know my risk for mouth and throat cancer” (Shepperd, Howell, & Logan, 2014).

The prevalence of information avoidance also varies based on how one defines avoidance. Rates of avoidance are typically much lower when one assesses avoidance in terms of intentions to avoid information versus actual avoidance behavior. Indeed, although almost 88% of rural black adults in one study signed up to attend an oral cancer examination, only 40.1% of the study participants who signed up actually attended their screening appointment (Shepperd, Emanuel, Howell, & Logan, 2015). In this case assessing avoidance via intentions rather than behavior resulted in a lower prevalence of information avoidance.

Predictors of Health Information Avoidance

A number of studies have examined individual differences in information avoidance and found that some people are more inclined than others to avoid information. Researchers have identified both demographic and personality predictors of avoidance.

Demographic Predictors of Avoidance

The findings regarding demographic predictors of health information avoidance are inconsistent. Whereas some studies find greater health information avoidance among younger adults than among older adults (e.g., Persoskie, Ferrer, & Klein, 2013), others find the opposite pattern (e.g., Emanuel et al., 2015), or no relationship (e.g., Shepperd et al., 2015). The

findings for gender are also mixed, with some studies finding the greatest health information avoidance among men (e.g., Kelly et al., 2010) and others finding the greatest health information avoidance among women (e.g., Emanuel et al., 2015).

In contrast to age and gender, education consistently predicts avoidance. Research links lower education to greater avoidance of general health information (Shepperd & Howell, 2015) and cancer risk feedback (Emanuel et al., 2015), greater avoidance of doctors (Persoskie et al., 2013), and lower health information seeking (Kelly et al., 2010).

Of course, demographic variables are not psychological variables *per se* but rather are proxies for one or more psychological constructs. For example, education is likely a proxy for dispositional curiosity or perhaps psychosocial resources. As we discuss presently, both greater coping resources and greater curiosity correspond with less information avoidance.

Personality Measures

Studies examining personality and information avoidance reveal two general predictors of avoidance: orientation toward information and personal and interpersonal resources. First, several studies offer evidence that people who are dispositionally open to information display less information avoidance. For example, one study that followed 316 undergraduate students over 4 months included several measures assessing orientation to information (e.g., dispositional curiosity, tolerance of uncertainty) as well a general measure of avoidance of health information. On every measure of openness to information, participants who were dispositionally open to new information reported less inclination to avoid health information (Shepperd & Howell, 2015).

Second, dealing with psychosocial threats demands psychosocial resources (Howell et al., 2014). Not surprisingly, several studies find that having fewer personal and interpersonal resources (e.g., coping resources, social support) corresponds with greater information avoidance (Howell et al., 2014). For example, in one study students who reported having more coping resources were less likely to avoid viewing a personal UV photo depicting sun damage (Dwyer, Shepperd, & Stock, 2015). In a second study, having more threat-management resources (i.e., being more optimistic, spontaneously self-affirming) corresponded with less avoidance of genetic screening results (Taber et al., 2014), and a third study found that reports of greater coping ability and having a network of close friends that one can talk to about one's health corresponded with less avoidance of health risk feedback (Howell et al., 2014). These studies confirm findings from experimental studies showing that making people feel socially isolated (via social exclusion) increases their avoidance of health risk feedback (Howell & Shepperd, 2017), whereas bolstering people's ability to manage bad news by affirming their personal worth reduces avoidance (Howell & Shepperd, 2012).

Causes of Health Information Avoidance

People avoid information for three broad reasons: The information potentially threatens how they wish to feel, think, or behave (Sweeny et al., 2010).

Affective Concerns

People are motivated to create and maintain positive affect and sometimes avoid health information to maximize positive emotions and minimize negative emotions (Sweeny et al., 2010).

Unfavorable health information can evoke a variety of negative emotions including sadness, fear, anger, regret, disappointment, and guilt. Numerous studies document that people avoid health information to avoid these unpleasant emotions. For instance, studies find that concerns over negative emotions prompt avoidance of doctors (Persoskie et al., 2013).

Of course, people also avoid information to preserve or enhance positive emotions. For example, people cite a desire to preserve hope as a motive for avoiding health information (Barbour et al., 2012). Importantly, people are more likely to avoid information if they anticipate the information will make them feel bad. By contrast, currently feeling bad does not appear to influence information avoidance (Persoskie et al., 2013).

Challenge to Self- or Worldview

Because people are motivated to create and maintain consistent and positive views of themselves and their worlds, they will avoid information that threatens their beliefs (Earl et al., 2009). Although few studies examine health information avoidance, theorizing and research on selective exposure—a type of information avoidance—suggests that people will avoid learning health information that challenges their beliefs. In one study, participants were less likely to enroll in a HIV-prevention counseling session to the extent that they had not used condoms in the past (Earl et al., 2009). By so doing, they avoided information that might challenge their belief that they are healthy and make good decisions.

Finally, people report that they sometimes avoid health information to maintain the belief that they are healthy (Barbour et al., 2012). For example, participants were more likely to avoid a UV photo to the extent that they thought it might reveal skin damage (including showing them to be “ugly”), particularly if they did not regularly engage in sun-protective behavior (Dwyer et al., 2015). Together, these findings suggest that just as people will avoid belief-inconsistent information generally, people avoid health information that might threaten desired beliefs about themselves or their health.

Challenge to Behavior

People are prone to avoid information that they believe might constrain them from behaving as they wish (Howell & Shepperd, 2013a). For example, several studies demonstrate that people are more likely to avoid learning their risk for a disease if they believe it will obligate them to take an unpleasant or disgusting definitive test (e.g., a cervical exam) than if they believed it would obligate a less invasive test (e.g., a cheek swab; Howell & Shepperd, 2013a). Similarly, study participants are more likely to avoid learning their risk for a disease if having the disease requires that they take a pill for the rest of their lives versus for a week (Howell & Shepperd, 2013a). Together, the evidence suggests that people avoid health information to the extent that the information obligates undesired action.

Interpersonal Motives

The three motives discussed thus far represent personal motives: People avoid information that threatens how they wish to think, feel, or behave. Yet researchers have long known that interpersonal motives also influence behavior. As such, if health information might become available to others, and that information might negatively influence how others think about them, feel about them, or behave toward them, people may avoid learning the information.

We know of no experimental research demonstrating that people avoid health information for interpersonal reasons. However, correlational evidence suggests that people may avoid information for interpersonal reasons. For instance, in a study of genetic screening, participants often report that receiving and sharing an undesirable diagnosis with close others might cause others to perceive them as a burden or desert them (Yaniv, Benador, & Sagi, 2004). Similarly, in a study on Alzheimer's genetic testing decisions, more than 50% of the people who chose to avoid Alzheimer's genetic testing cited concern about loved ones' reactions as an important reason not to get tested. These findings suggest that concerns with the role others play in health decisions may prompt avoidance.

Reducing Health Information Avoidance

Given that health information avoidance can lead to problematic health outcomes (e.g., delayed diagnosis and treatment, disease spread), it is important to understand how to reduce it. Here we review three interventions to reduce health information avoidance: self-affirmation, motives contemplation, and increasing perceptions of control.

Self-Affirmation

Self-affirmation involves prompting people to consider their personal values and strengths (see Sherman, 2013 for a review). When people facing threats self-affirm (typically by writing or thinking about a recent demonstration of an important value), they are better able to focus on aspects of themselves that are not under threat and to draw on the psychological resources necessary to manage threat (Sherman, 2013). Similarly, affirmation should reduce information avoidance because it reduces perceptions of the resources required and increases perceptions of resources available to deal with threatening information. Consistent with this reasoning, people who are affirmed are more likely to view the results of online health risk calculators even when the information might obligate unwanted action (Howell & Shepperd, 2012).

Motives Contemplation

Contemplation involves prompting people to evaluate the pros and cons of seeking versus avoiding information. One approach to prompting contemplation is to have people list their reasons for seeking and their reasons for avoiding threatening information and then having them evaluate the strength of each reason. This intervention has successfully reduced avoidance of health risk feedback for a variety of diseases including cardiovascular disease and melanoma (Howell, Ratliff, & Shepperd, 2016). The mechanisms underlying the effectiveness of contemplation remain unclear. Some research suggests that contemplation encourages rational decision making (Howell & Shepperd, 2013b). Other research suggests that it prompts people to justify intuitive reactions (Howell et al., 2016). Regardless of the mechanism, contemplation reduces avoidance only when people view seeking as the prudent course of action (e.g., when disease is controllable), but not in situations in which people view avoiding as the prudent course of action (e.g., when disease is uncontrollable; Howell & Shepperd, 2013b). As such, interventionists must exercise caution in using contemplation as a remedy for avoidance.

Increasing Perceived Control

We noted earlier that obligating behavior can prompt information avoidance (Howell & Shepperd, 2013a). In the health context, uncontrollable diseases represent a threat to people's ability to control their worlds, and people are often more inclined to avoid information about uncontrollable diseases than controllable diseases (Melnik & Shepperd, 2012; Yaniv et al., 2004). Importantly, emphasizing the controllable aspects of disease can reduce avoidance. For example, women in one study were more likely to choose to learn their breast cancer risk from an online risk calculator if they read about the controllable predictors of breast cancer (e.g., diet, exercise) than if they read about uncontrollable predictors of breast cancer (e.g., genetics; Melnik & Shepperd, 2012).

Summary

As people's access to health information grows, and interventionists try to promote healthy behavior through personal feedback and data, understanding people's reticence to receive health feedback becomes increasingly informative and important. Health information avoidance is the motivated avoidance of available health information and can be problematic, especially when it leads to delayed diagnosis. The prevalence of health information avoidance is highly variable, but avoidance seems to occur least among people who are dispositionally open to new information and most among people who lack psychosocial resources. Evidence suggests that information avoidance is a defensive behavior motivated by a desire to protect affect, cognition, and behavior. Interventions that reduce threat (e.g., affirmation), require people to contemplate the pros and cons of avoidance (e.g., motives contemplation), or increase personal control (e.g., emphasizing controllability) are effective in reducing health information avoidance. Although research on health information avoidance has answered several important questions, many more questions remain unanswered. The field would benefit from systematic examination information avoidance in a variety of health contexts. Moreover, additional research is needed on potential interventions to reduce harmful avoidance and to determine when (and for whom) intervention strategies are most effective. Finally, more research is needed to understand the downstream consequences of avoidance for health.

Author Biographies

Jennifer L. Howell, PhD, is an assistant professor at Ohio University. Her research focuses primarily on psychological threat management and health. She seeks to understand the proactive and reactive strategies people use to manage bad news, particularly about their health. This broad umbrella encompasses work on decision making, responses to feedback, defensiveness, physical health behaviors, risk, coping, individual differences, social cognition, attitudes, and communication.

Nikolette P. Lipsey is a graduate student at the University of Florida. She studies information avoidance and impression management. With an emphasis on health-related contexts, her research examines how perceptions of audiences influence information avoidance.

James A. Shepperd, PhD, is the R. David Thomas Endowed Professor of Psychology at the University of Florida and the research director for the Southeast Center for Research to

Disparities in Oral Health. Most of his research examines how people manage threatening information, which includes topics such as optimism, maintaining desired self-views, and information avoidance. Much of his work involves applying psychological theory to addressing health concerns such as screening for cancer.

References

- Barbour, J. B., Rintamaki, L. S., Ramsey, J. A., & Brashers, D. E. (2012). Avoiding health information. *Journal of Health Communication, 17*(2), 212–229. doi:10.1080/10810730.2011.585691
- Dwyer, L. A., Shepperd, J. A., & Stock, M. L. (2015). Predicting avoidance of skin damage feedback among college students. *Annals of Behavioral Medicine, 49*(5), 685–695. doi:10.1007/s12160-015-9703-6.
- Earl, A., Albarracín, D., Durantini, M. R., Gunnoe, J. B., Leeper, J., & Levitt, J. H. (2009). Participation in counseling programs: High-risk participants are reluctant to accept HIV-prevention counseling. *Journal of Consulting and Clinical Psychology, 77*(4), 668–679. doi:10.1037/a0015763
- Emanuel, A. S., Kiviniemi, M. T., Howell, J. L., Hay, J. L., Waters, E. A., Orom, H., & Shepperd, J. A. (2015). Avoiding cancer risk information. *Social Science & Medicine, 147*, 113–120.
- Howell, J. L., Crosier, B. S., & Shepperd, J. A. (2014). Does lacking threat-management resources increase information avoidance? A multi-sample, multi-method investigation. *Journal of Research in Personality, 50*, 102–109. doi:10.1016/j.jrp.2014.03.003
- Howell, J. L., Ratliff, K. A., & Shepperd, J. A. (2016). Automatic attitudes influence information avoidance. *Health Psychology, 35*(8), 816–823. doi:10.1037/hea0000330
- Howell, J. L., & Shepperd, J. A. (2012). Reducing information avoidance through affirmation. *Psychological Science, 23*(2), 141–145. doi:10.1177/0956797611424164
- Howell, J. L., & Shepperd, J. A. (2013a). Behavioral obligation and information avoidance. *Annals of Behavioral Medicine, 45*(2), 258–263. doi:10.1007/S12160-012-9451-9
- Howell, J. L., & Shepperd, J. A. (2013b). Reducing health-information avoidance through contemplation. *Psychological Science, 24*(9), 1696–1703. doi:10.1177/0956797613478616
- Howell, J. L., & Shepperd, J. A. (2016). Establishing an information avoidance scale. *Psychological Assessment*. ePub ahead of print. doi:10.1037/pas0000315
- Howell, J. L., & Shepperd, J. A. (2017). Social exclusion, self-affirmation, and health information avoidance. *Journal of Experimental Social Psychology, 68*, 21–26. doi:10.1016/j.jesp.2016.05.005
- Kelly, B., Hornik, R., Romantan, A., Schwartz, J. S., Armstrong, K., DeMichele, A., ... Kim, A. (2010). Cancer information scanning and seeking in the general population. *Journal of Health Communication, 15*(7), 734–753. doi:10.1080/10810730.2010.514029
- Melnyk, D., & Shepperd, J. A. (2012). Avoiding risk information about breast cancer. *Annals of Behavioral Medicine, 44*(2), 216–224. doi:10.1007/S12160-012-9382-5
- Persoskie, A., Ferrer, R. A., & Klein, W. M. (2013). Association of cancer worry and perceived risk with doctor avoidance: An analysis of information avoidance in a nationally representative US sample. *Journal of Behavioral Medicine, 37*(5), 977–987. doi:10.1007/s10865-013-9537-2
- Shepperd, J. A., Emanuel, A. S., Howell, J. L., & Logan, H. L. (2015). Predicting scheduling and attending for an oral cancer examination. *Annals of Behavioral Medicine, 49*(6), 828–838. doi:10.1007/s12160-015-9717-0
- Shepperd, J. A., & Howell, J. L. (2015). Responding to psychological threats with deliberate ignorance: Causes and remedies. In P. J. Carroll, R. M. Arkin, & A. Wichman (Eds.), *Handbook of personal security* (pp. 257–274). New York, NY: Taylor & Francis.
- Shepperd, J. A., Howell, J. L., & Logan, H. (2014). A survey of barriers to screening for oral cancer among rural Black Americans. *Psycho-Oncology, 23*(3), 276–282. doi:10.1002/pon.3415
- Sherman, D. K. (2013). Self-affirmation: Understanding the effects. *Social and Personality Psychology Compass, 7*(11), 834–845. doi:10.1111/spc3.12072

- Sweeny, K., Melnyk, D., Miller, W. A., & Shepperd, J. A. (2010). Information avoidance: Who, what, when, and why. *Review of General Psychology, 14*(4), 340–353. doi:10.1037/a0021288
- Taber, J. M., Klein, W. M., Ferrer, R. A., Harris, P. R., Shepperd, J. A., & Biesecker, L. G. (2014). Information avoidance tendencies, spontaneous self-affirmation, and interest in genetic sequencing feedback. *Annals of Behavioral Medicine, 49*(4), 616–621. doi:10.1007/s12160-014-9679-7
- Yaniv, I., Benador, D., & Sagi, M. (2004). On not wanting to know and not wanting to inform others: Choices regarding predictive genetic testing. *Risk Decision and Policy, 9*(4), 317–336. doi:10.1080/14664530490896573

Suggested Reading

- Emanuel, A. S., Kiviniemi, M. T., Howell, J. L., Hay, J. L., Waters, E. A., Orom, H., & Shepperd, J. A. (2015). Avoiding cancer risk information. *Social Science & Medicine, 147*, 113–120.
- Howell, J. L., Crosier, B. S., & Shepperd, J. A. (2014). Does lacking threat-management resources increase information avoidance? A multi-sample, multi-method investigation. *Journal of Research in Personality, 50*, 102–109.
- Howell, J. L., & Shepperd, J. A. (2012). Reducing information avoidance through affirmation. *Psychological Science, 23*(2), 141–145.
- Shepperd, J. A., & Howell, J. L. (2015). Responding to psychological threats with deliberate ignorance: Causes and remedies. In P. J. Carroll, R. M. Arkin, & A. Wichman (Eds.), *Handbook of personal security* (pp. 257–274). New York, NY: Taylor & Francis.
- Sweeny, K., Melnyk, D., Miller, W. A., & Shepperd, J. A. (2010). Information avoidance: Who, what, when, and why. *Review of General Psychology, 14*(4), 340–353. doi:10.1037/a0021288

Health Literacy

Amber S. Emanuel¹ and Kristel M. Gallagher²

¹*Psychology Department, University of Florida, Gainesville, FL, USA*

²*Thiel College, Greenville, PA, USA*

In a world where we seem to have access to a wealth of health knowledge and expertise at our fingertips, it may be difficult to imagine that the strongest predictor of an individual's health status is literacy skills (World Health Organization, 2013). Age, income, employment status, education level, and race—each of these factors certainly has a place in the equation, but literacy skills, and specifically *health literacy* skills, are a driving force in the promotion of optimal health. As an invisible factor in health, health literacy skills tend to be both overlooked and misunderstood by the medical community and general population alike. However, two out of every five people in the United States suffer from low health literacy (Kutner, Greenberg, Jin, & Paulsen, 2006), greatly putting their health at risk.

The National Institute of Health defines health literacy as the “degree to which individuals have the capacity to obtain, process and understand basic health information and services needed to make appropriate health decisions” (Institute of Medicine, 2004). In this definition, there are four key verbs that are important to note when studying health literacy: obtain, process, understand, and decide. The ability to *obtain* health information is the piece of health literacy with which most people are familiar because it is most associated with reading and writing skills. Since obtaining basic health information in the age of digital media is often as simple and quick as an Internet search, it is easy to overlook health literacy as a real problem. However, being able to gather health information is only a small part of having adequate health literacy skills. Including the ability to *process* and ultimately *understand* health information in the definition means that health literacy skills are rooted in deeper cognition, rather than surface level processing. Yet the most integral term in the definition of health literacy is *decide*. Individuals must not only have the ability to obtain, process, and understand health information and services, but they must also be able to use that information to make appropriate decisions regarding medical care. It is here that the real value of health literacy skills can be recognized.

Health literacy, then, is a functional type of literacy that goes far beyond the mere level of one's reading and writing skills. There are some clear similarities between health literacy and

literacy, such that both need an ability to recognize and understand words, search texts, and make text-based inferences. However, there are also some important differences. Consider the following example: “A two-year old is diagnosed with an inner ear infection and prescribed an antibiotic. Her mother understands that her daughter should take the prescribed medication twice a day. After carefully studying the label on the bottle and deciding that it doesn’t tell her how to take the medicine, she fills a teaspoon and pours the antibiotic into her daughter’s painful ear” (Parker, Ratzan, & Lurie, 2003). Here, the mother likely possesses adequate literacy skills—she recognized and understood the words on the label, knew to search the text on the label for instructions, and made a judgment on how to best proceed with the treatment given the location of the infection. However, she did not fully understand the relationship between the infection and the antibiotic, suggesting a low level of health literacy.

As highlighted in the above example, literacy and health literacy are not always connected. The focus of health literacy is narrower than general literacy. Unsurprisingly, health literacy focuses on health, being able to understand common health-related vocabulary and the health-care system (Vernon, Trujillo, Rosenbaum, & DeBuono, 2007). Health literacy also encompasses knowledge of the basic operation of the human body: how medications work, what causes diseases, and the relationship between lifestyle factors and health-related outcomes. Thus, health literacy is distinct from literacy.

Measures of Health Literacy

Since health literacy skills are unique, standardized measures of overall literacy are not equipped to accurately gauge an individual’s level of health literacy. Thus, there are a number of scales that specifically measure health literacy. Some are lengthier and provide a more in-depth picture of an individual’s level of health literacy, used mainly in the research domain, while others have been designed for ease of implementation in clinical settings. Many of these measures (e.g., Test of Functional Health Literacy [TOFHLA], Short Assessment of Health Literacy, Newest Vital Sign) are available in both English and Spanish. See further readings for a review of health literacy measures and their sources (Agency for Health Research and Quality, 2016).

The *TOFHLA* is a 50-item reading comprehension test and a 17-item numerical ability test that takes 22 min to administer. A shortened version of the *TOFHLA* (*s-TOFHLA*) takes approximately 12 min to administer. The 14-item *All Aspects of Health Literacy Scale* is a self-report measure with three subscales: functional health literacy, communicative health literacy, and critical health literacy. A slightly shorter questionnaire is the *Rapid Estimate of Adult Literacy in Medicine*, for which participants are asked to verbalize health-related words (e.g., allergic, jaundice, anemia), and a score of six or less puts them at risk for low health literacy. Similarly, the *Short Assessment of Health Literacy* is an 18-item measure of comprehension and pronunciation of health-related items. For example, participants are shown a stem (such as “infection”) and asked to say the word and then say which of two words (“plant or virus”) are most similar to the stem word.

Shorter measures include the 6-item *Newest Vital Sign* and the 3-item *Set of Brief Screening Questions* (SBSQ). The *Newest Vital Sign* uses a fictitious nutrition label from a carton of ice cream as the stimulus for the question prompts, involving both comprehension and numeracy problem-solving skills. A variation of the SBSQ is the *Single-Item Literacy Screener*, which uses the question, “How often do you need to have someone help you when you read instructions, pamphlets, or other written material from your doctor or pharmacy?” There are also some

disease-specific scales, such as the *Diabetes Numeracy Test* and the *Breast Cancer Literacy Assessment Tool*.

Regardless of the scale used to measure health literacy, simply asking whether the person understands the given health information is insufficient. Individuals are often reluctant to admit that they do not understand health information, especially if they have limited overall literacy as well. Further, as with overall literacy and most cognitive tasks, it is unreasonable to expect individuals to “know what they don’t know” when it comes to health information. One way providers have managed to avoid these issues is to use an indicator of health literacy that is short and practical, such as asking patients to read a prescription label.

Rates of Health Literacy

Unfortunately, health literacy is a common problem for many individuals. Commonly characterized as the health literacy “crisis,” up to two-thirds of patients may not be able to comprehend key health information essential to navigating the healthcare system, such as how to take medication or schedule a follow-up appointment (Williams, Davis, Parker, & Weiss, 2002). In 2003, the National Assessment of Adult Literacy (NAAL) measured health literacy in their large-scale national assessment of 20,000 adults. Importantly, the 2003 NAAL was the first large-scale assessment of literacy that included a specific measure of health literacy. Health literacy was characterized in the NAAL by participants’ responses to a series of 28 assessment tasks, such as reading health-related materials and answering questions to demonstrate comprehension of the material. Based on correct answers to these questions, respondents were divided into either Below Basic, Basic, Intermediate, or Proficient levels of health literacy. Participants in the “Proficient” level could perform tasks such as finding the definition of a medical term by searching a complex document and calculating health insurance costs for a year using only a monthly cost table. Only 12% of American adults possessed these proficient health literacy skills, while 36% had inadequate skills to make appropriate health decisions, falling into the bottom categories of “Basic” and “Below Basic.” The remaining participants performed at an “Intermediate” level, possessing the skills necessary to read a prescription label and decide on the appropriate times to take the medication.

Individuals in the “Basic” and “Below Basic” levels, approximately 77 million Americans, are at a particular disadvantage in the healthcare system. Having low levels of health literacy means that they may not be able to perform even the most basic of health activities necessary to take care of themselves and loved ones. These activities include circling the date of a medical appointment on a hospital appointment slip, following directions on a prescription drug label, or using a chart to adhere to a childhood immunization schedule, among others (Kutner et al., 2006).

Predictors of Health Literacy

Given the many problems associated with low levels of health literacy, can researchers identify its predictors? Several sociodemographic factors are associated with low health literacy. Most studies suggest an association between lower educational attainment and low health literacy (e.g., Baker, Gazmararian, Sudano, & Patterson, 2000). Lower socioeconomic status, measured using income, has also been linked to lower health literacy (Baker et al., 2000), though not all studies have found a significant association (Williams et al., 2002). Older adults are also

more likely to have low health literacy (e.g., Morrow et al., 2006), though this relationship may be explained by cognitive ability (Baker et al., 2000; Morrow et al., 2006).

Ethnicity may also be a predictor of health literacy. Most studies have compared the health literacy of Black and White individuals and found that, on average, Blacks have lower health literacy than Whites (e.g., Baker et al., 2002). Race may also interact with other sociodemographic variables. For instance, in a survey of American veterans, Black veterans had lower health literacy than White veterans, and low health literacy was especially pronounced in younger Black veterans (Rodríguez et al., 2013).

Other studies have concentrated on whether health literacy may partially explain health disparities (e.g., Sentell & Haplin, 2006). In some studies, health literacy mediates disparities between White and Black participants. For example, health literacy explains race differences in nonadherence to HIV regimes, misinterpretation of medication labels, prostate-specific antigen (PSA) levels among newly diagnosed prostate cancer patients, lack of health insurance in children, and quality of life among Medicare enrollees (Berkman, Sheridan, Donahue, Halpern, & Crotty, 2011). Similarly, in a sample in which 29% of older-aged participant had below-basic health literacy skills, health literacy mediated race disparities and education-related disparities in self-reported health status (Bennett, Chen, Soroui, & White, 2009). Thus, health literacy seems to explain some well-known health disparities.

In sum, the most consistent predictors of low health literacy seem to be low educational attainment, low socioeconomic status, and older age. However, many studies have also found that these sociodemographic predictors interact with each other or mediate each other, so these relationships are complex.

Monetary Cost of Low Health Literacy

One reason health literacy has started to garner attention in both the research and clinical realms is that low health literacy is expensive. The estimated cost of low health literacy to the US economy is between 106 and 236 billion dollars annually (Vernon et al., 2007). In a report about these issues, Vernon et al. (2007) estimated that the economic savings of improving health literacy in the United States could translate into enough funds to insure 47 million Americans—that is, every uninsured person in the United States, according to the 2006 Census Bureau.

Additionally, Medicaid patients with third grade or below reading levels cost \$7,500 more per year to insure than Medicaid patients with reading skills above the third grade level (see Nielsen-Bohlman, Panzer, & Kindig, 2004). Individuals with low health literacy are hospitalized more often, up to twice as likely to be admitted to the hospital than individuals with adequate health literacy (Baker, Parker, Williams, & Clark, 1998). This increase in hospitalization is also prevalent in Medicare patients (Baker et al., 2002), further placing a burden on the economic system. Thus, low health literacy is a serious concern for many facets of the health-care system.

Medical Decision Making and Health Literacy

Health literacy is an important predictor of patients' medical decisions. For example, low health literacy predicted wanting more aggressive treatment at end of life, independently of education and race (Vollandes et al., 2008). If the researchers had not measured health literacy,

they may have concluded that race and education uniquely predicted end-of-life decisions. Similarly, health literacy is a significant predictor of adherence to HIV medication regime, and including health literacy levels eliminated the health disparity that Blacks were more likely to be nonadherent (Osborn, Paasche-Orlow, Davis, & Wolf, 2007). Among the elderly, low health literacy is associated with poor self-care of chronic diseases, excess use of emergency care services, low use of preventative health services, and an increased risk for mortality (Baker et al., 2007; Berkman et al., 2011). In a recent meta-analysis of health literacy (Berkman et al., 2011), low health literacy was associated with poor ability to adhere to a medication regime, lower use of preventative services (mammography screening, influenza immunizations), greater use of emergency care, more hospitalization, and lower overall physical health status.

It should be noted that several potential mediators and moderators help to explain these relationships. For instance, knowledge, patient self-efficacy, norms, and stigma may be mediators, helping to explain the strong relationship between health literacy and medical decision making. Potential moderators may be social support and characteristics of the healthcare system (Berkman et al., 2011).

Physical health outcomes have also been linked to low levels of health literacy. In a British sample, low health literacy was associated with worse general physical fitness, greater body mass index (BMI), and fewer natural teeth. Health literacy skills are correlated with both adult and childhood cognitive ability, suggesting that health literacy may be an aspect of general cognition. Also, in this particular sample, education was a strong predictor of health literacy. The link between education and health literacy in this sample as compared with US samples may be due to differences between the UK and US education systems (Möttus et al., 2014).

While health literacy is related to BMI overall, it is also specifically related to obesity. Remarkably, obesity in school-aged children has been found to be associated with not just parental obesity, but also with their parents' health literacy. Further, obesity in adolescents is associated with the adolescents' own health literacy (Chari, Warsh, Ketterer, Hossain, & Sharif, 2014). Thus, physical health is influenced not only by one's own health literacy but also by parents' health literacy skills in childhood and early adolescence. Based on these findings, interventions to improve health literacy skills may be effective at improving the physical health of multiple generations.

Interventions Improving Health Literacy

Like general literacy, individuals can become more literate in the domain of health. However, much of the focus in this area has been on the healthcare provider rather than the healthcare recipient. Removing literacy-related barriers to health by increasing providers' abilities to communicate effectively and properly with low health literacy individuals has been where the most significant attention has been placed. Indeed, the need for healthcare providers to increase their ability to effectively communicate health information is critical, as many patients understand as little as 50% of what is communicated during healthcare visits (Ong, De Haes, Hoos, & Lammes, 1995).

A popular method that has been introduced to healthcare providers is the "teach-back" or "teach-to-goal" method (Batterham, Hawkins, Collins, Buchbinder, & Osborne, 2016; DeWalt et al., 2011). This method is used after a healthcare provider has explained some form of health information to the patient. The provider will ask the patient to explain—in his or her own words—the information as he or she understood it. As part of this approach, providers are taught to ask open-ended questions, avoid the use of yes-or-no questions, and take

responsibility for any misunderstanding that the patient has (DeWalt et al., 2011). Along with the “teach-back” method, another way providers can increase patients’ understanding is to use “living room language,” avoiding medical jargon, abbreviations, or complicated words (DeWalt et al., 2011). Using visual aids or images instead of relying solely on words may be another way to supplement information for low-literate individuals (Murphy, Chesson, Walker, Arnold, & Chesson, 2000).

Importantly, adequate health literacy is often seen as the exception rather than the rule, and both healthcare providers and health researchers need to remember to communicate in a manner that is understandable to all. Health professionals often overestimate patients’ health literacy abilities and fail to recognize that a problem exists. Because of this gap in the communication process, healthcare providers need to learn a set of communication skills that are conducive to increasing patients’ understanding. Addressing the obvious challenge of working with patients who have low health literacy, many clinicians and researchers alike have put forth guidelines to enhance the interaction between providers and patients (e.g., Parker, 2000).

Much overlap exists in these guidelines, but based on the research presented, several key components are worth note. First, healthcare providers (and researchers) need simply to be aware that low health literacy is a serious barrier for many patients and take the time to assess patients’ health literacy skills. Being sensitive to the fact that this problem exists in two out of five Americans and taking the time to cue into patient speech and behavior patterns can smooth the patient–provider interaction and be more beneficial for the patient. Second, the use of medical language should be kept to a minimum amount necessary to convey adequate and appropriate information. Instead, “living room” language is simpler and creates less confusion for a low health literate patient who may be unfamiliar with even basic medical terminology. Third, the amount of information a patient receives during each interaction should be limited and repeated throughout the visit. Because patients forget more as the amount of information increases, information for retention is best presented in small chunks. Interactive approaches, such as the “teach-back” method, can simultaneously increase patient retention of information and confirm to the provider that the patient understands what is being asked of him or her. For example, a patient can teach the doctor how to take his or her medication according to what has been discussed in the visit. Lastly, because many low health literate patients are reluctant to ask questions, providers should help their patients ask questions and even encourage them to write down any questions before an appointment or bring a spouse or family member who may overhear information the patient misses.

In addition to communicating orally with patients, it is important to design health-related materials, so all individuals can understand the health information. With the increasing complexity of the healthcare system, patient education materials are following suit, and healthcare providers are relying more and more on written materials to educate patients about disease and treatment options. Yet a large number of studies have shown that the majority of standard education materials are written at levels far exceeding the reading and comprehension abilities of most Americans (Williams et al., 2002). While the average reading skill of Americans is around the eighth grade level, one study found that only 19% of health education pamphlets for parents with infants were written below a ninth grade level—making nearly 81% of materials inaccessible for the majority of parents (Davis et al., 1994). In terms of health promotion, many public health messages may also be inaccessible for individuals with low health literacy. Health messages in magazines, on billboards, or on clinic posters informing patients about the value of preventive behaviors like flu shots or regular mammograms may be problematic for

those with low health literacy because they often require some understanding of medical knowledge (Parker, 2000).

One way to address these communication issues is by presenting only the most essential information, or presenting essential information first, which has been shown to help low-numeracy individuals to better understand information (e.g., Peters, Hibbard, Slovic, & Dieckmann, 2007). Other suggestions include receiving input or feedback from target groups to ensure the information is clearly conveyed and culturally appropriate (Batterham et al., 2016), as well as using more pictures and less text. As an example, presenting comparable information about the harms and benefits of two drugs in tables rather than in narrative text improved understanding in one study (Tait, Voepel-Lewis, Zikmund-Fisher, & Fagerlin, 2010; though see Peters et al., 2007).

Adding video to a verbal narrative may also help low health literate individuals understand materials (Vollandes et al., 2009). A meta-analysis of interventions for individuals with low health literacy found that the same intervention may have opposite effects depending on an individual's health literacy level (Sheriden et al., 2011). For example, adding traffic light signage to information aided the understanding of those with high literacy but decreased the understanding of those with low literacy. Thus, healthcare communication and messages aimed at making it easier to understand health information may not be "one size fits all."

Future Directions in Health Literacy

Health literacy is now recognized as a dynamic, multifaceted skill set (Berkman et al., 2011; Chin et al., 2011), and the US government is placing importance on increasing health literacy. Health literacy is part of the Affordable Care Act in the United States and is outlined as an objective in Healthy People 2020, and the US Department of Health and Human Services has written a "National Action Plan to Improve Health Literacy."

In the Affordable Care Act, the law required the enrollment process to be streamlined for Medicaid, the Children's Health Insurance Program, and state-based insurance exchanges that clear information be presented about insurance plans and benefits and for health literacy to be incorporated into professional training (Koh et al., 2012). In Healthy People 2020, objectives under the category of "Health Communication and Health Information Technology" related to health literacy are to improve the health literacy of the population and increase the proportion of persons who report that their healthcare providers have satisfactory communication skills. The US Department of Health and Human Services' National Action Plan to Improve Health Literacy includes seven goals to improve health literacy. These goals include promoting healthcare system changes that improve health information and communication, supporting and expanding efforts to provide culturally and linguistically appropriate health information services, and increasing basic research and evaluation of practices and interventions to improve health literacy.

Thus, the current focus in the field of health literacy is not so much on individual-level interventions aimed at increasing a single person's health literacy skills but rather system-level changes to improve how healthcare providers communicate, how healthcare systems are set up and managed, and how health communication materials are constructed. These changes are not easy to implement, nor will they provide an immediate fix for the health literacy "crisis," but they are a necessary step forward to promote and maintain optimal health for the most vulnerable patients. As one of the strongest predictors of an individual's health status is health literacy, the conversations surrounding the health literacy "crisis" should progress in the coming years, no longer overlooked and misunderstood, but evolving and expanding to improve the lives of many.

Author Biographies

Amber S. Emanuel, PhD is a lecturer in the Department of Health Education and Behavior at the University of Florida. Her research focuses on using social psychological theories to formulate and study effective health messages for eliciting health behavior change.

Kristel M. Gallagher, PhD is an assistant professor of psychology at Thiel College where she oversees the undergraduate psychology specialization in social psychology, focusing her research on the application of social psychology to health. Her research centers around the study of health behavior change, examining both theoretical and practices approach to motivation and health-related decision making. Past research includes the study of health message framing, including a meta-analysis published in the *Annals of Behavioral Medicine*.

References

- Baker, D. W., Gazmararian, J. A., Sudano, J., & Patterson, M. (2000). The association between age and health literacy among elderly persons. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 55, S368–S374. <http://dx.doi.org/10.1093/geronb/55.6.s368>
- Baker, D. W., Gazmararian, J. A., Williams, M. V., Scott, T., Parker, R. M., Green, D., ... Peel, J. (2002). Functional health literacy and the risk of hospital admission among Medicare managed care enrollees. *American Journal of Public Health*, 92, 1278–1283. <http://dx.doi.org/10.2105/ajph.92.8.1278>
- Baker, D. W., Parker, R. M., Williams, M. V., & Clark, W. S. (1998). Health literacy and the risk of hospital admission. *Journal of General Internal Medicine*, 13, 791–798. <http://dx.doi.org/10.1046/j.1525-1497.1998.00242.x>
- Batterham, R. W., Hawkins, M., Collins, P. A., Buchbinder, R., & Osborne, R. H. (2016). Health literacy: Applying current concepts to improve health services and reduce health inequalities. *Public Health*, 132, 3–12. <http://dx.doi.org/10.1016/j.puhe.2016.01.001>
- Bennett, I. M., Chen, J., Soroui, J. S., & White, S. (2009). The contribution of health literacy to disparities in self-rated health status and preventive health behaviors in older adults. *Annals of Family Medicine*, 7, 204–211. <http://dx.doi.org/10.1093/geronb/55.6.s368>
- Berkman, N. D., Sheridan, S. L., Donahue, K. E., Halpern, D. J., & Crotty, K. (2011). Low health literacy and health outcomes: An updated systematic review. *Annals of Internal Medicine*, 155, 97–107. <http://dx.doi.org/10.7326/0003-4819-155-2-201107190-00005>
- Chari, R., Warsh, J., Ketterer, T., Hossain, J., & Sharif, I. (2014). Association between health literacy and child and adolescent obesity. *Patient Education and Counseling*, 94, 61–66. <http://dx.doi.org/10.1016/j.pec.2013.09.006>
- Chin, J., Morrow, D. G., Stine-Morrow, E. A., Conner-Garcia, T., Graumlich, J. F., & Murray, M. D. (2011). The process-knowledge model of health literacy: Evidence from a componential analysis of two commonly used measures. *Journal of Health Communication*, 16, 222–241. <http://dx.doi.org/10.1080/10810730.2011.604702>
- Davis, T. C., Mayeaux, E. J., Fredrickson, D., Bocchini, J. A., Jackson, R. H., & Murphy, P. W. (1994). Reading ability of parents compared with reading level of pediatric patient education materials. *Pediatrics*, 93, 460–468.
- DeWalt, D. A., Broucksou, K. A., Hawk, V., Brach, C., Hink, A., Rudd, R., & Callahan, L. (2011). Developing and testing the health literacy universal precautions toolkit. *Nursing Outlook*, 59, 85–94. <http://dx.doi.org/10.1016/j.outlook.2010.12.002>
- Institute of Medicine. (2004). In L. Nielsen-Bohlman, A. Panzer, & D. A. Kindig (Eds.), *Health literacy: A prescription to end confusion*. Washington, DC: National Academies Press.
- Koh, H. K., Berwick, D. M., Clancy, C. M., Baur, C., Brach, C., Harris, L. M., & Zerhusen, E. G. (2012). New federal policy initiatives to boost health literacy can help the nation move beyond

- the cycle of costly 'crisis care'. *Health Affairs*, 10–1377. <http://dx.doi.org/10.1377/hlthaff.2011.1169>
- Kutner, M., Greenburg, E., Jin, Y., & Paulsen, C. (2006). The Health Literacy of America's Adults: Results from the 2003 National Assessment of Adult Literacy. NCES 2006-483. *National Center for Education Statistics*.
- Morrow, D., Clark, D., Tu, W., Wu, J., Weiner, M., Steinley, D., & Murray, M. D. (2006). Correlates of health literacy in patients with chronic heart failure. *The Gerontologist*, 46, 669–676. <http://dx.doi.org/10.1093/geront/46.5.669>
- Möttus, R., Johnson, W., Murray, C., Wolf, M. S., Starr, J. M., & Deary, I. J. (2014). Towards understanding the links between health literacy and physical health. *Health Psychology*, 33, 164–173. <http://dx.doi.org/10.1037/a0031439>
- Murphy, P. W., Chesson, A. L., Walker, L., Arnold, C. L., & Chesson, L. M. (2000). Comparing the effectiveness of video and written material for improving knowledge among sleep disorders clinic patients with limited literacy skills. *Southern Medical Journal*, 93, 297–304. <http://dx.doi.org/10.1097/00007611-200093030-00010>
- Nielsen-Bohlman, L., Panzer, A. M., & Kindig, D. A. (Eds.) (2004). *Health literacy: A prescription to end confusion*. Washington, DC: National Academies Press.
- Ong, L. M., De Haes, J. C., Hoos, A. M., & Lammes, F. B. (1995). Doctor-patient communication: A review of the literature. *Social Science & Medicine*, 40, 903–918. [http://dx.doi.org/10.1016/0277-9536\(94\)00155-m](http://dx.doi.org/10.1016/0277-9536(94)00155-m)
- Osborn, C. Y., Paasche-Orlow, M. K., Davis, T. C., & Wolf, M. S. (2007). Health literacy: An overlooked factor in understanding HIV health disparities. *American Journal of Preventive Medicine*, 33, 374–378. <http://dx.doi.org/10.1016/j.amepre.2007.07.022>
- Parker, R. (2000). Health literacy: A challenge for American patients and their health care providers. *Health Promotion International*, 15, 277–283. <http://dx.doi.org/10.1093/heapro/15.4.277>
- Parker, R. M., Ratzan, S. C., & Lurie, N. (2003). Health literacy: A policy challenge for advancing high-quality health care. *Health Affairs*, 22, 147–153. <http://dx.doi.org/10.1377/hlthaff.22.4.147>
- Peters, E., Hibbard, J., Slovic, P., & Dieckmann, N. (2007). Numeracy skill and the communication, comprehension, and use of risk-benefit information. *Health Affairs*, 26, 741–748. <http://dx.doi.org/10.1377/hlthaff.26.3.741>
- Rodríguez, V., Andrade, A. D., García-Retamero, R., Anam, R., Rodríguez, R., Lisigurski, M., ... Ruiz, J. G. (2013). Health literacy, numeracy, and graphical literacy among veterans in primary care and their effect on shared decision making and trust in physicians. *Journal of Health Communication*, 18, 273–289. <http://dx.doi.org/10.1080/10810730.2013.829137>
- Sentell, T. L., & Halpin, H. A. (2006). Importance of adult literacy in understanding health disparities. *Journal of General Internal Medicine*, 21, 862–866. <http://dx.doi.org/10.1111/j.1525-1497.2006.00538.x>
- Tait, A. R., Voepel-Lewis, T., Zikmund-Fisher, B. J., & Fagerlin, A. (2010). The effect of format on parents' understanding of the risks and benefits of clinical research: A comparison between text, tables, and graphics. *Journal of Health Communication*, 15, 487–501. <http://dx.doi.org/10.1080/10810730.2010.492560>
- Vernon, J. A., Trujillo, A., Rosenbaum, S. J., & DeBuono, B. (2007). *Low health literacy: Implications for national health policy*. Washington, DC: Department of Health Policy, School of Public Health and Health Services, George Washington University.
- Volandes, A. E., Paasche-Orlow, M., Gillick, M. R., Cook, E. F., Shaykevich, S., Abbo, E. D., & Lehmann, L. (2008). Health literacy not race predicts end-of-life care preferences. *Journal of Palliative Medicine*, 11, 754–762. <http://dx.doi.org/10.1089/jpm.2007.0224>
- Williams, M. V., Davis, T., Parker, R. M., & Weiss, B. D. (2002). The role of health literacy in patient-physician communication. *Journal of Family Medicine*, 34, 383–389.
- World Health Organization. (2013). *The solid facts: Health literacy*. Copenhagen, Denmark: Author.
- Agency for Health Research and Quality. (2016). Health literacy measurement tools. Retrieved from <https://www.ahrq.gov/health-literacy/quality-resources/tools/literacy/index.html>

- Baker, D. W., Wolf, M. S., Feinglass, J., Thompson, J. A., Gazmararian, J. A., & Huang, J. (2007). Health literacy and mortality among elderly persons. *Archives of internal medicine*, 167(14), 1503–1509.
- Volandes, A. E., Paasche-Orlow, M. K., Barry, M. J., Gillick, M. R., Minaker, K. L., Chang, Y., ... Mitchell, S. L. (2009). Video decision support tool for advance care planning in dementia: Randomised controlled trial. *BMJ*, 338, b2159.
- Sheridan, S. L., Halpern, D. J., Viera, A. J., Berkman, N. D., Donahue, K. E., & Crotty, K. (2011). Interventions for individuals with low health literacy: A systematic review. *Journal of Health Communication*, 16, 30–54.

Suggested Reading

- Agency for Healthcare Research and Quality. (2016). Health literacy measurement tools—revised. Retrieved from <https://www.ahrq.gov/professionals/quality-patient-safety/quality-resources/tools/literacy/index.html>.
- Institute of Medicine. (2004). In L. Nielsen-Bohlman, A. Panzer, & D. A. Kindig (Eds.), *Health literacy: A prescription to end confusion*. Washington, DC: National Academies Press. Retrieved from <https://www.nap.edu/catalog/10883/health-literacy-a-prescription-to-end-confusion>
- Osborne, H. (2004). *Health literacy from A to Z: Practical ways to communicate your health message*. Sudbury, MA: Jones and Bartlett Publishers.
- U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. (2010). *National action plan to improve health literacy*. Washington, DC: Author.
- Zarcadoolas, C., Pleasant, A., & Greer, D. S. (2009). *Advancing health literacy: A framework for understanding and action*. San Francisco, CA: Wiley.

Healthcare Treatment Seeking and Avoidance

Nils Olsen, Phillip J. Moore, Steven Hyde,
and Lauren A. Fowler

*Department of Psychological and Brain Sciences, The George Washington
University, Washington, DC, USA*

It is health that is real wealth and not pieces of gold and silver.

—Mahatma Gandhi

Given the defining impact of mental and physical health on well-being, effective healthcare is a crucial aspect of human life. Beyond its direct impact within the personal and public domains, healthcare also plays a central economic role in society, representing over \$4 trillion of spending within the United States in 2015 (Munro, 2014).

In light of the above, even the most advanced, comprehensive healthcare system is only effective to the extent that it is utilized, for when people fail to seek—or actively avoid—needed treatment, they not only threaten their own health but also increase health risks (and costs) to the broader public. It is thus crucial to understand treatment seeking and avoidance, including how they are developed and maintained, and their influence on individuals and society as a whole. To that end, this entry examines the following fundamental questions: (a) What constitutes healthcare treatment seeking and avoidance? (b) What factors influence seeking and avoidance? (c) What are their effects? (d) How can we understand treatment seeking and avoidance in the context of our current social and healthcare systems? (e) How can we improve treatment seeking and the corresponding outcomes?

Defining Treatment Seeking and Avoidance

Because treatment seeking and avoidance come in many forms, it is important to define them in the current context. For the purposes of this chapter, we are defining healthcare *treatment*

seeking as the active pursuit of healthcare resources for a health concern, including the following levels: (a) awareness (a necessary precursor), (b) information seeking (either independently or via professional consultation), (c) diagnosis, and, if necessary, (d) intervention (e.g., medication, equipment, exercise, and/or invasive procedures). We are defining *treatment avoidance* as a volitional forestalling of treatment for a recognized health problem, which includes a continuum of behavior from passive resistance (e.g., psychological denial) to delaying treatment and active refusal of care.

Factors Influencing Treatment Seeking and Avoidance

Demographic Factors

Individuals in poverty tend to have a general distrust of medical services, perceive these services as risky, and often express fears of being judged, losing resources, losing their children, and not receiving proper care (Canvin, Jones, Marttila, Burström, & Whitehead, 2007). Within these contexts, some physicians err on the side of overly vigilant treatment by sending individuals with reported chest pain to an intensive care unit—an action that can be unnecessarily dangerous if those individuals are not severely ill (Gigerenzer, 2007). Even in cases when medical services are extremely inexpensive or free of charge (e.g., in Montreal, Canada), avoidance can occur with people who have limited financial resources, such that aside from typical accessibility issues, avoidance has been linked with difficulties in gauging the seriousness of an issue and pride in one's masculinity (Dupéré, O'Neill, & Koninck, 2012). Individuals in poverty are also more likely to use an emergency department as their primary source of care, and this choice can lead to added expense, exposure to illness, and an increased length of stay (Lucas et al., 2009).

The highest levels of avoidance have been primarily found among individuals living in rural areas, younger men (18–35), and the uninsured, along with those reporting low trust in physicians, low confidence in personal health, and no consistently regular physician (Spleen, Lengerich, Camacho, & Vanderpool, 2014).

Psychosocial Factors

The results of a qualitative study in which individuals were interviewed to explain why they declined treatment highlighted the following reasons for avoidance: self-efficacy, threat minimization, fatalism and faith, distrust, need for additional information, desire to not know the diagnosis, futility, postponing intentions, physical discomfort from treatment, and quality of life (Sharf, Stelljes, & Gordon, 2005). Other reasons for avoiding care have included anticipating long wait times, believing that one can self-treat, expecting difficulty with obtaining an appointment, and fearing immigration issues (Asch, Leake, Anderson, & Gelberg, 1998).

A meta-analysis of compliance studies revealed that there are *hard* factors (e.g., duration of treatment, accessibility) and *soft* factors (e.g., beliefs, attitudes) that influence compliance, and chronic patients go through a cost–benefit analysis to determine if they would like to continue with treatment (Jin, Sklar, Min Sen Oh, & Chuen Li, 2008). A second meta-analysis revealed that men often avoid seeking medical care because they may prefer to avoid expressing emotions and because of health-based concerns, in some cases due to poor communication with healthcare providers, as well as fear, anxiety, and embarrassment (Yousaf, Grunfeld, & Hunter, 2015).

Those who felt their physicians were listening to them were less likely to avoid treatment (of the physical and psychological variety) during the following year, and certain individual differences (degree of distress and health status) were associated with avoidance level (Moore et al., 2004). Trust is particularly salient when it comes to protocol adherence and patients' reuse intentions, such that patients' trust in physicians has been linked with medical outcomes, physicians' medical training, physicians' prioritization of patients' treatment, and the extent to which physicians treat patients in a polite, friendly, and respectful manner (Olsen, Lind, Kennedy, & Chrabaszcz, 2008).

Additionally, physical embarrassment and concerns with being judged have been associated with avoidance behaviors (Consedine, Krivoshekova, & Harris, 2007), and others have further supported links between fear of embarrassment and avoidance, along with not having enough time to worry about oneself, minimizing health problems, and viewing health issues as normal (Hägglund & Wadensten, 2007).

Personality

Within the personality realm, help seeking has been linked to neuroticism, openness, agreeableness, and conscientiousness. The likelihood that a person seeks periodic health screening tends to be maximized at moderate levels of neuroticism; whereas extroversion and openness were negatively associated with the seeking of medical care, conscientiousness was positively correlated with care (Armon & Toker, 2013).

Type and Intensity of Health Problem

A study of 50 years of variations in patients' adherence to medical recommendations demonstrated that adherence levels can vary by disease (e.g., high for HIV, low for diabetes) and that in select (rare) instances, nonadherence may actually be adaptive (DiMatteo, 2004). Distrust in, and fear of, the medical community (whether warranted or not) can lead one to avoid medical care. For example, when it comes to colorectal cancer screenings within an ethnically diverse sample of adults, a fear of pain was associated with avoiding treatment and/or screenings (Bynum, Davis, Green, & Katz, 2012).

A meta-analysis of health beliefs, disease severity, and patient adherence revealed a possible curvilinear relationship between illness severity and propensity to secure care (DiMatteo, Haskard, & Williams, 2007), such that the seeking of care is highest in individuals with *moderately* severe illnesses.

Dynamic assessments of disease screenings have revealed that when a disease is serious and treatable, people tend to seek screens; however, when a disease is serious and untreatable, people tend to avoid them (Dawson, Savitsky, & Dunning, 2006). Perceived likelihood that a specific fear might be confirmed can also determine how individuals behave. For example, individuals who thought a cancer was incurable were less likely to seek medical care (Persoskie, Ferrer, & Klein, 2013).

Type and Source of Medical Treatment

People have been found to react defensively to threatening health-promoting information if it is relevant to them (van 't Riet & Ruiter, 2013). When seeking information, accessibility can override credibility, as health information is gleaned from multiple sources (Johnson, 2014). On the proactive front, and when considering vaccinations, the perceived likelihood of,

susceptibility to, and severity of possible illnesses were all positively correlated with seeking vaccinations (Brewer et al., 2007). Intention to vaccinate, on the other hand, has been found to be negatively correlated with the perceived risk of vaccinating—particularly via information gathered from vaccine-critical websites (Betsch, Renkewitz, Betsch, & Ulshöfer, 2010).

Impact of Treatment Seeking and Avoidance

Outcomes related to the seeking and avoidance of medical treatment include quality of life, medical costs (e.g., emergency department care, end of life), vaccination, diagnoses (e.g., identifying cancer at stage 1 vs. stage 4), script writing (prescribing), and testing. Of course, issues of overdiagnosis can surface, such that sometimes even a correct diagnosis does not necessarily lead to better health. In other cases, seeking medical care can lead to patterns of overmedicating. For example, while acute upper respiratory tract infection is considered to be one of the most common illnesses that physicians report in emergency departments, and given that it is generally not a serious condition, physicians tend to overprescribe antibiotics for patients in many of these cases (Xu et al., 2013).

Treatment Seeking and Avoidance in the Healthcare Context

Federman et al. (2001) sampled nearly 3,000 primary care patients, and after controlling for patient demographics, health status, technical satisfaction, and site of care, patients' avoidance of medical care was found to be explained by 2 predictors: dissatisfaction with time spent with physicians and perceptions that physicians were not listening to patients (as cited in Moore et al., 2004). Moore et al. (2004) sampled over 1,000 adults to measure the impact of physician–patient relationships as a driver of treatment avoidance for a confirmed medical or psychological issue. Of the five key dimensions that Moore et al. (2004) explored (time waiting, time with physicians, respect displayed by physicians, physicians' confirmation of patients' understanding, and physicians listening to patients' concerns), all five dimensions were correlated with each other, and all but *time waiting for physician* were significantly correlated with *avoidance* of medical and psychological treatment. Further, when the four dimensions correlated with avoidance were combined, the more patients perceived that physicians were *listening to them*, the less likely they avoided treatment for medical or psychological issues (Moore et al., 2004). Thus, according to Moore et al. (2004), patients' relationships with physicians significantly determined the extent to which they sought medical treatment, and the inclusion of patients' preferences was critical to developing and maintaining a shared-decision treatment strategy—all of which helped to reduce patient ambiguity, adherence issues, and avoidance of medical care (Sieber & Kaplan, 2000).

Is Seeking Always Positive and Avoidance Always Negative?

Certainly there are instances in which one's seeking of medical treatment is so vigilant that it approaches an excessive level (e.g., one can envision a positive correlation between obsessive–compulsive disorder and treatment seeking). There are likewise instances when avoiding treatment can have a positive impact. For example, when seeking treatment for a cancer with a timeline that exceeds a person's expected lifespan, or if relative to a much less invasive, more

conservative therapy, one must consider surgery that includes risks of infection, physical complications, medical errors, and even death, in these circumstances, it may (depending on the patient) be optimal to avoid immediate treatment.

Summary, Conclusions, and Recommendations

It appears that treatment accessibility, irrational fear assessment, and proactive vigilance on the part of individuals seeking care are just a few of the factors explaining a highly dynamic and complex relationship between individuals seeking medical treatment and those providing it. It is also true that patients can optimize their outcomes by taking an active role in the maintenance and treatment of their health.

Incentive Programs

El-Sadr and Branson (2015) examined the 2-year impact of financial incentives on patients' willingness to seek care and adhere to HIV medication protocols. To motivate patients along the lines of follow-up testing and appointments, patients identified as HIV+ received \$25 per lab test and \$100 per clinical appointment. Low viral counts signaled patients' adherence to their HIV medication protocols, and if patients' blood tests registered low HIV levels, patients could receive a \$70 gift card every 3 months. The results were, in some ways, counterintuitive. Despite the opportunity to receive a \$125 voucher for labs and follow-up care at sites offering incentives, patients in *both* the incentive and non-incentive conditions improved their follow-up with lab tests and appointments (i.e., no outcome differences). Ultimately, patients receiving financial incentives at sites with higher pre-study HIV showed the greatest HIV infection control (El-Sadr & Branson, 2015).

One possible explanation for treatment avoidance is that the traditional (paternalistic) approach of physicians as expert advisors does not always lead to optimal patient outcomes (Tefera & Olsen, 2015). When it comes to avoiding negative health behaviors (e.g., smoking) or approaching positive ones (e.g., HIV medication adherence), it has become increasingly important for architects of incentive programs—designed to improve patients' outcomes and quality of life—to consider dynamic approaches that include research from the medical, psychological, and behavioral economics literatures. Such an interdisciplinary marriage of data could better inform healthcare practitioners and policy makers alike, as they strive toward exceptional patient care and as patients look to make optimal healthcare decisions (Tefera & Olsen, 2015).

Wearable Technology: Putting Patients in the Driver's Seat

With the onset of an industry geared toward wearable health tracking devices, recent developments include the following technologies: accelerometers, consumable pills, biosensors embedded within a patch, and crystals inserted into a wearable skin-like attachment—all of which can track and transmit data to a corresponding application or companion software (e.g., heart rate, temperature, breathing rate, smoking covariates, tissue pressurization, steps taken, body position; Kosir, 2015). There are even reports of a smart bra that monitors the condition and rhythm of breast tissue to alert the wearer of possible breast cancers and smart contact lenses that utilize the user's natural tears to measure glucose levels (e.g., for diabetes care; Kosir, 2015).

Health Treatment and Health Maintenance

As important as maintaining one's medical treatments has become, the maintenance of one's health is also crucial. From diet to exercise to stress mitigation, balancing one's opportunities for health promotion with a dynamic lifestyle is critical in today's fast-paced environment. Perhaps as the availability of interactive wearable health technology increases, individuals can continue to monitor their own health in real time and present detailed and dynamic data to healthcare providers. It is with this potential trend in place that individuals can focus not only on maximizing the quality of their healthcare but also on (proactively) optimizing the quality of their health.

À votre santé.

Author Biographies

Nils Olsen (PhD, Social Psychology, University of North Carolina–Chapel Hill) is an assistant professor of organizational sciences at the George Washington University and conducts research within the following domains—(a) individual decision making (e.g., medical), (b) strategic decision making (e.g., Olympic Games), and (c) procedural justice (e.g., physician–patient, lawyer–client)—and has publications appearing in the *Journal of Personality and Social Psychology*, *Academic Emergency Medicine*, and *Handbook of the London 2012 Olympic and Paralympic Games*.

Phillip J. Moore (PhD, Social Psychology, University of California–San Diego) is an associate professor of applied social psychology at the George Washington University and conducts research that is designed to explore how psychological, environmental, and biological factors combine to influence important health-related decisions, behaviors, and conditions (e.g., obesity-related outcomes, terrorism and other emergencies, stressful events) and has publications appearing in the *Journal of Personality and Social Psychology*, *Cognition and Emotion*, and *Health Psychology*.

Steven Hyde (PhD student, Organizational Behavior, University of Texas–San Antonio). He received his master's degree, in 2015, in organizational management from the George Washington University and his bachelor's degree in psychology in 2013 from Southern Utah University. Steven's primary researcher interests are in decision making and behavioral strategy.

Lauren A. Fowler (PhD student, Applied Social Psychology, The George Washington University) received her BS in psychology and mathematics with a concentration in statistics from Eastern Michigan University. Her research focuses on the biological, psychological, and social determinants of weight-related health behaviors.

References

- Armon, G., & Toker, S. (2013). The role of personality in predicting repeat participation in periodic health screening. *Journal of Personality*, 81, 452–464. doi:10.1111/jopy.12021
- Asch, S., Leake, B., Anderson, R., & Gelberg, L. (1998). Why do symptomatic patients delay obtaining care for tuberculosis? *American Journal of Respiratory and Critical Care Medicine*, 157, 1244–1248. doi:10.1164/ajrcrm.157.4.9709071
- Betsch, C., Renkewitz, F., Betsch, T., & Ulshöfer, C. (2010). The influence of vaccine-critical websites on perceiving vaccination risks. *Journal of Health Psychology*, 15, 446–455. doi:10.1177/1359105309353647

- Brewer, N. T., Chapman, G. B., Gibbons, F. X., Gerrard, M., McCaul, K. D., & Weinstein, N. D. (2007). Meta-analysis of the relationship between risk perception and health behavior: The example of vaccination. *Health Psychology, 26*, 136–145. doi:10.1037/0278-6133.26.2.136
- Bynum, S. A., Davis, J. L., Green, B. L., & Katz, R. V. (2012). Unwillingness to participate in colorectal cancer screening: Examining fears, attitudes, and medical mistrust in an ethnically diverse sample of adults 50 years and older. *American Journal of Health Promotion, 26*, 295–300. doi:10.4278/ajhp.110113-QUAN-20
- Canvin, K., Jones, C., Marttila, A., Burström, B., & Whitehead, M. (2007). Can I risk using public services? Perceived consequences of seeking help and health care among households living in poverty: Qualitative study. *Journal of Epidemiology and Community Health, 61*, 984–989. doi:10.1136/jech.2006.058404
- Consedine, N. S., Krivoshekova, Y. S., & Harris, C. R. (2007). Bodily embarrassment and judgment concern as separable factors in the measurement of medical embarrassment: Psychometric development and links to treatment-seeking outcomes. *British Journal of Health Psychology, 12*, 439–462. doi:10.1348/135910706X118747
- Dawson, E., Savitsky, K., & Dunning, D. (2006). “Don’t tell me, I don’t want to know”: Understanding people’s reluctance to obtain medical diagnostic information. *Journal of Applied Social Psychology, 36*, 751–768. doi:10.1111/j.0021-9029.2006.00028.x
- DiMatteo, M. R. (2004). Variations in patients’ adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care, 42*, 200–209. doi:10.1097/01.mlr.0000114908.90348.f9
- DiMatteo, M. R., Haskard, K. B., & Williams, S. L. (2007). Health beliefs, disease severity, and patient adherence: A meta-analysis. *Medical Care, 45*, 521–528. doi:10.1097/MLR.0b013e318032937e
- Dupéré, S., O’Neill, M., & Koninck, M. D. (2012). Why men experiencing deep poverty in Montréal avoid using health and social services in times of crisis. *Journal of Health Care for the Poor and Underserved, 23*, 781–796. doi:10.1353/hpu.2012.0071
- El-Sadr, W., & Branson, B. (2015). TLC-Plus: A study to evaluate the feasibility of an enhancement test, link to care, plus treat approach for HIV prevention in the United States. *HPTN 065 (TLC-Plus) Overall Study Fact Sheet, VI.0*. Retrieved from <https://www.hptn.org/research/studies/hptn065>
- Federman, A. D., Cook, E. F., Russell, S., Puopolo, A. L., Haas, J. S., Brennan, T. A., & Burstin, H. R. (2001). Intention to discontinue care among primary care patients: Influence of physician behavior and process of care. *Journal of General Internal Medicine, 16*, 668–674. doi:10.1111/j.1525-1497.2001.01028.x
- Gigerenzer, G. (2007). *Gut feelings: The intelligence of the unconscious*. New York, NY: Viking. ISBN: 978-0-670-03863-3
- Hägglund, D., & Wadensten, B. (2007). Fear of humiliation inhibits women’s care-seeking behaviour for long-term urinary incontinence. *Scandinavian Journal of Caring Sciences, 21*, 305–312. doi:10.1111/j.1471-6712.2007.00481.x
- Jin, J., Sklar, G. E., Min Sen Oh, V., & Chuen Li, S. (2008). Factors affecting therapeutic compliance: A review from the patient’s perspective. *Therapeutics and Clinical Risk Management, 4*, 269–286. ISSN: 1176-6336
- Johnson, J. D. (2014). Health-related information seeking: Is it worth it? *Information Processing & Management, 50*, 708–717. doi:10.1016/j.ipm.2014.06.001
- Kosir, S. (2015). Wearables in healthcare. Retrieved from <https://www.wearable-technologies.com/2015/04/wearables-in-healthcare/>
- Lucas, R., Farley, H., Twanmoh, J., Urumov, A., Olsen, N., Evans, B., & Kabiri, H. (2009). Emergency department patient flow: The influence of hospital census variables on emergency department length of stay. *Academic Emergency Medicine, 16*(7), 597–602. doi:10.1111/j.1553-2712.2009.00397.x
- Moore, P. J., Sickel, A. E., Malat, J., Williams, D., Jackson, J., & Adler, N. E. (2004). Psychosocial factors in medical and psychological treatment avoidance: The role of the doctor-patient relationship. *Journal of Health Psychology, 9*, 421–433. doi:10.1177/1359105304042351

- Munro, D. (February 2, 2014). Annual U.S. Healthcare Spending Hits 3.8 Trillion. *Forbes, Pharma and Healthcare*. Retrieved from <http://www.forbes.com/sites/danmunro/2014/02/02/annual-u-s-healthcare-spending-hits-3-8-trillion/>
- Olsen, N., Lind, E. A., Kennedy, C., & Chrabaszcz, J. (2008). Trust, protocol adherence, and re-use intentions: A look at legal clients and medical patients. *20th Annual Convention of the Association for Psychological Science*, Chicago, IL.
- Persoskie, A., Ferrer, R. A., & Klein, W. M. P. (2013). Association of cancer worry and perceived risk with doctor avoidance: An analysis of information avoidance in a nationally representative US sample. *Journal of Behavioral Medicine*, 37, 977–987. doi:10.1007/s10865-013-9537-2
- Sharf, B. F., Stelljes, L. A., & Gordon, H. S. (2005). ‘A little bitty spot and I’m a big man’: Patients’ perspectives on refusing diagnosis or treatment for lung cancer. *Psycho-Oncology*, 14, 636–646. doi:10.1002/pon.885
- Sieber, W. J., & Kaplan, R. M. (2000). Informed adherence: The need for shared medical decision making. *Controlled Clinical Trials*, 21, 233–240. doi:10.1016/S0197-2456(00)00084-2
- Spleen, A. M., Lengerich, E. J., Camacho, F. T., & Vanderpool, R. C. (2014). Health care avoidance among rural populations: Results from a nationally representative survey. *The Journal of Rural Health*, 30, 79–88. doi:10.1111/jrh.12032
- Tefera, L., & Olsen, N. (2015). *Paying patients to take their own medication?* (Unpublished manuscript). Washington, DC: Department of Emergency Medicine and Department of Organizational Sciences and Communication, The George Washington University.
- van ‘t Riet, J., & Ruiter, R. A. C. (2013). Defensive reactions to health-promoting information: An overview and implications for future research. *Health Psychology Review*, 7, S104–S136. doi:10.1080/17437199.2011.606782
- Xu, T., Roberts, D., Sulapas, I., Martinez, O., Berk, J., & Baldwin, J. (2013). Over-prescribing of antibiotics and imaging in the management of uncomplicated URIs in emergency departments. *BMC Emergency Medicine*, 13, 1–6. doi:10.1186/1471-227X-13-7
- Yousaf, O., Grunfeld, E. A., & Hunter, M. S. (2015). A systematic review of the factors associated with delays in medical and psychological help-seeking among men. *Health Psychology Review*, 9, 264–276. doi:10.1080/17437199.2013.840954

Suggested Reading

- DiMatteo, M. R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology*, 23, 207–218. doi:10.1037/0278-6133.23.2.207
- Sawesi, S., Rashrash, M., Phalakornkule, K., Carpenter, J. S., & Jones, J. F. (2016). The impact of information technology on patient engagement and health behavior change: A systematic review of the literature. *Journal of Medical Internet Research—Medical Informatics*, 4, e1. doi:10.2196/medinform.4514
- Webb, T. L., Joseph, J., Yardley, L., & Michie, S. (2010). Using the internet to promote health behavior change: A systematic review and meta-analysis of the impact of theoretical basis, use of behavior change techniques, and mode of delivery on efficacy. *Journal of Medical Internet Research*, 12, e4. doi:10.2196/jmir.1376
- Williams, D. R., Priest, N., & Anderson, N. B. (2016). Understanding associations among race, socioeconomic status, and health: Patterns and prospects. *Health Psychology*, 35, 407–411. doi:10.1037/hea0000242

Health-Related Uncertainty

Jada G. Hamilton¹, Margaux C. Genoff¹,
and Paul K. J. Han²

¹*Department of Psychiatry and Behavioral Sciences, Memorial Sloan
Kettering Cancer Center, New York, NY, USA*

²*Center for Outcomes Research and Evaluation, Maine Medical Center
Research Institute, Portland, ME, USA*

Uncertainty is the subjective perception of ignorance, a state of knowing that there is something that one does not know (Han, Klein, & Arora, 2011). In medicine and healthcare, people are frequently confronted with uncertainty. For example, uncertainty can be perceived when a patient becomes aware of the probabilities of medication side effects, when a person hears a news report on how expert panels disagree about cancer screening recommendations, when parents consider whether their child should have a new vaccine that has a limited and evolving evidence base, or when a physician is deciding between multiple diagnoses that fit a patient's nonspecific set of symptoms.

These and myriad other instances of health-related uncertainty present significant challenges for laypeople, patients, healthcare providers, and health policy makers. For instance, uncertainty is a substantial source of stress for patients and families in acute and chronic disease contexts (e.g., Mishel, 1997). Research indicates that health-related uncertainty has implications for people's emotional well-being, decision making, and health behaviors, and investigating these diverse responses will allow researchers and clinicians to develop strategies to facilitate people's understanding of and adaptation to uncertainty. This chapter provides a concise overview of different types of uncertainty present in healthcare, ways in which people respond to uncertainty, and approaches to managing uncertainty, illustrated with examples and data from the cancer prevention and control context.

Defining Uncertainty

Establishing a clear definition of health-related uncertainty is important for promoting research and improving understanding of this phenomenon, and scholars from disciplines including

communication, decision sciences, engineering, health services, nursing, and psychology have presented various descriptions of this construct (e.g., Babrow, Kasch, & Ford, 1998; Brashers, 2001; Mishel, 1997). Here we focus on a conceptualization developed by Han et al. (2011), which integrates these perspectives into a multidimensional taxonomy of uncertainty in health-care. This taxonomy provides a comprehensive tool for categorizing the varieties of uncertainty in healthcare by specifying three distinct dimensions of uncertainty: source, issue, and locus.

Source, the type of uncertainty, includes probability (uncertainty about the likelihood or risk of an event arising from the randomness or indeterminacy of the future), ambiguity (uncertainty regarding the strength, validity, consistency, or adequacy of risk estimates or risk information), and complexity (aspects of an event that make it difficult to understand, such as multiple possible causes or outcomes; Han et al., 2011). Delineating the different sources of health-related uncertainty underscores the fact that uncertainty is not entirely synonymous with a lack of knowledge, but rather can arise due to the characteristics and quality of available information. For example, uncertainty due to probability reflects in part the conceptual challenges inherent to translating risk estimates derived from a population to the level of the individual, and uncertainty due to ambiguity can be present in situations where a large body of evidence exists yet is conflicting in nature.

The second dimension is *issue*, or the substantive content of the uncertainty, which includes scientific uncertainty (disease centered, regarding aspects such as diagnosis or prognosis), practical uncertainty (healthcare system centered, regarding the structure or process of health-care such as procedures to access care), and personal uncertainty (patient centered, regarding psychosocial or existential aspects such as implications of disease for one's personal relationships; Han et al., 2011). The dimension of issue reflects the multiple contexts and challenges that span the trajectories of acute and chronic diseases. Furthermore, the conceptualization of practical and personal uncertainty incorporates issues of great interest to individuals facing a health threat, consistent with the current emphasis on patient-related outcomes in health.

The third dimension of uncertainty is *locus*, or where the perception of uncertainty resides. Uncertainty can exist in the mind of the patient, the healthcare provider, neither, or both (Han et al., 2011). Dyadic exchanges between patients and providers are a fundamental aspect of medicine necessary for the diagnosis and management of disease and for the development of a shared understanding of relevant information and its associated uncertainties. By integrating the dimension of locus, the taxonomy explicitly acknowledges this relational nature of healthcare.

Responses to Uncertainty

Han et al.' (2011) taxonomy provides a useful framework for categorizing varieties of health-related uncertainty; however, it does not make predictions about how individuals will respond to uncertainty. Past work on this topic has generally focused on individual responses to different sources (e.g., probability, ambiguity) or issues (e.g., scientific, personal) of uncertainty and confirms that health-related uncertainty can have distinct effects on psychological and behavioral outcomes.

Psychological Outcomes

Most research suggests that health-related uncertainty has adverse effects on people's cognitions and emotions. Some of these challenges arise from how people process uncertain information. The cognitive demands of information that is high in volume and complex in nature

can cause people to experience a sense of confusion and information overload (Babrow et al., 1998). Furthermore, people tend to utilize heuristics, or mental shortcuts, to make inferences when information about the probability of future events is limited, which can lead to systematic biases and errors in decision making and judgment. Consistent with a phenomenon known as “ambiguity aversion” (Ellsberg, 1961), people also tend to form pessimistic appraisals of risk information that is lacking in precision, reliability, credibility, or adequacy (for additional details, see chapter in this volume by Carpenter & Han). For example, study participants who were presented with an ambiguous quantitative risk estimate of the likelihood of drug side effects (operationalized as a numerical range versus a single point estimate) reported poorer comprehension, greater perceptions of risk for the side effects, and lower perceptions of credibility and trust in the individual conveying the risk estimate (Longman, Turner, King, & McCaffery, 2012).

Both scientific and personal issues of uncertainty have been associated with emotional outcomes in the cancer context. Uncertainty about one’s symptoms, prognosis, and social and physical functioning can be extremely stressful for cancer patients and survivors. Cross-sectional studies have indicated that greater perceptions of cancer-related uncertainty are associated with poorer emotional well-being among cancer patients and survivors (e.g., Hall, Mishel, & Germino, 2014). Similarly, longitudinal data indicate that decreased perceptions of cancer-related uncertainty are associated with improvements in global quality of life over time among prostate cancer patients and their spouses (Song et al., 2011).

Behavioral Outcomes

In the presence of uncertainty, people can struggle to make medical decisions. For example, parents of children with Fanconi anemia (a rare inherited cancer predisposition syndrome) reported greater difficulty in making a decision about a risky medical treatment and were less likely to choose the treatment, when they perceived greater ambiguity due to conflicting expert opinions and greater uncertainty due to the probabilistic nature of treatment outcomes, respectively (Hamilton et al., 2013). Mediation analyses suggested that parents’ perceived uncertainties increased their emotional concerns about the treatment, which in turn promoted these adverse decisional outcomes. Similarly, an investigation of patient–physician communication revealed that breast cancer patients experienced poorer surgical decisional satisfaction when their physicians communicated greater scientific uncertainty regarding surgical options (Politi, Clark, Ombao, Dizon, & Elwyn, 2011). However, patients’ level of involvement in the decision-making process moderated this association such that dissatisfaction following the communication of scientific uncertainty was greatest among patients who were less involved in the decision.

It seems intuitive that people would seek information when confronted with uncertainty; yet in some instances people choose to actively avoid information (Brashers, 2001). Avoiding health information that is ambiguous (e.g., information perceived as untrustworthy, contradictory, or imprecise) or complex in nature can allow people to minimize feelings of confusion; in other cases, avoiding information allows people to retain a beneficial sense of uncertainty and hope about their future health.

Physical Outcomes

A small body of evidence across diverse samples suggests that health-related uncertainty may affect people’s physical well-being. For instance, greater perceptions of cancer-related

uncertainty have been associated with increased fatigue and insomnia among breast cancer survivors (Hall et al., 2014). Potential associations between health-related uncertainty and physical functioning warrant additional exploration, as does the extent to which such relationships may be explained by people's psychological responses to uncertainty.

Individual Differences in Responses to Uncertainty

Although people have unique reactions to health-related uncertainty, their stable traits and personality characteristics can lead to some predictable psychological and behavioral responses. Broadly speaking, these individual characteristics reflect specific tendencies for how people interpret and understand uncertain information and situations.

One relevant trait is *intolerance of uncertainty*, a dispositional characteristic involving a set of emotional, cognitive, and behavioral responses to an unknown outcome. This trait is theorized to arise from negative beliefs about uncertainty, and uncertainty-intolerant people tend to interpret uncertain situations as threatening and anxiety provoking (Koerner & Dugas, 2008). Intolerance of uncertainty has been conceptualized as both a desire for predictability and uncertainty paralysis, a tendency to avoid decision making or action in the presence of uncertainty (Bredemeier & Berenbaum, 2008). People with high desire for predictability are motivated to feel more certain about the future, require less information to feel certain, and are selective about the information they seek out (Bredemeier & Berenbaum, 2008). Uncertainty paralysis causes people to overestimate the likelihood of negative outcomes (Bredemeier & Berenbaum, 2008), which may contribute to avoidance of action in uncertain situations.

Their interpretation of uncertain health threats as unacceptable should influence uncertainty-intolerant people's information seeking and health behaviors. Consistent with this prediction, Rosen, Knauper, and Sammut (2007) study of women's HPV knowledge found that higher intolerance of uncertainty was linked to greater behaviors associated with information monitoring (such as intentions to discuss HPV with other people) and preferences for clinicians to spend more time discussing HPV test results.

Need for cognitive closure, an aversion toward uncertainty and ambiguity with a preference for stability in beliefs and expectations (Kruglanski & Webster, 1996), is another trait that influences people's responses to health-related uncertainty. Need for cognitive closure is a motivational tendency to pursue information, and this need can also fluctuate as a result of situational factors (e.g., fatigue, noise) that place demands on one's information-processing abilities (Kruglanski & Webster, 1996). Individuals with a heightened need for closure, whether due to dispositional or situational factors, are motivated to seek out information to obtain an answer that eliminates confusion and uncertainty. This need for closure may be at odds with the need for validity in information processing, triggering an urgency to resolve uncertainty at the expense of an exhaustive collection of information (Kruglanski & Webster, 1996).

Although empirical evidence is limited, this trait has been associated with cancer-related behaviors and emotions. Eiser and Cole (2002) observed that women with a higher need for cognitive closure were more likely to express intentions to have cervical cancer screening or to have undergone screening than were women with a lower need for cognitive closure. The motivational drive to obtain definitive information may prompt cancer screening among those with greater need for closure. In addition, Hamilton et al. (2012) demonstrated that the extent to which parents of children with Fanconi anemia experienced the phenomenon of ambiguity aversion (i.e., adverse psychological responses to ambiguous information; Ellsberg,

1961) was dependent on their need for cognitive closure. Specifically, among parents with a high need for cognitive closure, greater perceptions of ambiguity about their child's medical treatment were strongly associated with increased worry about the treatment; this relationship was not observed among parents with a low need for closure. Individuals with a higher need for cognitive closure may benefit from targeted efforts to manage affective implications of health-related uncertainty.

Dispositional optimism can help some people to handle uncertainty in healthcare, including the fundamentally unpredictable risk of cancer. Optimism is a generalized expectancy for positive rather than negative outcomes, and those who are optimistic either see positive outcomes in all situations or disassociate their mood from external factors (see Carver, Scheier, & Segerstrom, 2010). Dispositional optimism is associated with lower emotional distress and the use of adaptive coping strategies (Carver et al., 2010), as well as with lower perceived cancer risk (Hamilton & Lobel, 2015). Optimism may also influence how people interpret health messages. Han et al. (2012) used a web-based survey to present participants with textual and visual representations (i.e., icon arrays with varying degrees of randomness) of the risk of colorectal cancer. Those who were more optimistic perceived less uncertainty about their colorectal cancer risk overall, while those who were less optimistic experienced heightened uncertainty when risk information was presented in a random format.

A final characteristic that can affect how people react to uncertainty in healthcare is *numeracy*, the ability to understand and use numbers. Numbers are frequently used in conveying probabilistic risks because quantifying descriptors are more useful and less ambiguous than qualitative descriptors (Schwartz, Woloshin, Black, & Welch, 1997). Unfortunately, numerical presentations can be challenging for those with low numeracy to understand, limiting their risk comprehension and healthcare utilization. In a study evaluating women's abilities to assess their breast cancer risk, Schwartz et al. (1997) found that women with low numeracy inaccurately assessed their risk for breast cancer.

Managing Uncertainty

Uncertainty arising from limited access to available health information or the complex nature of a health issue may be reduced by the provision of additional, clarifying information. However, health-related uncertainty often exists because knowledge about the causes, course, and consequences of illness is evolving. When definitive evidence and scientific consensus regarding a disease or treatment are lacking, health-related uncertainty can be unavoidable and irreducible (Han et al., 2011). Such instances may necessitate the adoption of strategies for managing health-related uncertainty, as opposed to minimizing or eliminating uncertainty.

Coping With Uncertainty

As described in the transactional model of stress and coping (Lazarus & Folkman, 1984), in response to a stressor (e.g., health-related uncertainty), people perform a primary appraisal to determine the nature of the stressor and its implications for their well-being, followed by a secondary appraisal to determine which resources they have available for coping with the stressor. These primary and secondary appraisals represent processes that could be targeted to promote adaptive outcomes in the face of health-related uncertainty. Primary appraisals are critical (Babrow et al., 1998): uncertainty can be viewed as a distressing problem or, alternatively, as an opportunity for hope. Empirical evidence suggests that positive primary appraisals

of uncertainty contribute to psychological adjustment. For instance, whereas some cancer patients with late-stage disease participating in clinical trials viewed uncertainty about their prognosis as a source of anxiety, others interpreted this uncertainty as reasons for hope and optimism about their future and the likelihood of receiving benefits from the clinical trial (Brown & deGraaf, 2013). Similarly, among men with prostate cancer undergoing watchful waiting (a disease management option in which an identified asymptomatic prostate cancer is monitored for progression instead of adopting immediate treatment), the appraisal of cancer-related uncertainty as a danger was associated with feelings of worry, negative mood, and decisional intolerance, whereas the appraisal of uncertainty as an opportunity contributed to perceptions of increased options and hope (Bailey, Wallace, & Mishel, 2007).

Achieving a positive primary appraisal of health-related uncertainty is not always possible. Uncertainty can also be managed through the secondary appraisal process and use of coping strategies, which are cognitive or behavioral efforts to manage demands that are perceived as stressful or as exceeding one's resources (Lazarus & Folkman, 1984). Coping strategies can be categorized as emotion focused (aimed at controlling negative emotions through efforts such as distraction or seeking comfort from others) versus problem focused (aimed at resolving or addressing a threat, such as through planning or preparation; Lazarus & Folkman, 1984). Alternatively, coping strategies can be categorized as approach oriented (approaching a stressor such as through problem solving or expressing emotions) versus avoidance oriented (avoiding a stressor such as through denial or withdrawal; Suls & Fletcher, 1985).

Which coping strategies one uses and the psychological outcomes achieved depend on numerous factors including aspects of the stressor, situation, and individual (e.g., Lazarus & Folkman, 1984; Suls & Fletcher, 1985). For instance, an individual's primary appraisal of uncertainty can shape the coping process. Mishel and Sorenson (1991) demonstrated that gynecological cancer patients' appraisal of health-related uncertainty as a danger was associated with greater use of emotion-focused coping, whereas appraisal of uncertainty as an opportunity was associated with the use of proactive problem-focused coping. There is no consensus regarding which coping strategies are most effective or adaptive in response to health-related stressors, including uncertainty. For example, although avoidance-oriented coping is commonly associated with maladaptive outcomes, empirical evidence suggests that the effectiveness of avoidance-oriented coping may depend in part on the temporal nature of health-related uncertainty. Avoidance-oriented coping can mitigate distress when the experience of uncertainty is time limited and short, such as when women are undergoing follow-up testing from an abnormal mammogram (Heckman et al., 2004). Conversely, avoidance-oriented coping can increase distress when uncertainty is unrelenting, including among cancer survivors facing the constant possibility of recurrence (Stanton, Danoff-Burg, & Huggins, 2002). Research is needed to understand how different characteristics of health-related uncertainty, including its source, temporality, and controllability, influence the selection and adaptability of various coping strategies.

Shared Decision Making

Shared and informed medical decision making can also help to manage uncertainty. Shared decision making is a process by which a healthcare provider educates patients about available options and the benefits, harms, and personal impressions of these options and elicits the patient's individual preferences and values in order to achieve a shared understanding of a given medical decision and ultimately make a decision together. Shared decision making is a critical component of patient-centered care and is particularly appropriate for

“preference-sensitive” decisions in which an objectively best option is lacking (Politi, Lewis, & Frosch, 2013). For example, the US Preventive Services Task Force, an independent panel of nongovernment experts in prevention and evidence-based medicine, recommends shared and informed decision making between healthcare providers and women ages 40–49 years regarding the use of mammography due to inadequate evidence about the net benefit of this screening test for these women (U.S. Preventive Services Task Force, 2009).

As part of a shared decision-making process, providers should clearly and openly communicate the uncertainties associated with available options, such as uncertainties due to probabilities, ambiguities, or complexities of the situation. By acknowledging these uncertainties and addressing patients’ confusion and emotional reactions, providers can help patients to tolerate health-related uncertainty and make a high-quality medical decision. In spite of the potential benefits of discussing uncertainty with their patients, providers infrequently disclose such information (Politi et al., 2011). For example, fewer than a quarter of American men report being informed that experts disagree about the use of prostate-specific antigen (PSA) testing to screen for prostate cancer or that no one is sure that PSA testing saves lives (Leyva et al., 2016), although professional societies recommend that providers discuss these issues due to inconsistent evidence about the net benefit of PSA testing.

Numerous barriers may prevent providers from initiating discussions of health-related uncertainty, including limited time, lack of training, and concerns about patients’ reactions. Fears about adverse patient reactions may be warranted: discussions of health-related uncertainty can contribute to less decision satisfaction among patients (Politi et al., 2011), although some discomfort or anxiety may be a consequence of patients being truly engaged in the decision-making process (Politi et al., 2013). Strategies are needed for overcoming providers’ barriers to the disclosure of health-related uncertainty, such as communication skills training or insurance reimbursement for shared decision-making discussions, as well as for empowering and supporting patients as they navigate complex medical decisions.

Conclusion

Uncertainty is a pervasive challenge in healthcare. To date, researchers have characterized some of the distinctive ways in which different types of health-related uncertainty influence people’s cognitions, emotions, decisions, and behaviors, as well as how people’s traits and personalities shape these responses. Promising approaches for managing uncertainty exist, including the promotion of adaptive coping strategies and shared and informed medical decision making; however, additional research is needed to determine for whom and in which situations these approaches are most effective and to develop strategies for helping patients and providers adopt these approaches and address the challenges of health-related uncertainty.

Author Biographies

Jada G. Hamilton, PhD, MPH is an assistant attending psychologist in the Department of Psychiatry and Behavioral Sciences at Memorial Sloan Kettering Cancer Center. She is a social and health psychologist whose work examines psychosocial issues in health and cancer prevention and control. Her program of behavioral research aims to translate advances in genetic and genomic medicine into improvements in cancer care and public health.

Margaux C. Genoff, MA, is a doctoral student in clinical psychology at the New School for Social Research. Her research interests are in the psychology of gender and the role of culture in psycho-oncology and health disparities.

Paul K. J. Han, MD, MA, MPH, is director of the Center for Outcomes Research and Evaluation at Maine Medical Center. He is a behavioral and health services researcher and a general internist and palliative medicine physician. His main research interests are in risk communication and shared decision making, and his work focuses on the communication and management of uncertainty in healthcare.

References

- Babrow, A. S., Kasch, C. R., & Ford, L. A. (1998). The many meanings of uncertainty in illness: Toward a systematic accounting. *Health Communication, 10*(1), 1–23. doi:10.1207/s15327027hc1001_1
- Bailey, D. E., Jr., Wallace, M., & Mishel, M. H. (2007). Watching, waiting and uncertainty in prostate cancer. *Journal of Clinical Nursing, 16*(4), 734–741. doi:10.1111/j.1365-2702.2005.01545.x
- Brashers, D. E. (2001). Communication and uncertainty management. *Journal of Communication, 51*(3), 477–497. doi:10.1111/j.1460-2466.2001.tb02892.x
- Bredemeier, K., & Berenbaum, H. (2008). Intolerance of uncertainty and perceived threat. *Behaviour Research and Therapy, 46*(1), 28–38. doi:10.1016/j.brat.2007.09.006
- Brown, P., & deGraaf, S. (2013). Considering a future which may not exist: The construction of time and expectations amidst advanced-stage cancer. *Health, Risk & Society, 15*(6–7), 543–560. doi:10.1080/13698575.2013.830081
- Carver, C. S., Scheier, M. F., & Segerstrom, S. C. (2010). Optimism. *Clinical Psychology Review, 30*(7), 879–889.
- Eiser, J. R., & Cole, N. (2002). Participation in cervical screening as a function of perceived risk, barriers and need for cognitive closure. *Journal of Health Psychology, 7*(1), 99–105. doi:10.1177/1359105302007001657
- Ellsberg, D. (1961). Risk, ambiguity, and the savage axioms. *The Quarterly Journal of Economics, 75*(4), 643–669.
- Hall, D. L., Mishel, M. H., & Germino, B. B. (2014). Living with cancer-related uncertainty: Associations with fatigue, insomnia, and affect in younger breast cancer survivors. *Supportive Care in Cancer, 22*(9), 2489–2495. doi:10.1007/s00520-014-2243-y
- Hamilton, J. G., Han, P. K. J., Hutson, S. P., Rife, S. C., Kobrin, S. C., Moser, R. P., & Alter, B. P. (2012). Responses to ambiguity are not universal: The moderating role of need for cognitive closure. *Annals of Behavioral Medicine, 43*, S167.
- Hamilton, J. G., Hutson, S. P., Moser, R. P., Kobrin, S. C., Frohnmayer, A. E., Alter, B. P., & Han, P. K. (2013). Sources of uncertainty and their association with medical decision making: Exploring mechanisms in Fanconi anemia. *Annals of Behavioral Medicine, 46*(2), 204–216. doi:10.1007/s12160-013-9507-5
- Hamilton, J. G., & Lobel, M. (2015). Psychosocial factors associated with risk perceptions for chronic diseases in younger and middle-aged women. *Women and Health, 55*(8), 921–942. doi:10.1080/03630242.2015.1061094
- Han, P. K. J., Klein, W. M., Killam, B., Lehman, T., Massett, H., & Freedman, A. N. (2012). Representing randomness in the communication of individualized cancer risk estimates: Effects on cancer risk perceptions, worry, and subjective uncertainty about risk. *Patient Education and Counseling, 86*(1), 106–113. doi:10.1016/j.pec.2011.01.033
- Han, P. K. J., Klein, W. M. P., & Arora, N. K. (2011). Varieties of uncertainty in health care: A conceptual taxonomy. *Medical Decision Making, 31*(6), 828–838. doi:10.1177/0272989x10393976
- Heckman, B. D., Fisher, E. B., Monsees, B., Merbaum, M., Ristvedt, S., & Bishop, C. (2004). Coping and anxiety in women recalled for additional diagnostic procedures following an abnormal screening mammogram. *Health Psychology, 23*(1), 42–48. doi:10.1037/0278-6133.23.1.42

- Koerner, N., & Dugas, M. (2008). An investigation of appraisals in individuals vulnerable to excessive worry: The role of intolerance of uncertainty. *Cognitive Therapy and Research*, 32(5), 619–638. doi:10.1007/s10608-007-9125-2
- Kruglanski, A. W., & Webster, D. M. (1996). Motivated closing of the mind: “Seizing” and “freezing”. *Psychological Review*, 103(2), 263–283.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer.
- Leyva, B., Persoskie, A., Ottenbacher, A., Hamilton, J. G., Allen, J. D., Kobrin, S. C., & Taplin, S. H. (2016). Do men receive information required for shared decision making about PSA testing? Results from a national survey. *Journal of Cancer Education*, 31(4), 693–701. doi:10.1007/s13187-015-0870-8
- Longman, T., Turner, R. M., King, M., & McCaffery, K. J. (2012). The effects of communicating uncertainty in quantitative health risk estimates. *Patient Education and Counseling*, 89(2), 252–259. doi:10.1016/j.pec.2012.07.010
- Mishel, M. H. (1997). Uncertainty in acute illness. *Annual Review of Nursing Research*, 15, 57–80.
- Mishel, M. H., & Sorenson, D. S. (1991). Uncertainty in gynecological cancer: A test of the mediating functions of mastery and coping. *Nursing Research*, 40(3), 167–171.
- Politi, M. C., Clark, M. A., Ombao, H., Dizon, D., & Elwyn, G. (2011). Communicating uncertainty can lead to less decision satisfaction: A necessary cost of involving patients in shared decision making? *Health Expectations*, 14(1), 84–91. doi:10.1111/j.1369-7625.2010.00626.x
- Politi, M. C., Lewis, C. L., & Frosch, D. L. (2013). Supporting shared decisions when clinical evidence is low. *Medical Care Research and Review*, 70(Suppl. 1), 113s–128s. doi:10.1177/1077558712458456
- Rosen, N. O., Knauper, B., & Sammut, J. (2007). Do individual differences in tolerance of uncertainty affect health monitoring? *Psychology & Health*, 22(4), 413–430.
- Schwartz, L. M., Woloshin, S., Black, W. C., & Welch, H. G. (1997). The role of numeracy in understanding the benefit of screening mammography. *Annals of Internal Medicine*, 127(11), 966–972.
- Song, L., Northouse, L. L., Braun, T. M., Zhang, L., Cimprich, B., Ronis, D. L., & Mood, D. W. (2011). Assessing longitudinal quality of life in prostate cancer patients and their spouses: A multi-level modeling approach. *Quality of Life Research*, 20(3), 371–381. doi:10.1007/s11136-010-9753-y
- Stanton, A. L., Danoff-Burg, S., & Huggins, M. E. (2002). The first year after breast cancer diagnosis: Hope and coping strategies as predictors of adjustment. *Psycho-Oncology*, 11(2), 93–102.
- Suls, J., & Fletcher, B. (1985). The relative efficacy of avoidant and nonavoidant coping strategies: A meta-analysis. *Health Psychology*, 4(3), 249–288.
- U.S. Preventive Services Task Force. (2009). Screening for breast cancer: U.S. Preventive Services Task Force recommendation statement. *Annals of Internal Medicine*, 151(10), 716–726, w-236. doi:10.7326/0003-4819-151-10-200911170-00008

Suggested Reading

- Han, P. K. J. (2013). Conceptual, methodological, and ethical problems in communicating uncertainty in clinical evidence. *Medical Care Research and Review*, 70(Suppl. 1), 14s–36s. doi:10.1177/1077558712459361
- Han, P. K. J., Klein, W. M. P., & Arora, N. K. (2011). Varieties of uncertainty in health care: A conceptual taxonomy. *Medical Decision Making*, 31(6), 828–838. doi:10.1177/0272989x10393976
- Mishel, M. H. (1999). Uncertainty in chronic illness. *Annual Review of Nursing Research*, 17, 269–294.
- Politi, M. C., Han, P. K., & Col, N. F. (2007). Communicating the uncertainty of harms and benefits of medical interventions. *Medical Decision Making*, 27(5), 681–695. doi:10.1177/0272989x07307270

Commonsense Modeling (CSM) of Health Behaviors

Howard Leventhal¹, L. Alison Phillips², Edith Burns³,
and Jenna Herold Cohen¹

¹*Department of Psychology, Rutgers, The State University of New Jersey,
New Brunswick, NJ, USA*

²*Department of Psychology, Iowa State University, Ames, IA, USA*

³*Medical College of Wisconsin, Milwaukee, WI, USA*

The commonsense model (CSM) was designed to describe the mechanisms underlying individuals' awareness and understanding of their current and future health and threats to health and how they plan to act or currently act and respond to feedback from their actions. The CSM is a dynamic control systems model that provides a conceptual framework for representing an individual's perceptions, expectations, and beliefs and actions about his/her body as he/she navigates daily life (Leventhal, Phillips, & Burns, 2016). It also provides a way for representing the moderating effects of cultural and individual differences. Although varying amounts of attention have been paid to the latter two topics, investigators have amassed a large body of data from a wide array of clinical and experimental settings regarding the CSM in general (Hagger, Koch, Chatzisarantis, & Orbell, 2017), and the CSM continues to suggest hypotheses for investigation.

Underlying Principles and Structure of the CSM

The CSM uses contemporary concepts from cognitive and social science to organize what people at different ages, including those who are ill and those who are well, say and do to manage ongoing and to prevent future health threats. The resulting conceptual framework proposes that all behaviors for controlling current or future health threats are generated and guided by mental models (i.e., *representations* of the threat, the *treatments or procedures for threat control*, and *action plans* for performance and evaluating outcomes). Representations

are generated when stimuli such as symptoms, changes in function, and the observation of illness in other individuals, and media messages activate underlying memory structures or illness *prototypes* acquired during an individual's prior health history. The factors defining the representation are generated by the particular prototypes that have been activated.

A second principle proposes that *prototypes*, *representations*, and *action plans* are multilevel, that is, they consist of concrete, perceptual experiences combined with abstract concepts. Perceptual experiences give meaning to abstract concepts and concepts give meaning in return (Brewer, Hallman, & Kipen, 2008). Understanding how representations and actions change over time requires modeling the interactions, both conscious and implicit, among concrete experiences and prototypes. A term such as "belief(s)" has a different meaning therefore, in the multilevel structure of the CSM than in the "health belief model" (Rosenstock, Strecher, & Becker, 1988) or the "theory of planned behavior" (Ajzen, 1991); these latter decision models focused on probabilities and utilities ("expectancy-value models") without specifying their relationships to experience. These frameworks can predict that an individual will seek and use medical care if she/he "believes" him or herself ill, without modeling the processes involved in how that individual *perceived* and *decided* that he or she was ill and how he or she decided that medical care was the appropriate action for self-management. The CSM provides conceptual starting points for describing interactions among the mechanisms underlying perceptions of health and illness. This deeper understanding is essential to move from prediction (which may involve widely different variables for different illnesses and populations) to developing effective interventions.

A third underlying principle is that all components of the CSM play a crucial role when predicting a specific outcome or planning an intervention to improve behavior and health outcomes. Thus, even when a particular variable predicts an outcome (e.g., painful symptoms are more likely than non-painful symptoms to lead to seeking medical care), factors that do not differ across pain levels and are not critical for making the prediction (e.g., having access to a medical practitioner) are critical for the occurrence of the behavior.

Structure and Operation of Commonsense Model

The processes involved in creating commonsense models of health and health threats are similar to that in other multifactorial control systems models (e.g., Carver & Scheier, 1982). All of these models hypothesize that health-related actions are generated by multiple interacting variables and ongoing feedback from action. The process is frequently initiated when a stimulus, for example, a headache, matches an *illness prototype* or memory structure of prior headaches, thereby activating an *illness representation* or mental model of the experience and its meaning. The process is also shaped by one of the prototypes at a "deeper, implicit level," the *acute prototype* that underlies and defines the properties of an array of specific conditions. Acute conditions are perceived as symptomatic, short lasting, with known causes, non-life threatening, and controllable. Representations for current ongoing conditions are influenced by the acute prototype; the expectations for its duration, causes, treatments, and action plans for implementing treatment are influenced by the underlying acute prototype. The differentiation between representations and prototypes is critical in commonsense modeling. Prototypes are the history of an individual's experiences of her or his physical and functional self and the history of specific conditions, the symptoms, and physical and cognitive functions associated with the illness. Prototypes form the "baseline comparisons" for deciding what is "normal health" and everyday illness. The mental models or representation of a condition that is active in mind at a given moment may contain only some of the features of an underlying prototype

and can share features of more than one prototype. As active working models, representations are more flexible than prototypes; representations motivate ongoing action and respond to feedback from action. The expectations built into prototypes and expressed in representations are critical for appraising the value of responses whether self-selected or professionally prescribed.

Both automatic and deliberative, that is, reasoned processes, are involved in the activation of representations. The proportion of one or the other is a function of prior experience with the initiating stimulus. Representations of common conditions will automatically generate *action plans* for controlling a disorder, whereas representations generated by novel and/or highly threatening symptoms will activate substantial amounts of deliberative, conscious processing. The balance between automatic and deliberative responding will depend upon familiarity with the threat and contextual factors ranging from contact with ill individuals to exposure to novel environments and consumption of unusual foods. Feedback from action provides evidence for the validity of the both illness and treatment representations. Uncertain feedback can stimulate questions such as “Did my distress continue because the treatment failed to reduce stomach acid or because the discomfort was not from my stomach?” (Bunde & Martin, 2006). How feedback updates the parameters of illness and treatment representations and action plans is a critical area for theoretical development and empirical research.

Prototypes and Representations

Language can only partially describe the processes involved in the activation of representations; computer models will prove essential for representing this rapidly moving complex process (Lowe & Norman, 2017). The complexity arises as cues, for example, symptoms, can activate multiple disease-specific prototypes and more than one implicit (nonconscious) deep-level prototype, such as *acute*, *episodic* (periodic flares), and *chronic* (part of me will never go away). The prototype activated depends in part upon the eliciting information, for example, previously experienced symptoms, illness in a family member, information from a doctor, and mass media. Interactions among the prototypes of the self, diseases and treatments, generate the mental models or representations that stimulate action plans, actions, and feedback.

The self-system contains multiple prototypes of an individual’s physical, cognitive, and emotional functions, systemic vulnerabilities, and prior illnesses in addition to their social roles, traits, and emotional states that are the typical focus of research in personality and social psychology. Prototypes are both conceptual (what I am, what I do) and experiential (i.e., somatic feel of self and its ongoing functions). Both the abstract and experiential levels generate expectations as to what I am when well and what I am when ill. They also link specific symptoms and functional changes to one or another illness. Prototypes are anchors against which changes in somatic sensations (symptoms) and function are experienced and evaluated as relevant or irrelevant to one’s physical integrity and functional soundness.

Many of the prototypes that influence the mental representations that regulate ongoing health behaviors reside at a “deep” level. Evidence for the presence of deep-level prototypes can be found in many forms. One source of particular relevance to health psychologists is data validating the accuracy of individuals’ overall sense of physical and functional health: “self-assessment of health” (SAH). SAH, rated on a five-point scale (poor, fair, good, very good, or excellent), is a consistent, valid predictor of mortality in multiple community samples (Idler & Benyamini, 1997) and has outperformed “objective” medical indicators in predicting mortality in clinical samples. In summary, a diverse, multilevel array of prototypes establish expectations regarding health and function in multiple areas, setting the stage for action and the interpretation of

outcomes of self-generated behaviors and prescribed treatments. Deep-level prototypes of such self-assessed health likely influence whether symptoms and functional changes activate prototypes and representations of serious or mild chronic and acute conditions; the vigorous, “healthy” self may be more likely to attribute symptoms to minor than serious life-threatening events.

Illness Prototypes and Representations: A Multilevel Process

Illness prototypes are the repositories of experiences with prior illness in oneself, direct observations, and communications from family, friends, practitioners, Internet searches, and media. Some of these memory structures are universal; it is a rare individual who has not experienced an upset stomach or a cold one or more times. An illness representation is formed when an event, for example, a symptom or observation of someone who is ill, activates one or more prototypes. The parameters of representations, the expectations they generate, reside in one or more of five areas: *identity* (e.g., label and somatic experiences), *cause*, *control* (personal, doctor), *timelines* (time for onset, duration, and removal with treatment), and *consequences* (both immediate and long term). These factors affect the selection of actions and expectations for outcomes. Representations that attribute the causes and/or select the treatments of a somatic state that are inconsistent with the conditions underlying biology can generate responses that fail to ameliorate target symptoms and delay appropriate treatment.

Disease-specific prototypes are connected to or “nested in” deep-level prototypes, structures that affect the parameters of disease-specific prototypes and the mental representations that are active in the selection of treatments and appraisal of treatment outcomes. The “acute” prototype is the deep-level “default”; it defines the attributes and sets the expectations for virtually all everyday conditions including the selection of treatments and expected outcomes. For example, exposure to coughs and sneezes of someone with a bad head cold (cause) might lead one to expect a similar array of symptoms (identity) within 24 hours that will last 2–5 days (timeline), disrupt daily activities (consequence), and use various over-the-counter medications (control). The structure matches the experience of a vast array of acute conditions. Frequency of activation strengthens the acute prototype, making it the preferred model for representing particular illnesses. As the default, the acute prototype can define parameters and set expectations for conditions that are episodic and chronic, as well as those acute. Computer models suggests that the “acute prototype” may underlie the role of perceived control in connecting illness and treatment representations (Lowe & Norman, 2017).

The chronic prototype forms a distinct contrast with the acute prototype. Timelines are likely the major source of difference: chronic conditions are lifelong, though many vary over the lifespan, and the consequences are often life threatening. The two can overlap however, in the causal area, for example, life experiences and lifestyle behaviors are commonly cited as causal, though genetic causes are more commonly attributed for chronic conditions. The “acute and chronic” prototypes differ to a substantial degree for preferred treatments for control: over-the-counter medications and minor life changes are most often tried for acute conditions, while professionally prescribed treatment and/or major lifestyle changes are applied for conditions defined as chronic. Patients frequently attribute symptoms to chronic conditions that are asymptomatic, for example, hypertension, and misattribute symptoms to diseases that are highly threatening but silent during much of their developmental histories, for example, cancer.

The overlap between the acute and chronic prototype gives rise to a third deep structure, the “episodic prototype.” Episodic conditions generally last a lifetime; they are chronic but have symptomatic flares that match acute conditions; asthma is a prime example.

Self-management of such conditions (chronic, with acute episodes of symptoms) can be undermined when patients focus on symptom management and fail to treat the underlying relatively asymptomatic chronic component. For example, many patients with asthma who agree that “I’ll have asthma all my life” also agree they “only have it when having symptoms” (Halm, Mora, & Leventhal, 2006); these patients fail to use inhalers to control their underlying chronic inflammation. Problems also arise when the target symptoms are unrelated or poorly correlated with the underlying condition. Early-stage problems may arise when experimenting with alternative procedures (e.g., special diets, medication) delays care seeking. Later-stage problems may arise when successful symptom resolution leads to nonadherence to long-term treatment, allowing the underlying condition to worsen.

Prototypes and representations of treatments are similar in structure and content to those for illnesses. Treatment procedures are chosen on the basis of the match between their properties and those of the illness. The selection can be automatic, that is, reaching for antiseptic for an abrasion to the skin or deliberative. Deliberative decisions will be likely when an initial automatic selection fails (e.g., antiseptic did not prevent infection, so now call doctor’s office) or when presenting symptoms are uncertain and/or of threatening magnitude.

Action plans are brought into play by the activation of illness and treatment representations. The concept was introduced in experimental studies examining the impact of fear-arousing messages on preventive health actions such as taking tetanus inoculations (Leventhal, 1970). These studies were the basis for the parallel framework in which one pathway involved cognitions of the threat of tetanus that led to changes in attitudes and intention immediately after the message and led to action, that is, getting an inoculation to prevent tetanus in the days or weeks following message exposure, when the threat information was combined with an action plan. A separate pathway involving the activation of fear emotion during exposure also influenced attitudes and intentions immediately post exposure to the threat message, but had essentially no effect on being inoculated, in the absence of an action plan. Reviews of studies over recent decades are consistent with the assumptions underlying the original parallel processing model and its revisions (Tannenbaum et al., 2015).

Action plans are often overlearned, implicit, and automatic, for example, a headache that disrupts sleep can move the restless sleeper to the medicine cabinet, removing and opening a bottle of aspirin or acetaminophen, removing two or three tablets, putting them into the mouth, and downing them with a glass of water. This well-structured and rehearsed action plan reflects a history of prior headache management. The expectations generated by illness and treatment representations led to performance, most of which was automatic, requiring few deliberative decisions. Forming a new or novel action plan requires considerably more mental and behavioral steps; the cognitive processes involved in planning will differ from those involved in the execution of well-learned, preexistent plans. The planner has to visualize the action, its details, the environment in which it takes place, and how best to insert the action into the environment. The third and perhaps most important set of factors is perceiving behavioral sequences—what he or she does at particular times and places that are compatible or incompatible with the proposed action. This latter step is critical for the management of chronic conditions such as diabetes, chronic reflux, and hypertension, illnesses that may require multiple medications taken more than once a day and benefit from substantial changes in lifestyle, for example, diet and physical activity. How does one integrate the taking of multiple medications into daily life, change food preferences, times and quantities eaten, locate places to walk? Specifically, how does one plan and initiate behaviors that become habitual (Phillips, Leventhal, & Leventhal, 2013)? Qualitative studies, focus groups, and intensive open-ended interviews indicate that producing effective action plans involves strategies for

attending to the environmental details and behavioral patterns to identify habitual action sequences consistent with initiating and sustaining treatment (Brooks et al., 2015).

Using and Misusing the Commonsense Model

As stated in the opening, the CSM provides a conceptual framework for representing what people do and say as to how they manage ongoing and prospective illness threats. The CSM is designed to describe how things happen; it is a process-oriented conceptual framework designed to suggest new hypotheses and techniques for measurement and intervention, and it is open to modification when demanded by systematic observation and experiment. As is the case with all process models, there are problems in how it is used; we discuss the most important.

Confusing Method and Theory

A number of standardized instruments have been devised to measure the components of the CSM. Although useful, measures are not equivalent to concepts; the Illness Perception Questionnaire (IPQ and IPQ-R; Moss-Morris et al., 2002) is conflated with the concept of “illness representations” in a meta-analysis by Brandes and Mullan (2014). These measures are but one of many possible ways of assessing illness representations; they are not identical to the concepts and are not dynamic and do not describe process (Phillips, Leventhal, & Burns, 2017). The conflation of validated instruments with concepts, for example, the IPQ and IPQ-R, may lead reviewers to exclude studies using disease-specific measures to assess illness and/or treatment representations, an omission that reflects failure to understand the difference between measures and concepts. This also reflects an uncritical acceptance of a one-size-fits-all approach for hypothesis testing as spelled out in a response to the claims made in recent meta-analysis of IPQ and related instruments (Phillips et al., 2017).

In addition, one might ask why investigators conducted studies and meta-analyses assessing the relationship of measures of illness representations to long-term adherence for chronic conditions rather than assessing treatment representations. A possible answer is that the availability of validated measures, IPQ and IPQ-R, stimulated research, reminding one of the saying, “If you have a hammer, everything looks like a nail.” The CSM hypothesizes, however, that illness representations moderate, and treatment representations mediate, adherence for both chronic and acute conditions (see Horne et al., 2013). Illness representations are not irrelevant in the adherence process; they set the targets, that is, the outcome expectations for evaluating treatment efficacy. How effectively and quickly the treatment impacts symptoms or “objective” measures determines whether it can control the disturbance and whether it addresses its causes (Rottmann, 2016). The variables in the CSM do not operate independently. Useful though it may be, multiple regression modeling does not address these issues especially when used blindly.

When to Assess Specific Components of the CSM

The confusion of concepts and measures reflects a deeper misunderstanding of control systems models; no single variable operates without the contribution of the others. By contrast, regression modeling often leads investigators to conclude incorrectly that a predictor is responsible by itself for variance in an outcome. Illness representations are important for motivating action at

symptom onset (typical for acute conditions), setting criteria for evaluating treatment efficacy, and understanding complex conditions that combine acute and chronic features. Continuing treatment after the initiation of treatment will involve satisfying expectations of both the illness and treatment, that is, what symptoms will it control, functions will improve, and time to and duration of efficacy. When implemented by a simple and effective action plan, the factors important during acquisition may fade in importance and be forgotten once a behavior is habitual (Gardner, 2015). The following examples illustrate how different illnesses and clinical settings allow investigators to understand how the CSM operates in diverse contexts.

Regarding acute conditions and care seeking in primary care, Cameron, Leventhal, and Leventhal (1995) found that new symptoms are critical for care seeking and that care seekers' symptom representations are more likely to have an identity, long timeline, and severe consequences and are frequently targets for efforts to control.

Regarding care seeking in an emergency care setting, common sense represents recurrent heart attacks as acute, with one event unrelated to the next. Onset of symptoms (e.g., chest and/or shoulder pain, profuse sweating) as sudden and rapid and symptoms are recognized by laypersons and practitioners alike. However, significant numbers of individuals with MI present with "atypical" symptoms (Grosmaître et al., 2013). For example, pain can be experienced in the upper abdomen rather than the chest and interpreted as gastric rather than heart. Bunde and Martin (2006) described how the perception and interpretation of atypical symptoms related to delays in seeking hospital care by individuals having an MI; those quick to seek care had "textbook symptoms" (e.g., chest or shoulder pain, profuse sweating); and a prior history of cardiac problems. Those who were slower to seek care reported gastric pain, chronic fatigue, breathlessness, and swollen feet. For the physician, breathlessness, chronic fatigue, and swollen feet are signs of heart failure, while for the layperson, they are misperceived as being unrelated to a serious heart condition and instead taken as commonsense indicators of aging; after all, "one's heart is not in one's feet." Patients articulate these misperceptions: "When you hear about having heart problems ... you're supposed to feel maybe a pain in your left arm, maybe a pain in your chest, or pressure... It would have been clearer to me if I had chest pain and then I would have said, okay, I'll call and say I'm having chest pain..." (Horowitz, Rein, & Leventhal, 2004). Symptoms related to the problems of everyday life, for example, stomachache and fatigue, are unlikely to stimulate care seeking, and even dramatic events unrelated to the prototype can be "ignored"; for example, as one heart failure patient said, "I guess that I could have gone to the doctor after I had that collapse on the hallway floor. It might have been a good idea" (Horowitz et al., 2004).

Regarding adherence for chronic conditions without and with acute, episodic features, the influence of deep-level acute and chronic models on the representation of illness and self-management is evident among both hypertensive and asthmatic patients. Hypertension lies at one extreme on the dimension of symptom experience, as hypertension is asymptomatic for the vast majority of patients. Though patients are told, and most agree that, "People can't tell whether their blood pressure is up" (80% of hypertensive sample), the same respondents believe they "can tell when my own blood pressure is up" (92% of same sample) from symptoms, for example, headaches, dizziness, and warm faces. A majority (70%) also believed treatment moderated their symptoms (Meyer, Leventhal, & Guttman, 1985). The coherence of the representations of hypertension and medication treatment, consistent with an underlying acute model seemingly "resting" on a chronic model, was likely responsible for high levels of adherence and good blood pressure control. Newly diagnosed individuals with hypertension (73% of 65) and non-hypertensive patients (48% of 50) in the same clinic believed they could tell when their blood pressure was elevated.

Asthma, a chronic (often asymptomatic) inflammatory process punctuated by episodic and highly symptomatic episodes that can be life threatening, was represented as both chronic and acute. Although 92% of a sample of 198 asthmatic patients believed asthma was chronic, that is, they would definitely, probably, or possibly always have it, more than half (53%) of the sample also believed they only had asthma when symptomatic; asthma was not there when they were asymptomatic (Halm et al., 2006). These patients controlled flares, but were unlikely to use peak flow meters to monitor their status, and make and keep routine visits for asthma (see also Kaptein et al., 2008). Using medication when symptomatic while claiming asthma a life-long condition represents a joining of acute and chronic models in an episodic model at the deep level.

Automating Action for Long-Term Self-Management

It is clear that many patients adhere to treatment for lifelong periods, though it is often less than half of patients sampled who manage to do so consistently. Two aspects of long-term adherence have been examined: (a) how did highly adherent patients achieve *and* sustain lifetime self-management and (b) how can practitioners enhance effective management among patients who are nonadherent?

Regarding patient strategies for forming routines, Phillips et al. (2013) found that patients who developed habitual routines for consistent use of antihypertensive medication linked medication use to specific environmental cues, for example, to specific times and places. Factors typically related to the initiation of treatment, for example, treatment-related beliefs, the presence or absence of barriers to adherence, and feedback confirming treatment efficacy, were unrelated to habitual performance. In sum, once perfected, high levels of adherence are maintained independent of the conditions essential for treatment initiation. The importance of routines for long term was again made clear in a recent study of 306 low-income patients with asthma; 68% were African American and/or Hispanic and over 60 years of age (Brooks et al., 2015). Only 38.6% reported high levels of adherence to daily medication. Highly adherent patients integrated daily use of their inhalers with existent habit patterns. An example of such a routine was “.... putting the inhaler in the bathroom and using it when I get up in the morning.” This group of “super-adherers” was 3.7 times more adherent than patients who did not integrate medication with daily routines. Although initiating and converting adherence into systematic habit is more common among educated, financially well-off patients, any patient who does so is highly adherent; strategies work for those who use them! Bolman, Arwert, and Völlink (2011) reported similar findings, observing that habit strength for taking prophylactic asthma medication predicted patient adherence, along with medication beliefs; they too contributed to adherence.

Highly adherent patients with diabetes, “experts” in controlling A1c, reported similar strategies (Tannenbaum et al., 2015). They stated that although it is necessary to recognize the long-term health threats associated with diabetes, one needed to put these thoughts aside and focus on action: “You just cannot be a bystander in this disease.” Their strategies included monitoring daily behavior to identify times and places for incorporating medication use. They searched for “start points,” for example, “You change your food in the super market, not when you sit down to eat,” and urged a gradual approach to change, “take one day at a time,” that gave their bodies time to adjust to new routines such as exercise. They emphasized the need to experiment and monitor blood glucose levels to detect safe and risky foods: “tested a lot at first—to test out my food” and recommended seeking assistance when confused, for example,

“called nurse to discuss reading.” They were clear on the importance of establishing routines or habits: “testing became second nature, like tying my shoes”; “in the morning, wash face, brush teeth, test blood.” By monitoring, testing, and continually updating behavior, they created a coherent commonsense system that operated automatically.

Regarding communication in the clinical setting to facilitate routines for adherence, Phillips, Leventhal, and Leventhal (2012) asked patients to complete a brief questionnaire the day following a clinic visit. The questions assessed whether the physician discussed commonsense factors and action plans to manage the condition (“The doctor told me how to monitor my problem to see if the treatment is working”) and whether the physician understood their feelings about their problem (“My doctor understood my feelings about this problem”). When interviewed 1 month later about treatment adherence, problem resolution, and subsequent visits to the emergency room (ER), patients who endorsed the commonsense items a month earlier were more adherent, had better problem resolution, and were less likely to go to the ER. In contrast, patients who had praised their practitioners’ psychosocial skills a month earlier reported both poorer adherence and problem resolution, though they were still highly satisfied with the visit. In short, satisfaction was unrelated to adherence and negatively related to problem resolution. Further, all patients who had visited the ER post clinic visit had higher scores on the psychosocial items. This study illustrates the importance of spelling out the details of when and how to perform self-treatment and what to expect during after doing it; doing so ensures effective communicators. How one defines a problem sets the stage for action and evaluation of treatment efficacy (Omer, Hwang, Esserman, Howe, & Ozanne, 2013). Though telling patients when, where, and how to respond—giving them an action plan—is effective in providing strategies for action, it is not the same as assisting them with acquiring the skills for planning on their own or for building automatic management routines.

Concluding Comments and Future Directions

The CSM embeds patients’ experiences, their symptoms, temporal expectations, concrete causes, and outcomes, in a conceptual framework of contemporary cognitive science. The CSM differs from models that focus on beliefs and/or utilities abstractions imposed on patients by the investigator that do not capture the concrete details of everyday life and performance. These details are essential for developing effective interventions. Although studies are few in number, positive results have been reported for interventions to modify postpartum depression (Howell et al., 2012) and increase adherence to antihypertensive medications. Research is needed to determine whether standardized interventions can assist patients with forming habitual routines for daily self-management, critical for control of multiple common chronic conditions, and whether similar interventions can be effective for lifestyle behaviors. The CSM also reshapes our views of constructs central in behavioral research; habit is an example. Habits are generally addressed as overt, that is, visible responses, and they often are. Habits reside, however, throughout the CSM; there are habits for attending and monitoring the body and its functions, habits for responding to somatic cues, and habits for seeking advice. Habitual or automatic processes function in conjunction with more conscious, deliberative processing throughout the operation of commonsense modeling, and habits can be “objects” in attention. Communications that enhance patient attention to and adoption of responses in specific settings and times of day will be critical for habit formation for long-term adherence.

It is also necessary to enhance awareness of the deeper structures underlying illness and treatment prototypes and habitual behavior. Patients need to be aware of the influence on their expectations and behaviors of implicit, deeper-level prototypes such as the acute model. Deciding and/or forgetting to continue treatments after months and years of adherence for a serious chronic condition because “I haven’t had any symptoms for 2 years” is consistent with an acute view of the illness, but dangerous for control and longer-term health. Data on heart attacks are illustrative of the problem, showing that medication adherence declines precipitously a year or two post attack; in the absence of symptoms, it may seem that the threat is over and that one can and indeed should stop treatment. The chronic template—that treatment is part of oneself for life—needs to be built into the system, with particular attention to conditions that are chronic but have episodic flares (e.g., asthma, diabetes, congestive heart failure); treating flares alone is insufficient for long-term control. It is also unknown if or how underlying prototypes of the self-change over time. If one expects treatment to result in a return to “normal,” and the “normal” prototype in the implicit mind of the 65- or 75-year-old individual is what they were at 50 years of age, it is not surprising they may feel that a treatment is ineffective even though it is doing its job biologically. Additional details on the role of social context and influence of affective processes on the cognitive behavioral pathway for self-management are being spelled out in detail not possible in an encyclopedia entry. The CSM is a work in progress; its “Bayesian” properties and their implications for practitioner patient communications to enhance adherence are the next major steps in the advancement of its theoretical framework and empirical testing.

Author Biographies

Howard Leventhal, PhD, Member, National Academy Medicine-NAS; Distinguished Professor of psychology, Rutgers University; Adjunct Professor, Mt. Sinai and Northwell University. He has graduated 65+ PhD students. His research has spanned topics related to fear and action plans, preparation for medical procedures, smoking cessation, affective and cognitive co-effects on health behavior; postpartum symptom reduction, symptom perception and interpretation, and patient self-management of illness. He is the primary developer of the commonsense model of self-regulation.

L. Alison Phillips, PhD, Assistant Professor of Psychology, Iowa State University. Dr. Phillips’ research focuses on the psychosocial and contextual factors, including patient-provider communication, required for health habit development for chronic illness self-management (medication adherence, exercise). Her work utilizes mobile technology (electronic monitoring pill bottles and accelerometers/Fitbits) to objectively measure behavior in healthy adults and in patients with hypertension, type 2 diabetes, and stroke, as well as patients in primary care.

Edith Burns, MD, Professor of Medicine, Professor Population Health-Epidemiology, Medical College of Wisconsin, Clinical Professor of Medicine Marquette University, Fellow Gerontological Society of America. Mentor to >20 T35 and MSTAR medical student research trainees and >20 geriatric medicine fellows. Her research topics are immunology of aging, successful aging, self-management of chronic illness (e.g., diabetes, CHF, joint impairment), patient expectations and timelines for outcomes, risks for hospital readmission after elective surgery, and multimorbidity in geriatric populations.

Jenna Herold Cohen, MS, 5th year, Doctoral Candidate in Clinical Psychology at Rutgers University. Her research interests include factors that aid in adaptive cancer survivorship. Her dissertation will improve knowledge of factors affecting fear of recurrence among patients who have completed treatment for lymphoma. This work is being conducted under the supervision of Howard Leventhal, PhD, with committee members from Memorial Sloan Kettering Cancer Center (Smita Banerjee, PhD) and clinical faculty at Rutgers University.

References

- Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, 50(2), 179–211.
- Bolman, C., Arwert, T. G., & Völlink, T. (2011). Adherence to prophylactic asthma medication: Habit strength and cognitions. *Heart & Lung: The Journal of Acute and Critical Care*, 40(1), 63–75.
- Brandes, K., & Mullan, B. (2014). Can the common-sense model predict adherence in chronically ill patients? A meta-analysis. *Health Psychology Review*, 8, 129–153.
- Brewer, N. T., Hallman, W. K., & Kipen, H. M. (2008). The symmetry rule: A seven-year study of symptoms and explanatory labels among Gulf War veterans. *Risk Analysis*, 28(6), 1737–1748. doi:10.1111/j.1539-6924.2008.01118.x
- Brooks, T. L., Leventhal, H., Wolf, M. S., O'Connor, R., Morillo, J., Martynenko, M., ... Federman, A. D. (2015). Strategies used by older adults with asthma for adherence to inhaled corticosteroids. *Journal of General Internal Medicine*, 29(11), 1506–1512.
- Bunde, J., & Martin, R. (2006). Depression and prehospital delay in the context of myocardial infarction. *Psychosomatic Medicine*, 68(1), 51–57.
- Cameron, L., Leventhal, E. A., & Leventhal, H. (1995). Seeking medical care in response to symptoms and life stress. *Psychosomatic Medicine*, 57(1), 37–47.
- Carver, C. S., & Scheier, M. F. (1982). Control theory: A useful conceptual framework for personality—social, clinical, and health psychology. *Psychological Bulletin*, 92(1), 111–135.
- Gardner, B. (2015). A review and analysis of the use of 'habit' in understanding, predicting and influencing health-related behavior. *Health Psychology Review*. doi:10.1080/17437199.2013.876238
- Grosmaître, P., Le Vasseur, O., Yachouh, E., Courtial, Y., Jacob, X., Meyran, S., & Lantelme, P. (2013). Significance of atypical symptoms for the diagnosis and management of myocardial infarction in elderly patients admitted to emergency departments. *Archives of Cardiovascular Diseases*, 106(11), 586–592.
- Hagger, M. S., Koch, S., Chatzisarantis, N. L. D., & Orbell, S. (2017). The common sense model of self-regulation: Meta-analysis and test of a process model. *Psychological Bulletin*. doi:10.1037/bul0000118
- Halm, E. A., Mora, P., & Leventhal, H. (2006). No symptoms, no asthma: The acute episodic disease belief is associated with poor self-management among inner-city adults with persistent asthma. *Chest*, 129(3), 573–580.
- Horne, R., Chapman, S. C. E., Parham, R., Freemantle, N., Forbes, A., & Cooper, V. (2013). Understanding patients' adherence-related beliefs about medicines prescribed for long-term conditions: A meta-analytic review of the Necessity-Concerns Framework. *PLoS One*. doi:10.1371/journal.pone.0080633
- Horowitz, C. R., Rein, S. B., & Leventhal, H. (2004). A story of maladies, misconceptions and mishaps: Effective management of heart failure. *Social Science & Medicine*, 58(3), 631–643.
- Howell, E. A., Balbierz, A., Wang, J., Parides, M., Zlotnick, C., & Leventhal, H. (2012). Reducing postpartum depressive symptoms among black and Latina mothers: a randomized controlled trial. *Obstetrics and gynecology*, 119(5), 942–949.

- Idler, E. L., & Benyamini, Y. (1997). Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior*, 38, 21–37.
- Kaptein, A. A., Hughes, B. M., Scharloo, M., Fischer, M. J., Snoei, L., Weinman, J., & Rabe, K. F. (2008). Illness perceptions about asthma are determinants of outcome. *Journal of Asthma*, 45(6), 459–464.
- Leventhal, H. (1970). Findings and theory in the study of fear communications. *Advances in Experimental Social Psychology*, 5, 119–186.
- Leventhal, H., Phillips, L. A., & Burns, E. A. (2016). The common-sense model of self-regulation (CSM): A dynamic framework for understanding illness self-management. *Journal of Behavioral Medicine*, 39, 935–946.
- Lowe, R., & Norman, P. (2017). Information processing in illness representation: Implications from an associative-learning framework. *Health Psychology*, 36(3), 280–290.
- Meyer, D., Leventhal, H., & Guttman, M. (1985). Common-sense models of illness: The example of hypertension. *Health Psychology*, 4(2), 115.
- Moss-Morris, R., Weinman, J., Petrie, K., Horne, R., Cameron, L., & Buick, D. (2002). The revised illness perception questionnaire (IPQ-R). *Psychology & Health*, 17(1), 1–16.
- Omer, Z. B., Hwang, E. S., Esserman, L. J., Howe, R., & Ozanne, E. M. (2013). Impact of ductal carcinoma in situ terminology on patient treatment preferences. *JAMA Internal Medicine*, 173(19), 1830–1831.
- Phillips, L. A., Leventhal, H., & Burns, E. (2017). Choose (and use) your tools wisely: “Validated” measures and advanced analyses can provide invalid evidence for/against a theory. *Journal of Behavioral Medicine*. doi:10.1007/s10865-016-9807-x
- Phillips, L. A., Leventhal, H., & Leventhal, E. A. (2012). Physicians’ communication of the common-sense self-regulation model results in greater reported adherence than physicians’ use of interpersonal skills. *British Journal of Health Psychology*, 17(2), 244–257.
- Phillips, L. A., Leventhal, H., & Leventhal, E. A. (2013). Assessing theoretical predictors of long-term medication adherence: Patients’ treatment-related beliefs, experiential feedback and habit development. *Psychology & Health*, 28(10), 1135–1151.
- Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1988). Social learning theory and the health belief model. *Health Education Quarterly*, 15(2), 175–183.
- Rottmann, B. (2016). Searching for the best cause: Roles of mechanism beliefs, autocorrelation, and exploitation. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 42(8), 1233–1256. doi:10.1037/xlm0000244
- Tannenbaum, M. B., Hepler, J., Zimmerman, R. S., Saul, L., Jacobs, S., Wilson, K., & Albarracín, D. (2015). Appealing to fear: A meta-analysis of fear appeal effectiveness and theories. *Psychological Bulletin*, 141(6), 1178–1204.
- Tannenbaum, M. L., Leventhal, H., Breland, J. Y., Yu, J., Walker, E. A., & Gonzalez, J. S. (2015). Successful self-management among non-insulin-treated adults with Type 2 diabetes: A self-regulation perspective. *Diabetes Medicine*, 32, 1504–1512. doi:10.1111/dme.12745

Suggested Reading

- Brooks, T. L., Leventhal, H., Wolf, M. S., O’Conor, R., Morillo, J., Martynenko, M., ... Federman, A. D. (2015). Strategies used by older adults with asthma for adherence to inhaled corticosteroids. *Journal of General Internal Medicine*, 29(11), 1506–1512.
- Leventhal, H., Weinman, J., Leventhal, E., & Phillips, L. A. (2008). Health psychology: The search for pathways between behavior and health. *Annual Review of Psychology*, 59, 477–505. doi:10.1146/annurevpsych.59.103006.093643
- Levine, J. C., Burns, E., Whittle, J., Fleming, R., Knudson, P., Flax, S., & Leventhal, H. (2016). Randomized trial of technology-assisted self-monitoring of blood glucose by low-income seniors:

- Improved glycemic control in type 2 diabetes mellitus. *Journal of Behavioral Medicine*, 39(6), 1001–1008.
- Phillips, L. A., Cohen, J., Burns, E. A., Abrams, J., & Renninger, S. (2016). Self-management of chronic illness: The role of 'habit' vs reflective factors in exercise and medication adherence. *Journal of Behavioral Medicine*, 39(6), 1076–1091. doi:10.1007/s10865-016-9732-z
- Phillips, L. A., Leventhal, H., & Leventhal, E. A. (2012). Physicians' communication of the Common-Sense Self-Regulation Model results in greater reported adherence than physicians' use of interpersonal-skills. *British Journal of Health Psychology*, 17(2), 244–257. doi:10.1111/j.2044-8287.2011.02035.x

Implicit Processes and Health Behavior Change

Aya Avishai and Paschal Sheeran

Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Prominent theories in health psychology, including the health belief model, protection motivation theory, social cognitive theory, the transtheoretical model, and the theory of planned behavior, all assume that changing people's conscious beliefs (e.g. behavioral intentions, risk appraisals) will promote health behavior change. However, meta-analyses indicate that a medium-to-large change in intention engenders only a small-to-medium change in behavior and that a large change in risk appraisal has only a small effect on behavior. Dual-process models of health behavior explain this modest impact of conscious beliefs on behavior by pointing out that health behavior is governed not only by conscious beliefs but also by implicit processes that could activate conflicting behavioral schemata. More particularly, the reflective–impulsive model (RIM) proposes that there are two information-processing modes: reflective and impulsive. The reflective mode is slow and based on rules of language and logic. The key processes are reasoning (e.g., from knowledge about the consequences of an action to a decision to act) and intending. The impulsive mode, on the other hand, draws upon the store of associations that the person has acquired over many experiences. The key impulsive process is spreading activation whereby perceptual input activates related elements in the associative store. This mode of information processing is fast and occurs outside of awareness. Accordingly, the defining feature of implicit processes is that people do not realize, nor intend, their impact on health decisions and actions.

The present entry focuses on the implications of implicit processes for interventions to promote health behavior change. In an effort to organize the large and disparate literature on this topic, Figure 1 presents a framework for intervening with implicit processes. The organizing principle concerns the nature and target of the intervention (what factor is modified and how) and suggests that three overarching intervention approaches can be identified:

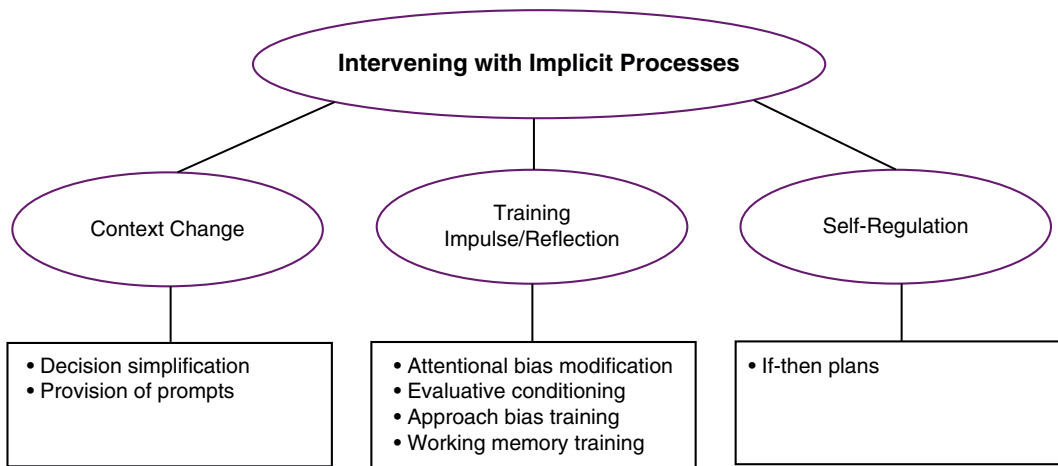


Figure 1 A framework for intervening with implicit processes.

- *Context change* interventions alter features of the physical or social setting in order to change health behaviors. Context change interventions are of two broad types: interventions geared at simplifying decisions and interventions that install prompts. The provision of prompts can guide behavior via the activation of goals or norms or by increasing self-monitoring.
- *Training impulse/reflection* involves practice-based interventions that are geared at modifying either impulsive responses (i.e., attentional bias, implicit affect, or approach bias) or the person's reflective control over impulsive responding.
- *Self-regulation* interventions give people tools to better align their thoughts, feelings, and actions with their health goals. Forming if-then plans or *implementation intentions* (Gollwitzer & Sheeran, 2006) is the most extensively researched and best validated of these tools.

Changing the Context

Decision Simplification

Simplifying decisions reduces the burden on the reflective system and helps to ensure that implicit choices are healthful. Health decisions can be simplified by the options that are made available, the ease with which those options can be selected, and the default options that are already in place. Correlational studies indicate that the availability of fresh fruit, vegetables, and low-fat milk is inversely related to overweight/obesity and that the availability of cigarettes is positively associated with smoking rates. When an intervention increased the availability of healthy food and drinks in vending machines by 50%, the proportion of healthy items that was purchased also increased (French et al., 2010).

Introducing variety can also improve health decisions: When individuals can choose a different fruit to one they had consumed earlier, then fruit is more likely to be selected than a sweet. The ease with which an option can be chosen also is important. An experiment among coffee shop consumers found that when the cognitive demands involved in making healthy eating decisions were lowered, there was a decline in the proportion of high-calorie items purchased (Allan, Johnston, & Campbell, 2015). Defaults also simplify decisions by creating a standard, preselected

option that does not require people to make an active choice. For instance, a country's default could be that all residents are registered as organ donors (and residents opt out if they do not want to register) or that residents are not registered as donors by default (and opt in if they wish to register). When countries with the different defaults are compared, countries with an opt-out default have higher rates of donor registration than countries with an opt-in default.

Provision of Prompts

Changing self-reported (explicit) goals and norms and increasing conscious monitoring of behavior are known to promote health behaviors. The provision of prompts can also instigate these processes implicitly. For instance, Papies and colleagues primed the goal of eating healthily among patrons of a butcher's store by putting a sign about a low-fat recipe at the entrance (or not). Restrained eaters ate fewer meat snacks that were available on the store counter when the sign was present than when it was absent. Similarly, a flyer about low-fat recipes also decreased snack purchases (by 75%) among overweight/obese patrons of a grocery store (see Papies, 2016, for a review).

Social norms also exert an implicit influence on health behavior. For instance, people eat more when they are in pairs or in groups of four than when they are alone, and people are largely unaware of how dining companions influence their eating behavior—citing taste and hunger as key influences instead. Portion size, plate size, and the amount of food on the plate can each activate consumption norms. Including photographs of vegetables on the bottom of food trays in a school cafeteria doubled the proportion of students eating green beans and tripled the proportion eating carrots (Reicks, Redden, Mann, Mykerezi, & Vickers, 2012).

Injunctive norms or social pressure can also be primed. People entering a hospital were twice as likely to engage in hand hygiene when a picture of a pair of eyes was placed over the sanitary gel dispenser (King et al., 2015). Subtle prompts can also promote self-monitoring. Geier, Wansink, and Rozin (2012) gave participants containers of potato chips and had them watch a movie. Participants who encountered a red potato chip following a fixed number of regular chips (and thus could monitor their consumption) ate half as many chips as participants who were given only regular chips. Similarly, in a study that examined eating wings at a sports bar, participants whose food debris was left on their plate ate less than those whose finished wings and bones were removed by the waitstaff (Wansink & Payne, 2007).

Training Impulse–Reflection

People who smoke, drink, or overeat tend to show biased attention to relevant stimuli, more positively evaluate those stimuli on implicit measures, and exhibit greater approach motivation in relation to those stimuli. Interventions have targeted both the respective impulses (attentional retraining for attentional bias, evaluative conditioning [EC] for implicit affect, and approach bias training for motivational bias) as well as people's reflective control over such impulses (working memory [WM] training).

Attentional Retraining

Attentional retraining modifies paradigms such as the Stroop and visual dot probe (VDP) so that participants gain practice at not giving attention to unhealthy stimuli. A single session of attentional retraining with heavy drinkers decreased attention to alcohol relative to soft drinks

(Schoenmakers, Wiers, Jones, Bruce, & Jansen, 2007), indicating that attentional bias could be altered. Craving for alcohol and preference behavior were not affected by the intervention, however. Repeated sessions appeared to have stronger effects. In the Alcohol Attention-Control Training Program (AACTP), participant bias is assessed, goals for attentional bias reduction are set, and participants' progress is monitored. Hazardous and harmful drinkers who undertook the program demonstrated a reduction in alcohol attentional bias as well as a reduction in alcohol consumption over 3 months (Fadardi & Cox, 2009).

Schoenmakers et al. (2010) undertook the first small RCT of repeated attentional retraining in alcohol-dependent patients. Results showed an increase in attentional bias in the control group and a significant decrease in attentional bias for alcohol stimuli in the experimental group. The experimental group was also discharged from treatment sooner and relapsed later than the control group. Attentional retraining also proved effective in reducing chocolate consumption. However, a recent double-blind RCT observed no reliable effect on smoking cessation outcomes.

Evaluative Conditioning

EC tasks repeatedly pair a focal stimulus (e.g., beer) with a highly valenced stimuli (e.g., illness) in order to alter the underlying affective associations. Participants who completed an EC task that paired beer with negative affect exhibited less positive attitudes, had fewer cravings, and consumed less beer after training than did those in the control group (Houben, Schoenmakers, & Wiers, 2010). In a second study, participants who underwent EC had more negative implicit attitudes regarding alcohol and also drank less alcohol during the subsequent week compared with controls. EC was also effective in altering eating attitudes and behavior. After training to associate snacks with unhealthy outcomes, participants' implicit attitudes toward those snacks were more negative, and they were less likely to choose a snack as a parting gift after the experiment (Hollands, Prestwich, & Marteau, 2011). Learning to associate fruit with positive images also increased the proportion of fruit selected in a behavioral choice task.

Approach Bias Training

Repeated consumption of unhealthy substances forges associations between those stimuli and approach responses. Approach bias training targets this automatic motivational tendency. Go/no-go, stop signal, and approach/avoidance tasks have each been used in approach bias training. Heavy drinkers who undertook a go/no-go task that paired beer with "no-go" responses exhibited more negative implicit attitudes toward alcohol and also consumed less alcohol (Houben, Nederkoorn, Wiers, & Jansen, 2011). A go/no-go task involving palatable foods engendered a significant reduction in weight over 1 month, and training was especially effective among participants with higher BMI at the outset of the intervention (Veling, van Koningsbruggen, Aarts, & Stroebe, 2014).

Stop signal training consistently presents behavioral stop signals in close temporal proximity to high-calorie foods in order to create strong associations between these foods and response inhibition. Stop signals proved effective in reducing snack consumption in multiple studies, and the observed effects were especially pronounced when participants were hungry or habitually consumed the relevant snack food.

Wiers, Eberl, Rinck, Becker, & Lindenmeyer (2011) examined the impact of approach/avoidance training on relapse rates among patients with alcoholism. Patients were instructed to respond to pictures of alcohol by making an avoidance movement (pushing the joystick) and to pictures of nonalcoholic soft drinks by making an approach movement (pulling the

joystick). Approach/avoidance training reduced approach bias and implicit attitudes toward alcohol and also led to better treatment outcomes 1 year later.

Working Memory Training

Automatic impulses have greater influence on behavior among participants with low WM capacity and when WM is compromised by cognitive load. The implication is that WM training could reduce the impact of antagonistic implicit processes on health behaviors. Houben, Wiers, and Jansen (2011) reported that 25 sessions of WM training (one session every 2 days) significantly increased memory span. Training also reduced weekly alcohol consumption both 1 week and 5 weeks post-training. WM training proved especially effective in reducing alcohol consumption among participants who had strong implicit preferences for alcohol at the outset. Although there is much debate about the magnitude and mechanisms of WM training transfer effects, research is ongoing to test the impact of WM training on other health behaviors.

Self-Regulation

If-then plans or *implementation intentions* are a key self-regulation tool in health psychology. Implementation intentions provide a structure in which people (a) identify key opportunities for, or obstacles to, performing health behaviors, (b) specify a way to respond to each opportunity and obstacle, and then (c) formalize a link between the opportunity or obstacle and the response:

If (opportunity/obstacle) arises, then I will (respond in this way)!

Meta-analyses indicate that forming implementation intentions promotes effective self-regulation (Gollwitzer & Sheeran, 2006) and leads to greater physical activity and healthy eating. Implementation intentions are an effective intervention for health behavior change because the opportunities or obstacles specified in plans become highly accessible and thus people are in a good position to identify their moment to act when they encounter it. Moreover, strong associations are forged between the opportunity/obstacle and the specified response, meaning that people are in a good position to seize that moment and respond exactly as they had spelled out in advance.

Evidence indicates that forming if-then plans endows responses with key features of automaticity (i.e., swift and effortless responding). Thus, although implementation intentions are formed in a conscious act of will, automatic processes are recruited that serve to promote the initiation of the planned responses. This marriage of explicit and implicit processes that characterizes the operation of if-then plans has been termed “strategic automaticity.” Neurophysiological evidence supports this characterization. Whereas action control by conscious intentions operates in a “top-down” manner, if-then plans operate in “bottom-up” fashion. The stimulus control of behavior observed in implementation intentions is akin to the operation of habits.

The previous section reviewed evidence that extensive practice-based training can help to overcome the unwanted impact of implicit goals, attitudes, and social influence on health behaviors. However, if-then plans also offer a simple and effective strategy for regulating unwanted implicit influence. For instance, Gollwitzer, Sheeran, Trötschel, and Webb (2011) observed that forming if-then plans about how to drive safely protected driving behavior from the antagonistic impact of priming the goal of being fast. Another study measured implicit attitudes toward chocolate and had participants form if-then plans to instigate reflection

whenever they were tempted to eat some (“If I am tempted to have chocolate, then I ask myself, ‘do I really want to do this?’”). Findings showed that if-then plans led to reduced chocolate consumption over the subsequent week. Whereas positive implicit attitudes predicted greater consumption among control participants, this association was actually reversed for participants who formed if-then plans (Sheeran, 2013). If-then plans also proved effective in reducing the implicit influence of binge drinker stereotypes on young people’s alcohol consumption in a field experiment over 1 month (Rivis & Sheeran, 2013). In sum, implicit processes are key to the effectiveness of implementation intentions in changing health behaviors by automatizing the initiation of healthful actions and controlling disruptive automatic influences.

Conclusion

Implicit processes have important implications for behavior change interventions. Implicit processes can be harnessed in order to promote healthful decisions (e.g., via context change interventions). Moreover, when implicit processes threaten to engender risk behavior, training impulse-reflection and forming if-then plans can prove effective in attenuating their influence. Although research on intervening with implicit processes is still in its early stages and much remains to be done to test interventions over extended periods among both representative samples and patients, this approach holds considerable promise. A key implication of dual-process theories is that behavior change interventions will be most effective if they target not only conscious beliefs but also implicit processes and exploit potential synergies between beliefs and implicit processes.

Acknowledgments

This entry was made possible through the support of a grant from the John Templeton Foundation (#23145). The opinions are the authors’ and do not necessarily reflect the views of the John Templeton Foundation.

Author Biographies

Aya Avishai is a PhD student in the Department of Psychology and Neuroscience at the University of North Carolina at Chapel Hill.

Paschal Sheeran is a professor in the Department of Psychology and Neuroscience at the University of North Carolina at Chapel Hill. His research takes a self-regulation perspective on emotion control and health behavior change and has been published in *Psychological Bulletin*, *Psychological Science*, and *Health Psychology*.

References

- Allan, J. L., Johnston, M., & Campbell, N. (2015). Snack purchasing is healthier when the cognitive demands of choice are reduced: A randomized controlled trial. *Health Psychology, 34*(7), 750–755. doi:10.1037/hea0000173
- Fadardi, J. S., & Cox, W. M. (2009). Reversing the sequence: Reducing alcohol consumption by overcoming alcohol attentional bias. *Drug and Alcohol Dependence, 101*(3), 137–145. doi:10.1016/j.drugalcdep.2008.11.015

- French, S. A., Hannan, P. J., Harnack, L. J., Mitchell, N. R., Toomey, T. L., & Gerlach, A. (2010). Pricing and availability intervention in vending machines at four bus garages. *Journal of Occupational and Environmental Medicine*, 52(Suppl. 1), S29–S33. doi:10.1097/JOM.0b013e3181c5c476
- Geier, A., Wansink, B., & Rozin, P. (2012). Red potato chips: Segmentation cues can substantially decrease food intake. *Health Psychology*, 31(3), 398–401. doi:10.1037/a0027221
- Gollwitzer, P. M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. *Advances in Experimental Social Psychology*, 38, 69–119. doi:10.1016/S0065-2601(06)38002-1
- Gollwitzer, P. M., Sheeran, P., Trötschel, R., & Webb, T. L. (2011). Self-regulation of priming effects on behavior. *Psychological Science*, 22(7), 901–907. doi:10.1177/0956797611411586
- Hollands, G. J., Prestwich, A., & Marteau, T. M. (2011). Using aversive images to enhance healthy food choices and implicit attitudes: An experimental test of evaluative conditioning. *Health Psychology*, 30(2), 195–203. doi:10.1037/a0022261
- Houben, K., Nederkoorn, C., Wiers, R. W., & Jansen, A. (2011). Resisting temptation: Decreasing alcohol-related affect and drinking behavior by training response inhibition. *Drug and Alcohol Dependence*, 116(1–3), 132–136. doi:10.1016/j.drugalcdep.2010.12.011
- Houben, K., Schoenmakers, T. M., & Wiers, R. W. (2010). I didn't feel like drinking but I don't know why: The effects of evaluative conditioning on alcohol-related attitudes, craving and behavior. *Addictive Behaviors*, 35(12), 1161–1163. doi:10.1016/j.addbeh.2010.08.012
- Houben, K., Wiers, R. W., & Jansen, A. (2011). Getting a grip on drinking behavior: Training working memory to reduce alcohol abuse. *Psychological Science*, 22(7), 968–975. doi:10.1177/0956797611412392
- King, D., Vlaev, I., Everett-Thomas, R., Fitzpatrick, M., Darzi, A., & Birnbach, D. J. (2015). "Priming" hand hygiene compliance in clinical environments. *Health Psychology*. doi:10.1037/hea0000239
- Papies, E. K. (2016). Health goal priming as a situated intervention tool: How to benefit from nonconscious motivational routes to health behavior. *Health Psychology Review*, 10(4), 408–424.
- Reicks, M., Redden, J. P., Mann, T., Mykerez, E., & Vickers, Z. (2012). Photographs in lunch tray compartments and vegetable consumption among children in elementary school cafeterias. *JAMA*, 307(8), 784–785. doi:10.1001/jama.2012.170
- Rivis, A., & Sheeran, P. (2013). Automatic risk behavior: Direct effects of binge drinker stereotypes on drinking behavior. *Health Psychology*, 32(5), 571–580. doi:10.1037/a0029859
- Schoenmakers, T., Wiers, R. W., Jones, B. T., Bruce, G., & Jansen, A. M. (2007). Attentional re-training decreases attentional bias in heavy drinkers without generalization. *Addiction*, 102(3), 399–405. doi:10.1111/j.1360-0443.2006.01718.x
- Schoenmakers, T. M., de Bruin, M., Lux, I. M., Goertz, A. G., Van Kerkhof, D. T., & Wiers, R. W. (2010). Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. *Drug and Alcohol Dependence*, 109(1–3), 30–36. doi:10.1016/j.drugalcdep.2009.11.022
- Sheeran, P. (2013, April). Self-control over automatic attitudes and behaviour. Keynote address presented at the Implicit Bias and Philosophy Conference, Sheffield, UK.
- Veling, H., van Koningsbruggen, G. M., Aarts, H., & Stroebe, W. (2014). Targeting impulsive processes of eating behavior via the internet. Effects on body weight. *Appetite*, 78(C), 102–109. doi:10.1016/j.appet.2014.03.014
- Wansink, B., & Payne, C. R. (2007). Counting bones: Environmental cues that decrease food intake. *Perceptual and Motor Skills*, 104(1), 273–276. doi:10.2466/PMS.104.1.273-276
- Wiers, R. W., Eberl, C., Rinck, M., Becker, E. S., & Lindenmeyer, J. (2011). Retraining automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychological Science*, 22(4), 490–497. doi:10.1177/0956797611400615

Suggested Reading

- Marteau, T. M., Hollands, G. J., & Fletcher, P. C. (2012). Changing human behavior to prevent disease: The importance of targeting automatic processes. *Science*, 337(6101), 1492–1495. doi:10.1126/science.1226918

- Sheeran, P., Gollwitzer, P. M., & Bargh, J. A. (2013). Nonconscious processes and health. *Health Psychology, 32*(5), 460–473. doi:10.1037/a0029203
- Wansink, B. (2006). *Mindless eating: Why we eat more than we think*. New York: Bantam.
- Wiers, R. W., Gladwin, T. E., Hofmann, W., Salemink, E., & Ridderinkhof, K. R. (2013). Cognitive bias modification and cognitive control training in addiction and related psychopathology: Mechanisms, clinical perspectives, and ways forward. *Clinical Psychological Science, 1*(2), 192–212. doi:10.1177/2167702612466547

Intimate Relationships and Physical Health

Fiona Ge*, Jana Lembke, and Paula R. Pietromonaco

*Department of Psychological and Brain Sciences, University of
Massachusetts Amherst, Amherst, MA, USA*

Romantic relationships are connected to physical health in many ways. Simply being in a romantic relationship generally predicts better health outcomes, but the quality of the relationship, individual differences such as attachment style (expectations that partners will or will not be supportive and responsive to one's needs), and couples' behavioral interaction styles also play a role in health. We review how each of these broad categories of predictors is associated with people's voluntary health-related behaviors, health-related physiological responses, and health and disease outcome and discuss applications of this research for medical practice.

Relationship Status and Quality

People who are in a happy relationship tend to evidence better health than those who are not in a relationship or are in a poor-quality relationship. According to a recent meta-analysis, marital quality is linked to health via multiple pathways, including physiological markers, health behaviors, and direct health outcomes (Robles, Slatcher, Trombello, & McGinn, 2014).

Physiological Health Correlates of Relationship Quality

Research examining relationship quality and endocrine function has focused on the hypothalamic–pituitary–adrenal (HPA) axis, a major stress response system that governs the release of cortisol, a steroid hormone. Cortisol is released into the bloodstream in response to environmental stressors, and it affects metabolic, immune, and nervous system functioning. Over time, the continued release of cortisol may have adverse effects on the body, which can increase

*The first and second authors contributed equally to this entry.

The Wiley Encyclopedia of Health Psychology: Volume 2: The Social Bases of Health Behavior, First Edition.

General Editor: Lee M. Cohen. Volume Editors: Kate Sweeny and Megan L. Robbins.

© 2021 John Wiley & Sons Ltd. Published 2021 by John Wiley & Sons Ltd.

susceptibility to illness and accelerate chronic disease. Conversely, a flattened cortisol slope (low reactivity) in response to a stressor also serves as an index of psychological maladjustment and is typically associated with self-regulatory problems under chronic stress. Consistent with this idea, poorer marital quality has been related to flatter cortisol responses throughout the day for men and women. Women in more satisfying relationships, however, show greater morning cortisol levels and a steeper decline in cortisol levels throughout a typical work day, indicating better physiological recovery from work than women who were less satisfied with their marriages.

Although partners' daily cortisol levels tend to be related, greater relationship satisfaction may shield spouses from the effects of each other's cortisol responses to stress. For instance, women's cortisol levels are less closely associated with their husband's cortisol levels when the women were more satisfied in their relationship. Additionally, spouses who reported greater marital satisfaction showed less convergence in cortisol patterns during a discussion of relationship conflict (Laws, Sayer, Pietromonaco, & Powers, 2015). Thus, high-quality relationships may minimize the extent to which one partner's physiological stress responses spill over to those of their partner, although the mechanisms through which this process occurs remain to be specified.

Functioning of the cardiovascular system is another biological indicator of health, and lower reactivity is generally better. Overall, individuals in high-quality marriages have lower cardiovascular reactivity than single individuals and even more so than individuals in low-quality marriages. Similarly, marital dissatisfaction has been associated with higher blood pressure among individuals with mild hypertension. These findings indicate that relationship quality—not just relationship status—matters for health.

A recent survey based on a US national sample found that greater marital adjustment was associated with less interleukin-6, a pro-inflammatory cytokine and a marker of inflammation, particularly for younger women (Whisman & Sbarra, 2012). For men, in contrast, being married buffered against elevations in C-reactive protein, another marker of inflammation, compared with men who were unmarried. It has also been found that older, happily married individuals may evidence stronger antibody responses than those who are unmarried, bereaved, or in an unhappy marriage. Furthermore, it has been found that people who perceive that their relationship contains both positive and negative aspects have greater inflammation compared with those who perceived their relationship in a primarily positive way, suggesting that relationship ambivalence has negative consequences for health.

Relationship Quality and Health Behavior

Being in a relationship can motivate health-enhancing behaviors, perhaps because those behaviors are viewed as a shared goal for both members of the couple. Furthermore, individuals in a relationship may have greater support in seeking healthcare and following treatment regimens. For instance, people who were married or had a partner were 1.5–2 times more likely to attend cardiac rehabilitation than their unpartnered counterparts (Molloy, Hamer, Randall, & Chida, 2008).

In some cases, however, relationships do not promote better health. Having a partner with unhealthy habits—such as smoking—may hinder the other partner's attempts to break similar habits. Satisfaction with one's relationship, not just relationship status, also relates to health. For instance, while married individuals generally show healthier sleep patterns than unmarried individuals, being in a more satisfying marriage is associated with fewer sleep disturbances in women. Couples in more satisfying marriages also are more likely to use healthcare services than couples in distressed marriages. In contrast, greater relationship dissatisfaction tends to promote health-compromising behaviors, such as greater alcohol consumption.

Relationship Quality, Mortality, and Disease Recovery

Mortality risk is generally lower among those who are married versus unmarried, who marry later in life, or who have been married for a longer period. In addition, being in a relationship has been linked to lower morbidity and higher survival rates after the diagnosis of different diseases or surgery, such as cardiovascular disease, coronary heart disease, hospital-diagnosed infectious diseases, and coronary artery bypass grafting.

Relationship quality has also been linked to physical health outcomes. For example, greater marital quality predicted a better survival rate over 8 years for patients with heart failure, especially among women (see Robles et al., 2014). Similarly, greater relationship negativity predicted a higher risk of coronary heart disease. Additionally, the risk of coronary artery calcification was higher when both partners had positive and negative (ambivalent) perceptions of each other than when one partner had ambivalent perceptions and the other perceived the partner as purely positive (Uchino, Smith, & Berg, 2014).

Attachment and Health

Based on day-to-day experiences in close relationships from childhood through adulthood, people develop attachment orientations (e.g., expectations, beliefs, goals) that guide how they think, feel, and behave with close partners. These attachment orientations also promote different ways of perceiving and regulating stress, which in turn affect health-related physiological patterns. Overreacting to and poorly recovering from relationship stressors—a tendency observed in insecurely attached individuals—can exhaust physiological systems and eventually produce health problems.

Physiological Health Correlates of Attachment

Both attachment avoidance and anxiety are associated with disruptions in the HPA axis, but because anxiously attached people are particularly sensitive to relationship stressors and are continually monitoring the relationship for signs of threat, they are susceptible to heightened HPA responses. For example, anxiously attached individuals exhibit elevated HPA axis activity during travel separations, a situation that threatens anxious people's need for their partner's closeness (see Pietromonaco, DeVito, Ge, & Lembke, 2015). Anxiously attached individuals also show increased cortisol levels in response to generalized stressors in experimental settings, and because they underestimate their partner's availability and care, they take longer than nonanxious individuals to recover from the stressor even when presented with support from their partner.

Recent evidence sheds light on the interplay of both partner's attachment on stress responses. Couples consisting of an anxious wife and an avoidant husband showed increased cortisol reactivity in anticipation of a discussion of relationship conflict followed by a sharp decline before the discussion began (Beck, Pietromonaco, DeBuse, Powers, & Sayer, 2013). This pattern was accompanied by less constructive behaviors during the conflict and was distinct from patterns observed in other attachment pairings. These findings, together with other recent work (see Pietromonaco & Powers, 2015), underscore the importance of the situational context (e.g., both partners' characteristics) in understanding the link between attachment and physiological responses.

Attachment insecurity (anxiety, avoidance, or both) is associated with cardiovascular risk factors. Attachment avoidance predicts elevated cardiovascular reactivity in the face of

attachment-related stressors, a likely consequence of avoidant people's defensive, repressive, and distancing coping strategies. Although a partner's presence should mitigate insecure individuals' sympathetic nervous system arousal in the face of stress, this does not seem to be the case. Avoidant and anxious individuals who were anticipating a stressful situation showed greater cardiovascular reactivity to stress in their partner's presence versus absence, suggesting that they were not comforted by their partner once the attachment system was activated, perhaps because they were anticipating not only the stressor but also the possibility of being rejected by their partners.

Attachment insecurity may impact health via immune functioning, which can be measured via speed of wound healing, inflammatory cytokines (IL-6), and T-cell counts. While this area of research is just developing, immune functioning seems to be impaired by attachment insecurity. Other work suggests that the link between attachment and immune functioning is moderated by gender. It is possible that some markers of immune functioning vary with one type of attachment insecurity but not the other or that other moderating variables are involved (e.g., relationship quality, situational aspects). Research building on these initial findings will yield a better understanding of the link between attachment insecurity and immune system dysregulation.

Attachment and Health Behavior

Attachment insecurity is linked to a variety of health-compromising behaviors, such as riskier sexual behavior, greater substance use, lower levels of exercise and higher rates of smoking, less participation in preventative healthcare, and poorer relationships with physicians.

Factors such as self-esteem and communication styles may partly explain the link between attachment and health behavior (see Pietromonaco et al., 2015). For example, securely attached university students held higher self-esteem than those who were insecurely attached, which in turn predicted their greater engagement in health behavior. Communication styles may explain the link between attachment and risky sexual behavior: anxiously attached individuals were less likely to talk with their partners about AIDS-related issues, which predicted riskier sexual behavior.

The Dyadic Influence of Attachment on Health

Research integrating work on attachment and social support has found that people low in attachment anxiety reported better health outcomes when they perceived their partners as more supportive, but individuals high in attachment anxiety did not benefit from their partners' greater support (Stanton & Campbell, 2014). Relatedly, individuals who were anxiously attached and who perceived lower social support showed greater severity of inflammatory bowel disease severity. Because anxious individuals' relationship fears may cause them to underestimate their partner's support, these findings indicate that individuals' *perceptions* of relationship support matter for health, not just the amount of support.

Another line of research has taken into account the interplay between both partners' attachment styles in relation to health. When caregivers and patients with Alzheimer's disease (AD) were both high in anxious attachment, the patients reported greater physical symptoms (e.g., pain, nausea); when caregivers were low in anxious attachment, patients' attachment anxiety was unrelated to their reported physical symptoms (Monin, Schulz, & Kershaw, 2013). Caregivers' low attachment anxiety may buffer AD individuals' perceptions of their symptoms, particularly for those who are anxiously attached.

Negative Relationship Interactions

Although high-quality relationships provide a source of support for individuals' health and well-being, relationship interactions—when they go wrong—can be a major source of stress (Robles et al., 2014).

Physiological Correlates of Conflict

Research on relationship conflict and endocrine responses has focused on the stress hormone cortisol. Greater positive behaviors during a conflict have been associated with increased cortisol levels, while spouses who displayed mainly negative behaviors showed no change in cortisol levels during the conflict, a nonresponsive pattern that is often linked to poorer outcomes. Another potentially problematic response pattern is heightened cortisol reactivity, which has been found among men who coped with stress by seeking support, women who received less spousal support, and men and women whose partners had greater need for social support.

Relationship-oriented factors also contribute to individuals' endocrine reactivity in response to a conflict. Greater demand-withdrawal communication patterns, characterized by wives' criticism and blaming and husbands' avoidance and disengagement, are associated with greater cortisol reactivity in response to a conflict for both spouses. Similarly, husbands' withdrawal in response to wives' negative behaviors was associated with wives' greater cortisol reactivity (Kiecolt-Glaser et al., 1996).

Negative relationship interactions evoke greater cardiovascular reactivity, such as blood pressure, heart rate, and cardiac output. Hostile interactions are linked to couples' increased blood pressure, whereas positive or neutral interactions are not linked to a change in blood pressure, implying that the negative aspects of relationship interactions typically outweigh the positive or neutral aspects of the discussions in influencing physiological reactivity. In fact, dispositionally hostile husbands (vs. low hostile husbands) paired with hostile wives evidenced greater resistance to blood flow through the cardiovascular system throughout a conflict discussion (Broadwell & Light, 2005). Other studies examining support provisions during conflict interactions found that low spousal support adversely impacts blood pressure (Heffner et al., 2004).

Engaging in marital conflict has been associated with dysregulation of the immune system, which has negative implications for health such as impaired wound healing (Robles et al., 2014). Conflicts characterized by hostile and negative behaviors are especially likely to elicit poorer immune responses. Social cognitive factors, such as the extent to which people think about their feelings and the circumstances during a conflict discussion, have been associated with immune responses such that high-level cognitive processing seems to buffer against inflammation in the face of stressful events (Graham et al., 2009).

Positive Relationship Interactions

Endocrine Pathway

Positive relationship interactions—such as showing physical affection or support—are important for health, likely because they reduce stress and protect against cortisol increases. Daily displays of intimacy are one way that relationship interactions confer health benefits. In one study, dual-career couples reported time spent on intimacy and provided multiple daily saliva

samples for cortisol analysis over a 1-week period. Greater amounts of intimacy on a given day appeared to increase positive affect, which in turn reduced cortisol, suggesting that positive affect generated by intimacy aids in stress reduction (Ditzen, Hoppmann, & Klumb, 2008). Similarly, husbands' supportive behaviors during heated conflict interactions helped down-regulate stress hormones in wives, perhaps because husbands' constructive engagement during conflict reduces wives' perceived threat and speeds recovery from physiological stress.

Cardiovascular Pathway

Simply being in a loving, low-conflict relationship supports physiological patterns necessary for good cardiovascular health for both men and women. For instance, the extent to which partners viewed their romantic relationships as positive, important, and secure predicts life satisfaction and reductions in nighttime blood pressure, especially for women. In a sample of 250 healthy older adults, frequent spousal interaction reduced progression of carotid artery thickening for men in well-adjusted marriages (Janicki, Kamarck, Shiffman, Sutton-Tyrrell, & Gwaltney, 2005).

Perceiving one's partner as supportive in times of stress also promotes physiological responses correlated with better cardiovascular health. One study of 94 married couples found that perceptions of informational support from one's partner helped reduce the effects of momentary, everyday life stress on systolic and diastolic blood pressure. This finding suggests that providing useful advice or helping with problem solving protects against blood pressure increases that are associated with cardiovascular risk. Other work suggests that it is not the type of support received during a conflict, but whether partners were happy with the support that is associated with blood pressure and cortisol reactivity (Heffner et al., 2004).

Relationships also may impact cardiovascular health via loving touch. A history of frequent physical contact between partners has been associated with higher oxytocin production and lower cardiovascular reactivity. Experimentally induced touch has a similar effect: cohabiting couples who engaged in handholding followed by a 20-s hug showed attenuated blood pressure and heart rate reactivity in response to a public speaking task compared with those in a no-touch control group (Grewen, Anderson, Girdler, & Light, 2003). Also, evidence suggests that positive physical contact (but not verbal support) can buffer against physiological responding to a stressor. Physical contact between partners can even be used as intervention to improve health. Compared with a no-treatment control group, couples participating in a 4-week-long "warm touch" intimacy-building intervention showed enhanced oxytocin, lower stress hormones, and, among husbands, lower systolic blood pressure (Holt-Lunstad, Birmingham, & Light, 2008). Clearly, warm and affectionate relationships contribute to lower sympathetic nervous system reactivity to acute stressors, a correlate of good cardiovascular health.

Immune Pathway

Provisions of support confer benefits for physical health via inflammatory responses over and above individual differences. For example, wives in more well-adjusted marriages showed lower blood levels of IL-6 (a marker of inflammation), even after controlling for significant demographic and health-related variables, and reports of partner *support* (but not negative partner interactions) were uniquely associated with IL-6 (Whisman & Sbarra, 2012).

Another way to measure immune functioning is via the body's ability to heal from injury. Neurochemical activity related to positive relationship functioning aids in wound healing: compared with individuals who showed lower levels of oxytocin, individuals exhibiting the highest levels (i.e., upper quartile) of oxytocin healed faster from experimentally induced

wounds (Gouin et al., 2010). Greater oxytocin and vasopressin also were associated with more positive communication behaviors and fewer negative behaviors during a social support marital interaction. Thus, supportive marital interactions may promote greater oxytocin and vasopressin response, which counteract stress and aid in wound healing.

Recommendations for Future Research and Conclusion

More work is needed to determine the conditions under which physiological reactivity to momentary relationship stressors translates to observable, long-term effects on health. For example, attachment is associated with subtle biological stress response patterns that likely translate to concrete health outcomes over time, but limited work has directly tested this link. Also, it is unclear how long it takes for psychophysiological responses (e.g., heightened cortisol reactivity) to produce a diagnosable disease. Because serious health problems such as cancer and arteriosclerosis can take years to develop, it will be important to examine the connection between relationship processes and health over time.

Additional work is needed on clinical samples such as couples in which one partner has a chronic illness. Recent research by Manne, Siegel, Kashy, and Heckman (2014) illustrated the potential for conducting basic psychological research with applications for medical practice: in a study of 254 women with breast cancer and their husbands, how couples viewed the cancer (e.g., going through it as a team) affected their intimacy, and this association was mediated by how they communicated (i.e., having greater perceived self-disclosure and responsiveness). Medical treatments for chronic illness that include a relationship component hold promise: A meta-analysis found that patients receiving a couple-oriented intervention showed greater improvements in their depressive symptoms, marital functioning, and pain than when only usual medical care was prescribed (Martire, Schulz, Helgeson, Small, & Saghafi, 2010). This research points to relationship interventions as potential nonpharmacological medical treatments. Indeed, research at the nexus of health psychology and relationship science has the promise to be translated to medical practice and lay a groundwork for developing new, low-risk methods of improving physical health.

Author Biographies

Fiona Ge is a PhD student in social psychology at the University of Massachusetts Amherst. She is interested in studying how social interactions between romantic partners shape individuals' emotional, physiological, and social well-being. She is also interested in examining how communication patterns among romantic couples vary by culture.

Jana Lembke is a social psychology graduate student at the University of Massachusetts Amherst. Her research focuses on the role of positive partner interactions in relationship well-being. She is currently examining how couples reconnect with each other following instances of relationship conflict and how these types of interactions may contribute to relationship satisfaction.

Paula R. Pietromonaco is a professor in the Department of Psychological and Brain Sciences at the University of Massachusetts Amherst. Her research focuses on attachment, emotion, and health. Her current work examines how couple members influence each other's psychological, behavioral, and physiological responses to stress and the extent to which these processes contribute to their emotional and physical health over time.

References

- Beck, L. A., Pietromonaco, P. R., DeBuse, C. J., Powers, S. I., & Sayer, A. G. (2013). Spouses' attachment pairings predict neuroendocrine, behavioral, and psychological responses to marital conflict. *Journal of Personality and Social Psychology*, 105(3), 388–424. <http://doi.org/10.1037/a0033056>
- Broadwell, S. D., & Light, K. C. (2005). Hostility, conflict and cardiovascular responses in married couples: A focus on the dyad. *International Journal of Behavioral Medicine*, 12(3), 142–152. http://doi.org/10.1207/s15327558ijbm1203_3
- Ditzen, B., Hoppmann, C., & Klumb, P. (2008). Positive couple interactions and daily cortisol: On the stress-protecting role of intimacy. *Psychosomatic Medicine*, 70(8), 883–889. <http://doi.org/10.1097/PSY.0b013e318185c4fc>
- Gouin, J.-P., Carter, C. S., Pournajafi-Nazarloo, H., Glaser, R., Malarkey, W. B., Loving, T. J., ... Kiecolt-Glaser, J. K. (2010). Marital behavior, oxytocin, vasopressin, and wound healing. *Psychoneuroendocrinology*, 35(7), 1082–1090. <http://doi.org/10.1016/j.psyneuen.2010.01.009>
- Graham, J. E., Glaser, R., Loving, T. J., Malarkey, W. B., Stowell, J. R., & Kiecolt-Glaser, J. K. (2009). Cognitive word use during marital conflict and increases in proinflammatory cytokines. *Health Psychology*, 28(5), 621–630. <http://doi.org/10.1037/a0015208>
- Grewen, K. M., Anderson, B. J., Girdler, S. S., & Light, K. C. (2003). Warm partner contact is related to lower cardiovascular reactivity. *Behavioral Medicine*, 29(3), 123–130. <http://doi.org/10.1080/08964280309596065>
- Heffner, K. L., Kiecolt-Glaser, J. K., Loving, T. J., Glaser, R., & Malarkey, W. B. (2004). Spousal support satisfaction as a modifier of physiological responses to marital conflict in younger and older couples. *Journal of Behavioral Medicine*, 27(3), 233–254.
- Holt-Lunstad, J., Birmingham, W. A., & Light, K. C. (2008). Influence of a “warm touch” support enhancement intervention among married couples on ambulatory blood pressure, oxytocin, alpha amylase, and cortisol. *Psychosomatic Medicine*, 70(9), 976–985. <http://doi.org/10.1097/PSY.0b013e318187aef7>
- Janicki, D. L., Kamarck, T. W., Shiffman, S., Sutton-Tyrrell, K., & Gwaltney, C. J. (2005). Frequency of spousal interaction and 3-year progression of carotid artery intima medial thickness: The Pittsburgh healthy heart project. *Psychosomatic Medicine*, 67(6), 889–896. <http://doi.org/10.1097/01.psy.0000188476.87869.88>
- Kiecolt-Glaser, J. K., Newton, T., Cacioppo, J. T., MacCallum, R. C., Glaser, R., & Malarkey, W. B. (1996). Marital conflict and endocrine function: Are men really more physiologically affected than women? *Journal of Consulting and Clinical Psychology*, 64(2), 324–332. <http://doi.org/10.1037/0022-006X.64.2.324>
- Laws, H., Sayer, A., Pietromonaco, P. R., & Powers, S. I. (2015). Longitudinal changes in spouses' HPA responses: Convergence in cortisol patterns during the early years of marriage. *Health Psychology*, 34(11), 1076.
- Manne, S. L., Siegel, S., Kashy, D., & Heckman, C. J. (2014). Cancer-specific relationship awareness, relationship communication, and intimacy among couples coping with early-stage breast cancer. *Journal of Social and Personal Relationships*, 31(3), 314–334. <http://doi.org/10.1177/0265407513494950>
- Martire, L. M., Schulz, R., Helgeson, V. S., Small, B. J., & Saghaei, E. M. (2010). Review and meta-analysis of couple-oriented interventions for chronic illness. *Annals of Behavioral Medicine*, 40(3), 325–342. <http://doi.org/10.1007/s12160-010-9216-2>
- Molloy, G. J., Hamer, M., Randall, G., & Chida, Y. (2008). Marital status and cardiac rehabilitation attendance: A meta-analysis. *European Journal of Cardiovascular Prevention & Rehabilitation*, 15(5), 557–561. <http://doi.org/10.1097/HJR.0b013e3283063929>
- Monin, J. K., Schulz, R., & Kershaw, T. S. (2013). Caregiving spouses' attachment orientations and the physical and psychological health of individuals with Alzheimer's disease. *Aging & Mental Health*, 17(4), 508–516. <http://doi.org/10.1080/13607863.2012.747080>

- Pietromonaco, P. R., DeVito, C., Ge, F., & Lembke, J. (2015). Health and attachment processes. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and research: New directions and emerging themes* (pp. 287–318). New York, NY: Guilford Press.
- Pietromonaco, P. R., & Powers, S. I. (2015). Attachment and health-related physiological stress processes. *Current Opinion in Psychology*, 1, 34–39. <http://doi.org/10.1016/j.copsyc.2014.12.001>
- Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological Bulletin*, 140(1), 140–187. <http://doi.org/10.1037/a0031859>
- Stanton, S. C. E., & Campbell, L. (2014). Psychological and physiological predictors of health in romantic relationships: An attachment perspective. *Journal of Personality*, 82(6), 528–538. <http://doi.org/10.1111/jopy.12056>
- Uchino, B. N., Smith, T. W., & Berg, C. A. (2014). Spousal relationship quality and cardiovascular risk dyadic perceptions of relationship ambivalence are associated with coronary-artery calcification. *Psychological Science*, 25(4), 1037–1042. <http://doi.org/10.1177/0956797613520015>
- Whisman, M. A., & Sbarra, D. A. (2012). Marital adjustment and interleukin-6 (IL-6). *Journal of Family Psychology*, 26(2), 290–295. <http://doi.org/10.1037/a0026902>

Suggested Reading

- Loving, T. J., & Slatcher, R. B. (2013). Romantic relationships and health. In J. A. Simpson & L. Campbell (Eds.), *The Oxford handbook of close relationships* (pp. 617–637). New York, NY: Oxford University Press.
- Robles, T. F. (2014). Marital quality and health implications for marriage in the 21st century. *Current Directions in Psychological Science*, 23(6), 427–432. doi:<http://doi.org/10.1177/0963721414549043>
- Wright, B. L., & Loving, T. J. (2011). Health implications of conflict in close relationships. *Social and Personality Psychology Compass*, 5(8), 552–562. <http://doi.org/10.1111/j.1751-9004.2011.00371.x>

Medical Decision Making

Meng Li¹ and Gretchen B. Chapman²

¹*Department of Health and Behavioral Sciences, University of
Colorado Denver, Denver, CO, USA*

²*Department of Psychology, Rutgers University–New Brunswick, New Brunswick, NJ, USA*

Medical decision making encompasses decisions made in all health and medicine contexts by patients, healthcare workers, health policy makers, and the general public. The study of medical decision making follows research on judgment and decision making more generally: It compares actual or descriptive decision making to normative or rational models of decision making, where the latter provide a benchmark for how decisions should be made. It also explores interventions that can improve decision making or guide choice behavior in a particular direction (i.e., toward selection of a particular healthy option).

Medical decisions are often difficult because they entail choosing among options with uncertain outcomes; they require trade-offs between competing goals, often with overwhelming amounts of information; they involve taking on immediate costs for long-term benefits; they may also require strategic interactions among multiple agents. Below we characterize medical decision making by discussing some selective theoretical and empirical issues related to each of these topics.

Risk and Uncertainty

Many medical decisions involve uncertain outcomes. For example, a patient must decide whether to undergo surgery, not knowing if the surgery will be successful. A physician must decide whether to recommend a preventive measure, such as cholesterol-lowering medication, not knowing whether the patient would be fine even without the medication. According to normative expected utility theory, the utility of each outcome (e.g., stroke) should be weighted by the probability that the outcome will occur. Consequently, it is critical that decision makers be able to assess, at least implicitly, the probability of key outcomes and to weight the utility of outcomes in proportion to their probability. In a number of cases, however, outcomes are systematically over- or underweighted, and low likelihood events tend to be overweighted and

thus have more influence on decisions than they should. This phenomenon is illustrated in the American response to the 2014 Ebola outbreak in West Africa. Although contracting Ebola in the United States was an extremely low likelihood event (only four cases of Ebola were diagnosed in the United States, only two of which were contracted in the United States), the response of some US public officials was indicative of a much higher level of risk. For example, in October 2014, the governors of New York and New Jersey announced a mandatory quarantine for any healthcare workers returning from treating Ebola patients in West Africa to the United States via a New York or New Jersey airport. It appears that decision makers draw a large distinction between an outcome with a 0% likelihood and one with a small positive likelihood. For example, people will pay more to reduce the risk of an adverse event from a toxin from 5 in 10,000 to 0 in 10,000 than they will pay for a risk reduction from 15 in 10,000 to 10 in 10,000, even though both are the same net reduction in risk (Viscusi, Magat, & Huber, 1987). Similarly, decision makers draw a large distinction between 100% and a proportion that is slightly smaller than 100%. Consequently, 100% of a small category can seem larger than 50% of a large category, whether the percentages refer to probability, effectiveness, disease coverage, or something else (Li & Chapman, 2009, 2013).

Difficult Trade-Offs

A medical decision can be especially difficult if it encapsulates a trade-off between two important but opposing goals or attributes. Trade-offs in medical decision making are prevalent, such as in the choice between quality and length of life in end-of-life decisions or in the design of screening programs that entail either greater false alarms or potential misses of true pathologies. According to normative multi-attribute utility theory, each attribute should be weighted according to its importance, and an overall utility (goodness) for each option could be computed by summing the weighted utility of all attributes. However, real-life decisions often violate this normative prescription, and the weight that people assign to attributes can shift depending on irrelevant factors, such as how the problem is described.

One often-debated issue in health policy settings involves the trade-off between efficiency and equality, such as in the allocation of scarce medical resources, including transplant organs, scarce vaccines, etc. For example, quality-adjusted life year (QALY) is a widely used metric to assess benefits in medical options. The rationale behind the use of QALYs is that more life years saved represents more net benefit, regardless of whose QALYs are saved (QALYs entail younger people being prioritized, as saving them produces more QALYs).

People also hold another important goal in the allocation of medical resources: equality in receiving such resources. People place great value on equality, to the point that they are willing to save fewer lives to achieve equal allocation of lifesaving resources (Ubel, Baron, & Asch, 2001). However, the preference for equity at the expense of efficiency is also volatile and subject to change depending on how the question is posed. For instance, in the choice between younger and older lives when not all lives can be saved, people usually prefer to save younger lives, demonstrating a preference for efficiency (Li, Vietri, Galvani, & Chapman, 2010), but such preferences fluctuate systematically. When people read descriptions on allocation outcomes (e.g., 500 20-year old people will be saved; 500 60-year old people will be saved), they prefer to save younger individuals; however, when they read general principles on how different lives should be prioritized under scarcity (e.g., “younger people should be valued more,” “older people should be valued more,” or “all lives should be valued equally regardless of age”), people demonstrate greater preference for equal allocation across young and old recipients (Li, 2012).

Another example in which preference for efficiency versus equality can be shifted with a fairly subtle change in description comes from a study by Colby, DeWitt, and Chapman (2015). Participants allocated 6 transplant kidneys to 12 potential recipients, 6 of whom had a high chance of transplant success. When the 12 recipients were presented in one group, participants tended to allocate the kidneys efficiently, giving them to the 6 recipients most likely to benefit from them. However, when the 12 recipients were presented in 2 groups of 6 each, participants tended to spread the kidneys across the 2 groups, even though this meant sacrificing efficiency.

Information Overload

All decisions entail a comparison and selection among multiple options, even if some options are not salient (e.g., choosing nothing, doing nothing). But what happens when there are a large number of options available, accompanied by a large amount of information about them? This is the situation many Americans face after the recent ratification of the Patient Protection and Affordable Care Act (ACA), which allows them to select from among dozens of healthcare plans. Providing too many healthcare plans can decrease decision quality. For example, Johnson, Hassin, Baker, Bajger, and Treuer (2013) presented participants with premium, co-pay, and deductible information for multiple health plans and found that participants frequently showed near-chance levels of performance in selecting the lowest cost plan because they gave too much weight to out-of-pocket expenses and deductibles. Although Johnson et al.'s participants made hypothetical choices, other researchers who analyzed data from actual health plan choices also found that consumers frequently chose dominated health plans—plans that were more expensive but provided no better benefits than alternative plans—because consumers focused too heavily on deductibles (Bhargava, Loewenstein, & Sydnor, 2015). Thus, choosing from a large choice set makes for a difficult psychological task, sometimes resulting in the decision maker violating normative principles such as dominance. Therefore, medical decisions can be improved by simplifying choice sets or providing smart defaults to decision makers.

Discounting Future Outcomes

Frequently, medical decisions involve delayed outcomes. For example, a calorie-dense diet and low levels of physical activity lead to increased risk of obesity and associated comorbidities such as diabetes and heart disease. However, these consequences are delayed, often by years or decades. Decision makers value delayed outcomes less than equivalent immediate outcomes; consequently, it is difficult for decision makers to forgo the immediate temptations of a piece of cheesecake or the TV episode instead of a trip to the gym. According to normative discounted utility theory, delayed outcomes should be discounted in a consistent manner, and thus people should not change their preferences between a larger later reward (e.g., the long-term benefit of weight loss) and a smaller sooner reward (e.g., the enjoyment of a piece of cheesecake) at different time points, that is, if you decided that the long-term benefit of weight loss is more important than the enjoyment of cheesecake that you can have soon, you should be able to stick to this decision at any given time. However, preference between these two types of rewards frequently changes with time: People often choose the larger later reward initially (such as on New Year's Day), when they are temporally distant from both rewards, but later fall for the temptation when it becomes available immediately (as the dessert tray rolls by). This inconsistent choice marks the typical pattern of self-control failure.

As a solution to this problem, decision makers will sometimes take advantage of the opportunity to precommit or bind themselves to the earlier preference for the larger later reward. For example, Schwartz et al. (2014) invited grocery store shoppers to lay their money on the line to precommit to buying more healthy foods such as fresh produce. The shoppers agreed to forfeit the discount they already received for buying healthy foods if they did not meet their healthy foods purchase goal. Indeed, these shoppers increased their healthy food purchases relative to a control group who merely indicated hypothetically whether they would precommit. Similarly, dieters who do not keep desserts in the house are taking advantage of a precommitment device, as are people who prepay for a gym membership in the hopes of motivating themselves to work out frequently to “get their money’s worth.” Another solution to the problem of discounted future outcomes is bundling temptation with a desired behavior, so one is allowed to indulge in a pleasure only when one engages in a necessary but unpleasant health behavior. For example, Milkman, Minson, and Volpp (2014) found that people visited the gym more often if they could listen to addictive audio books only when they were at the gym.

Strategic Behavior

Some medical decisions are made in a strategic setting where the choices made by an individual affect the outcomes of others while at the same time the decisions of others affect the outcomes experienced by the individual. Take vaccination as an example. Because of herd immunity, an individual’s vaccination protects not only herself from infection but also her social contacts, because she is now less likely to spread the virus to others. Consequently, if enough individuals in a population are vaccinated, the unvaccinated members are protected from infection. Thus, vulnerable individuals who cannot receive vaccination themselves (e.g., infants) can be cocooned in this fashion. Because of this positive externality from vaccination, an individual’s decision about whether to vaccinate may be affected by how many others in the population are vaccinating (Galvani, Reluga, & Chapman, 2007). This strategic interaction can be modeled using game theory (Chapman et al., 2012): individuals can “defect” by getting a free ride, that is, forgoing vaccination because they receive protection from others who vaccinated, or they can “cooperate,” that is, vaccinating in part to protect those around them.

Free riding may contribute to the anti-vaccination movement because those opposed to vaccination can forgo immunization without bearing much risk of contracting infectious disease, as long as the majority of the surrounding population is immunized. When critical numbers of those opposed to vaccination are clustered in one geographical region, however, herd immunity can dip below the threshold that is necessary to prevent the spread of disease, making the larger population vulnerable to an outbreak, such as the measles outbreak in a theme park in California in 2015. Other medical decisions that entail externalities include overuse of antibiotics, which does not affect the user negatively but contributes to the development of resistant strains of bacteria, and the over-ordering of expensive medical tests for which the physician does not pay but which will drive up healthcare costs and insurance premiums.

Numeracy

Normative models of decision making assume that decision makers will make informed decisions, provided that they are in possession of the relevant facts. However, decision makers vary in the capacity to comprehend and use information to approach choices. For example, numer-

acy, or the capacity to process numerical information, is essential in medical decision making, as such decisions frequently involve numbers (e.g., risk probabilities, efficacy information, cost, time). Numeracy levels among Americans are worrisome, with many people, even those who are highly educated, are particularly lacking in the ability to process percentages and fractions. These basic numerical competencies are critical in the medical context, such as when one needs to calculate medication dosage based on a child's body weight. A recent review (Reyna, Nelson, Han, & Dieckmann, 2009) concluded that patients who have low numeracy are more likely to misinterpret graphs containing medical information, more easily influenced by factors irrelevant to the decision at hand (such as how the information is presented), and more likely to overestimate small risks and have poorer health outcomes when the health condition requires a high degree of self-management.

Perhaps more disturbingly, medical professionals do not necessarily possess the level of numeracy that is required to correctly understand risk information. And the lack of capacity to comprehend risk information can incur serious consequences. For example, despite the commonly held belief that cancer screening saves lives, some cancer screening tests can produce a large number of false positives, encouraging overtreatment for nonprogressive cancer while yielding little or no life-saving benefit, and inflating the 5-year survival rate. Therefore, to evaluate the effectiveness of a screening test in saving lives, a reduced mortality rate should be used as the gold standard, instead of improved survival rates. Unfortunately, a study by Wegwarth, Schwartz, Woloshin, Gaissmaier, and Gigerenzer (2012) showed that about half of physicians do not share this knowledge and instead incorrectly believe that increased early detection constitutes evidence that such screening saves lives. This lack of numerical competency among physicians may have contributed to the ill-deserved enthusiasm toward cancer screening tests and the resulting financial and health toll of their overuse.

Healthy Nudges

Given the myriad ways in which actual medical decisions deviate from normative models of decision making, researchers and clinicians alike consider what can be done to improve medical decisions. Decision psychologists and behavioral economists have recently developed nudges (Thaler & Sunstein, 2008), or changes to the decision environment that facilitate selection of the healthy option, while maintaining freedom of choice. One well-known nudge is the default effect, or the tendency for people to stick with the option they will get automatically if they take no explicit action. For example, people prescheduled for a flu shot appointment (which they can cancel if they do not want it) are more likely to get vaccinated than are those who are not prescheduled (Chapman, Li, Colby, & Yoon, 2010). Although both groups have a choice to have a flu shot appointment or not, for the prescheduled group, the default is having a flu shot appointment, and for the comparison group the default is not having an appointment. Because most people tend to stick with their default status, and because having a flu shot appointment is a strong predictor of actually getting a flu shot, vaccination rates increased by 36% (from 33 to 45%) in the group with a default vaccination appointment relative to the group where an appointment has to be actively scheduled.

There are other types of nudges toward healthy behavior. For example, because people tend to conform their behavior to that of the group, giving people information about what others do will nudge them toward that same behavior. Social norms have been used to reduce caloric intake (by printing recommended calorie intake on menus, combined with menu calorie labeling; Roberto, Larsen, Agnew, Baik, & Brownell, 2010), to increase children's vegetable intake

at school lunch (by placing photographs of vegetables on wells of the lunch tray; Reicks, Redden, Mann, Mykerezi, & Vickers, 2012), and to encourage walking (by informing participants who wear pedometers how much others are walking; Chapman, Colby, Convery, & Coups, 2015). Another example of a healthy nudge is the position effect, in which food options displayed at prominent, easy-to-reach positions are chosen more frequently. Researchers have used the position effect to encourage healthy eating in cafeterias and restaurants (e.g., Rozin, Scott, & Dingley, 2011).

In summary, medical decision making includes not only decisions that are made in strictly medical settings but also decisions made in everyday life that impact health. Research on medical decision making can inform health professionals as well as the general public about the pitfalls in medical decisions, how to overcome them, and how to use decision biases to the advantage of people's health.

Author Biographies

Meng Li is an assistant professor in the Department of Health and Behavioral Sciences at the University of Colorado Denver. Her work, which has been featured on JAMA, Lance, Psychological Science, and other top journals, utilizes decision biases to “nudge” people toward healthy behavior, such as vaccination, healthy diet, and hand sanitizer use. Her more recent work explores policy issues, including resource allocation on money and health, price transparency in healthcare, and work–life balance policies.

Gretchen B. Chapman is a professor of psychology and a member of the Institute for Health and the Center for Cognitive Science at Rutgers University. Her research combines judgment and decision making with health psychology to examine preventive health behaviors such as vaccination. She conducts field studies to evaluate decision theoretic interventions designed to encourage health behavior and laboratory research to examine basic processes in decisions under uncertainty, strategic interactions, and allocation of scarce resources.

References

- Bhargava, S., Loewenstein, G., & Sydnor, J.R. (2015). Do individuals make sensible health insurance decisions? Evidence from a menu with dominated options. *NBER Working Paper No. w21160*. Reterived from <http://ssrn.com/abstract=2604841>
- Chapman, G. B., Colby, H., Convery, K., & Coups, E. J. (2015). Goals and social comparisons promote walking behavior. *Medical Decision Making*, 36(4), 472–478. doi:10.1177/0272989X15592156
- Chapman, G. B., Li, M., Colby, H., & Yoon, H. (2010). Opting in versus opting out of influenza vaccination. *The Journal of the American Medical Association*, 304(1), 43–44.
- Chapman, G. B., Li, M., Vietri, J., Ibuka, Y., Thomas, D., Yoon, H., & Galvani, a. P. (2012). Using game theory to examine incentives in influenza vaccination behavior. *Psychological Science*, 23, 1008–1015. doi:10.1177/0956797612437606
- Colby, H., DeWitt, J., & Chapman, G. B. (2015). Grouping promotes equality: The effect of recipient grouping on allocation of limited medical resources. *Psychological Science*, 26, 1084–1089. doi:10.1177/0956797615583978
- Galvani, A. P., Reluga, T. C., & Chapman, G. B. (2007). Long-standing influenza vaccination policy is in accord with individual self-interest but not with the utilitarian optimum. *Proceedings of the National Academy of Sciences of the United States of America*, 104, 5692–5697. doi:10.1073/pnas.0606774104

- Johnson, E. J., Hassin, R., Baker, T., Baiger, A. T., & Treuer, G. (2013). Can consumers make affordable care affordable? The value of choice architecture. *PLoS One*, 8(12). doi:10.1371/journal.pone.0081521
- Li Meng (2012). How do people value life? (Dissertation). Retrieved from <https://rucore.libraries.rutgers.edu/rutgers-lib/37432/>
- Li, M., & Chapman, G. B. (2009). "100% of anything looks good": The appeal of one hundred percent. *Psychonomic Bulletin & Review*, 16(1), 156–162. doi:10.3758/PBR.16.1.156
- Li, M., & Chapman, G. B. (2013). A big fish or a small pond? Framing effects in percentages. *Organizational Behavior and Human Decision Processes*, 122(2), 190–199. doi:10.1016/j.obhdp.2013.07.003
- Li, M., Vietri, J., Galvani, A. P., & Chapman, G. B. (2010). How do people value life? *Psychological Science*, 21, 163–167. doi:10.1177/0956797609357707
- Milkman, K. L., Minson, J. a., & Volpp, K. G. M. (2014). Holding the hunger games hostage at the gym: An evaluation of temptation bundling. *Management Science*, 60, 283–299. doi:10.1287/mnsc.2013.1784
- Reicks, M., Redden, J. P., Mann, T., Mykerezi, E., & Vickers, Z. (2012). Photographs in lunch tray compartments and vegetable consumption among children in elementary school cafeterias. *JAMA*, 307(8), 784–785. doi:10.1001/jama.2012.170
- Reyna, V. F., Nelson, W. L., Han, P. K., & Dieckmann, N. F. (2009). How numeracy influences risk comprehension and medical decision making. *Psychological Bulletin*, 135(6), 943–973.
- Roberto, C. A., Larsen, P. D., Agnew, H., Baik, J., & Brownell, K. D. (2010). Evaluating the impact of menu labeling on food choices and intake. *American Journal of Public Health*, 100(2), 312–318. doi:10.2105/AJPH.2009.160226
- Rozin, P., Scott, S., & Dingley, M. (2011). Nudge to nobesity I: Minor changes in accessibility decrease food intake. *Judgement and Decision Making*, 6(4), 323–332. Retrieved from <http://www.sjdm.org/journal/11/11213/jdm11213.pdf>
- Schwartz, J., Mochon, D., Wyper, L., Maroba, J., Patel, D., & Ariely, D. (2014). Healthier by precommitment. *Psychological Science*, 25(January), 538–546. doi:10.1177/0956797613510950
- Thaler, R., & Sunstein, C. (2008). *Nudge: Improving decisions about health, wealth, and happiness*. New Haven, CT: Yale University Press.
- Ubel, P. A., Baron, J., & Asch, D. A. (2001). Preference for equity as a framing effect. *Medical Decision Making*, 21(3), 180–189. doi:10.1177/0272989X0102100303
- Viscusi, W. K., Magat, W. A., & Huber, J. (1987). An investigation of the rationality of consumer valuation of multiple health risks. *RAND Journal of Economics*, 18(4), 465–479.
- Wegwarth, O., Schwartz, L. M., Woloshin, S., Gaissmaier, W., & Gigerenzer, G. (2012). Do physicians understand cancer screening statistics? A national survey of primary care physicians in the United States. *Annals of Internal Medicine*, 156(5), 340–349. doi:10.7326/0003-4819-156-5-201203060-00005

Suggested Reading

- Betsch, C., Böhm, R., & Chapman, G. B. (2015). Interventions to counter vaccine hesitancy—Using behavioral insights to increase vaccination policy effectiveness. *Policy Insights From the Behavioral and Brain Sciences*, 2(1), 61–73.
- Li, M., & Chapman, G. B. (2013). Nudge to health: Harnessing decision research to promote health behavior. *Social Psychology Compass*, 7(3), 187–198.
- Milkman, K. L., Rogers, T., & Bazerman, M. H. (2008). Harnessing our inner angels and demons: What we have learned about want/should conflicts and how that knowledge can help us redect short-sighted decision making. *Perspectives on Psychological Science*, 3(4), 324–338.
- Reyna, V. F., Nelson, W. L., Han, P. K., & Dieckmann, N. F. (2009). How numeracy influences risk comprehension and medical decision making. *Psychological Bulletin*, 135(6), 943–973.
- Thaler, R., & Sunstein, C. (2008). *Nudge: Improving decisions about health, wealth, and happiness*. New Haven, CT: Yale University Press.

Message Framing

Scout N. McCully and John A. Updegraff

Department of Psychological Sciences, Kent State University, Kent, OH, USA

Doctors, health psychologists, and medical professionals implore patients to adopt and maintain health behaviors, but effective persuasion often proves difficult. Message framing offers a relatively simple and versatile way to tailor a health message to increase its effectiveness. Message framing is built upon the idea that all decisions regarding health behaviors carry consequences; generally, engaging in a health behavior will yield more positive consequences than negative consequences, while failing to do so will yield more negative outcomes than positive outcomes. As such, messages about health behaviors can be framed to emphasize what can be gained by performing the behavior (gain framed) or what can be lost by not performing the behavior (loss framed). For example, a message about the flu shot could read, “If you get a flu shot, you will reduce your risk of getting the flu this year” (gain framed), or it could read, “If you do not get a flu shot, you will be at risk of getting the flu this year” (loss framed). The gain-framed message emphasizes the positive consequences of getting a flu shot, while the loss-framed message highlights the negative consequences associated with not getting a flu shot.

Message framing developed as an extension of prospect theory (Tversky & Kahneman, 1981), which suggests that people will make different choices when outcomes are framed as gains versus losses. People are more likely to make risky decisions when faced with a loss, as they would rather gamble for the possibility of no losses (risking greater losses along the way) than accept certain loss. Alternatively, people are risk averse when contemplating a gain, as they would rather maintain a certain gain—even a small gain—than risk losing it all. Rothman and Salovey (1997) extended prospect theory to health behavior messages, proposing that gain- and loss-framed messages would differentially affect behavior depending on the behavior the message endorsed.

After years of examining message framing across various behaviors, people, and situations, researchers have found no general advantage of one frame over the other (Gallagher & Updegraff, 2012). However, research suggests that in specific circumstances, gain-framed messages produce greater behavior change than loss-framed messages, while in other circumstances, loss-framed messages prevail. Thus, message framing research has focused on understanding the moderators or conditions that make either gain- or loss-framed messages more effective in producing behavior change.

Moderators of Message Framing Effects

Moderators of message framing effects can be organized into three categories: behavior-related moderators, person-related moderators, and context-related moderators (see Table 1 for a summary). In this entry, we use the term *behavior-related moderator* to refer to a characteristic of the target health behavior that affects the relative persuasiveness of a gain- or loss-framed message. A *person-related moderator* is a state or trait characteristic of the target individual or audience member that influences the relative effectiveness of a particular frame. A *context-related moderator* refers to a characteristic of the situation in which the message is presented that changes the effect of message frames.

Behavior-Related Moderators

The most influential and widely studied moderator of framing effects is the behavior-related moderator of prevention versus detection behaviors. As mentioned above, prospect theory (Tversky & Kahneman, 1981) proposes that when faced with a loss, people are more likely to make risky decisions, but when faced with a gain, people are risk averse. Rothman and Salovey (1997) theorized that illness detection behaviors (e.g., mammograms, cancer screenings) carry an element of risk because such tests may reveal a health threat. Alternatively, behaviors focused on promotion of health or prevention of illness (e.g., physical activity, sunscreen use) carry little risk because they generally support and improve health. Thus, loss-framed messages should be more effective at promoting high-risk detection behaviors, whereas gain-framed messages should be more effective at promoting low-risk prevention behaviors.

After two decades of research on framing effects in prevention and detection behaviors, a meta-analysis concluded that indeed gain-framed messages tended to be more effective than loss-framed messages in promoting prevention behaviors, but loss-framed messages were no more effective than gain-framed messages in promoting detection behaviors (Gallagher & Updegraff, 2012). For prevention behaviors, gain-framed messages produced significantly higher rates of behavior in the domains of smoking, skin cancer prevention, and physical activity. Effects of message framing were weak for safe sex, diet, oral health, and vaccination, but in no case was a loss-framed message significantly more effective than a gain-framed message in prevention behaviors. Although the effect size of the gain-frame advantage ($r = .083$) was

Table 1 Recommendations for framing health messages.

<i>Moderator</i>	<i>Recommended message frame</i>	
	<i>Gain frame</i>	<i>Loss frame</i>
Behavior-related moderators		
Prevention vs. detection	Prevention	<i>n/a</i>
Person-related moderators		
Motivational orientation	Approach oriented	Avoidance oriented
Culture	Individualistic	Collectivistic
Perceived susceptibility	Low susceptibility	High susceptibility
Self-efficacy	<i>n/a</i>	High self-efficacy
Context-related moderators		
Autonomy vs. heteronomy	Prime autonomy	Prime heteronomy
Color	<i>n/a</i>	Red

relatively weak, Gallagher and Updegraff (2012) suggest that given the complexity of health behavior determinants, even a relatively weak effect may work additively with other factors to produce meaningful changes in behavior. In contrast to prevention behaviors, detection behaviors were not significantly affected by message framing. However, in examining specific domains of detection behaviors, the meta-analysis found that loss-framed messages were marginally more effective in promoting breast cancer detection behaviors than gain-framed messages.

Behavior-related moderators provide high-level recommendations for framing, particularly useful when little or no information about the target audience is available. A notable example would be a message created for mass production (e.g., public service announcements), which could be made most effective by framing it according to the behavior's inherent risk. Based on Gallagher and Updegraff's (2012) findings, mass-media messages promoting preventive health behaviors (e.g., physical activity, nutrition, sunscreen use, condom use, oral healthcare) should emphasize the positive outcomes that can be gained by adopting and maintaining these behaviors. When encouraging detection behaviors, healthcare professionals can rely on person- and context-related moderators to help determine the most effective message frame.

Person-Related Moderators

Where behavior-related moderators offer broad recommendations for framing, person-related moderators allow healthcare professionals to tailor framed messages to a patient when characteristics of that person are known. A number of person-related variables have been found to moderate the effect of message frame on health behaviors including a person's motivational orientation, culture, perceived susceptibility, and self-efficacy.

Motivational orientation refers to the degree that a person is predominantly motivated by cues of reward (approach oriented) versus cues of punishment or threat (avoidance oriented). Because gain-framed messages emphasize the reward to be gained by performing a behavior, researchers theorized that gain-framed messages would be more congruent and thus more persuasive for approach-oriented individuals. Alternatively, loss-framed messages emphasize the punishment or negative consequences of not adopting a behavior and thus should be a better match for avoidance-oriented individuals. Indeed, research indicates that approach-oriented people are more responsive to gain-framed messages, whereas avoidance-oriented people are more responsive to loss-framed messages. For example, in the domain of oral health behavior, participants who were primarily approach oriented flossed more after reading a gain-framed article, and participants who were primarily avoidance oriented flossed more after reading a loss-framed article (Sherman, Mann, & Updegraff, 2006). Thus, a clinician may assess motivational orientation using a short survey and then maximize a message's effectiveness by framing it to be congruent with the patient's predominant orientation.

In addition to moderators borne from individual differences, cultural difference factors may also play a role in the relative effectiveness of gain- and loss-framed messages. In a study assessing the moderating influence of culture on flossing behavior, researchers found that people with low exposure to US culture, measured by proportion of life spent in United States and whether parents were born in the United States, flossed more after seeing a loss-framed message compared with a gain-framed message (Brick et al., 2015). While culturally congruent messages resulted in greater flossing, culturally incongruent messages led to the same levels of flossing as seeing no message.

Why might this be? Researchers have found systematic differences in motivational orientation across cultures, with collectivistic East Asian and Latino cultures tending to be more

avoidance oriented and individualistic White British and American cultures tending to be more approach oriented. It is possible that because of these cultural orientation propensities, loss-framed messages are more effective among people from (or primarily exposed to) collectivistic cultures, whereas gain-framed messages are more effective among people from (or primarily exposed to) individualistic cultures. Indeed, support for this explanation comes from a study that examined differences in how White and East Asian individuals living in Britain responded to framed messages promoting oral health (Uskul, Sherman, & Fitzgibbon, 2009). White British individuals were more persuaded by a gain-framed message, whereas East Asian individuals were more persuaded by a loss-framed message. Clinically, cultural background can be measured with simple demographic questions often obtained during intake, and as such offers a simple and practical way for health professionals to tailor framed messages to patients, and also may suggest ways to address cultural and ethnic disparities in health.

Perceived susceptibility to a health condition has been studied as another person-related moderator of framing effects, particularly in detection behaviors. Although detection behaviors were originally proposed as inherently risky, people's perceptions of the risk of these behaviors likely fall along a spectrum, with some perceiving very little risk and others perceiving great risk. Specifically, people who perceive themselves to be more susceptible to a disease may be more likely to perceive the detection behavior as risky. Indeed, research on mammography testing indicates that among those with high perceived susceptibility, loss-framed messages are more effective than gain-framed messages but that message frame does not affect behavior when perceived susceptibility is low (Gallagher, Updegraff, Rothman, & Sims, 2011). In the context of oral health (Updegraff, Brick, Emanuel, Mintzer, & Sherman, 2015), moderation by perceived susceptibility was also observed: a gain-framed message was more effective for those who perceived low susceptibility to oral health problems, and a loss-framed message was somewhat more effective for those who perceived high susceptibility.

Finally, self-efficacy has also been proposed as a moderator of message frame, such that loss-framed messages appear to be more effective for those with high confidence in their ability to perform a behavior. However, this finding is currently limited to studies of self-examinations and smoking cessation; similar studies have failed to replicate this moderation in the context of other behaviors such as immunization (Covey, 2014). Although person-related moderators may be difficult for clinicians to apply because they require pre-assessment and tailoring, they can offer valuable framing guidance when behavior-related cues are unclear.

Context-Related Moderators

In addition to behavior- and person-related moderators, context-related moderators may influence the relative effectiveness of gain- and loss-framed messages. Theoretically, context-related moderators could be manipulated within a persuasion situation to increase the effectiveness of a framed message, unlike relatively stable factors like behavior- and person-related moderators. One example of a context-related moderator is the role of perceived autonomy versus heteronomy in health decision making. Autonomy refers to the experience of engaging in actions based on one's personal interests and values, whereas heteronomy refers to engaging in actions based on some pressure or coercion from other people. Pavey and Churchill (2014) found that after priming autonomy (i.e., having people generate sentences with words like freedom, choice, decision), gain-framed message produced greater behavior change than loss-framed messages, but after priming heteronomy (i.e., having people generate sentences with words like pressure, control, must), loss-framed messages were more effective.

Additionally, the use of colors may moderate framing effects. In particular, the use of the color red—which often acts as a cue of threat, due to its association with blood and danger—has been shown to increase the persuasiveness of loss-framed messages (Gerend & Sias, 2009). The domain of context-related moderators requires further study, but it offers the unique benefit of allowing researchers and clinicians to amplify the strength of a framed message by modifying the context in which it is presented. For example, if the target behavior is prevention focused, and the target audience is primarily approach oriented, the clinician could choose a gain-framed message and theoretically amplify it by emphasizing the individual's autonomy and freedom of choice. However, researchers have only begun to investigate the role of context-related moderators, and additional empirical work is needed to replicate and extend these findings.

Mediators of Message Framing Effects

Why do gain- or loss-framed messages have a differential impact on persuasion and behavior? While much is known about the moderators of framing effects, much less is known about the psychological mechanisms—that is, mediators—that account for these effects. Indeed, there may be many processes that make a gain- or loss-framed message more effective, including conscious cognitive processes as well as more implicit or emotional processes. Most of the research that has looked at mediators of framing effects has focused on conscious cognitive processes, such as changes in consciously articulated attitudes or intentions toward the advocated health behavior, but many studies have failed to show that these factors consistently explain effects on behavior (see Gallagher & Updegraff, 2012 for review). It is possible, then, that more implicit or emotional reactions may explain the effects of framing on behavior.

However, to date, two mediators seem to have the greatest potential to explain the influences of framed messages on behavior: elaboration and the subjective sense of “feeling right.” Specifically, research has found that people tend to elaborate on a message more—that is, they process the message at a greater depth—when the message frame is congruent with their motivational orientation. For example, people receiving congruent messages were better able to discern strong and weak arguments about the importance of flossing than people who received incongruent messages (Updegraff, Sherman, Luyster, & Mann, 2007). In addition, congruently framed messages may be more likely to lead a person to have a subjective sense of “feeling right” about their reactions to a message than incongruently framed messages (Cesario, Higgins, & Scholer, 2008).

Interestingly, both the elaboration and the “feeling right” accounts highlight the importance of basing a framed health messages on strong, convincing information. If recipients are more likely to think deeply about and “feel right” about their reactions to a congruently framed health message, then incorporating unconvincing or flimsy information into the message may have the unintended effect of *decreasing* the effectiveness of the framing. Thus, the mediation of framing effects is complex and requires further study but is an important direction for future research.

Applying Message Framing

Message framing offers a relatively simple and versatile way for clinicians and health professionals to present health messages in a maximally persuasive way. Specifically, the moderators

discussed above offer a number of ways to decide how to determine the frame of the message. If a message is to be mass-produced, or if no information about the recipient is known, the message crafter should consider whether the behavior is a detection or prevention behavior and frame the message accordingly; that is, if the behavior is a prevention or promotion behavior, the clinician should gain-frame the message. However, if more information is known about the audience, the clinician may use some of these characteristics to determine an appropriate message frame. A recipient who is avoidance oriented or perceives high susceptibility may be more strongly persuaded by a loss-framed message. Alternatively, a person from an individualistic culture or with low perceived susceptibility may be better persuaded by a gain-framed message.

Under certain circumstances, having information about a number of moderators can pose difficult clinical decisions if those pieces of information propose conflicting message frames. For example, if the behavior is a detection behavior, and the target individual perceives high susceptibility, but the individual is also approach oriented and from an individualistic culture, which frame should be used? The high perceived susceptibility suggests a loss-framed message, but the motivational and cultural orientations suggest a gain-framed message. Clearly, conflicting information poses challenges for the clinician hoping to put this information to good use. Additional research that examines multiple moderators in concert is needed to determine relative strength and weights of these various moderators so that clinicians can make better informed decisions when selecting message frames.

Message framing is a particularly versatile application, as it can be applied through any number of clinically relevant media, from brochures and signs to videos to live doctor–patient conversations. With a simple modification to the way in which an action (or lack of action) is paired with a consequence, healthcare professionals may be able to increase the effectiveness of their communications and help patients make healthier decisions.

Author Biographies

Scout N. McCully is a doctoral student in psychological sciences at Kent State University. Her work seeks to improve people's success in adopting and maintaining health behaviors, with a focus on simple, scalable, theory-driven interventions.

John A. Updegraff is professor of psychological sciences at Kent State University. His research examines the effectiveness of tailoring persuasive health messages to match the ways in which people think about health behaviors and their environments in general. A central goal of his work is to develop theoretical frameworks and strategies for communicating health information in ways that are personally relevant and effective in promoting long-term behavior change.

References

- Brick, C., McCully, S. N., Updegraff, J. A., Ehret, P. J., Areguin, M. A., & Sherman, D. K. (2015). Impact of cultural exposure and message framing on oral health behavior: Exploring the role of message memory. *Medical Decision Making*, 6(7), 834–843. doi:10.1177/0272989X15570114
- Cesario, J., Higgins, E. T., & Scholer, A. A. (2008). Regulatory fit and persuasion: Basic principles and remaining questions. *Social and Personality Psychology Compass*, 2(1), 444–463. doi:10.1111/j.1751-9004.2007.00055.x

- Covey, J. (2014). The role of dispositional factors in moderating message framing effects. *Health Psychology, 33*(1), 52–65. doi:10.1037/a0029305
- Gallagher, K. M., & Updegraff, J. A. (2012). Health message framing effects on attitudes, intentions, and behavior: A meta-analytic review. *Annals of Behavioral Medicine, 43*(1), 101–116. doi:10.1007/s12160-011-9308-7
- Gallagher, K. M., Updegraff, J. A., Rothman, A. J., & Sims, L. (2011). Perceived susceptibility to breast cancer moderates the effect of gain- and loss-framed messages on use of screening mammography. *Health Psychology, 30*(2), 145–152. doi:10.1037/a0022264
- Gerend, M. A., & Sias, T. (2009). Message framing and color priming: How subtle threat cues affect persuasion. *Journal of Experimental Social Psychology, 45*(4), 999–1002. doi:10.1016/j.jesp.2009.04.002
- Pavey, L., & Churchill, S. (2014). Promoting the avoidance of high-calorie snacks: Priming autonomy moderates message framing effects. *PLoS One, 9*(7). doi:10.1371/journal.pone.0103892
- Rothman, A. J., & Salovey, P. (1997). Shaping perceptions to motivate healthy behavior: The role of message framing. *Psychological Bulletin, 121*(1), 3–19. doi:10.1037/0033-2909.121.1.3
- Sherman, D. K., Mann, T., & Updegraff, J. A. (2006). Approach/avoidance motivation, message framing, and health behavior: Understanding the congruency effect. *Motivation and Emotion, 30*, 165–169. doi:10.1007/s11031-006-9001-5
- Tversky, A., & Kahneman, D. (1981). The framing of decisions and the psychology of choice. *Science, 211*(4481), 453–458.
- Updegraff, J. A., Brick, C., Emanuel, A. S., Mintzer, R. E., & Sherman, D. K. (2015). Message framing for health: Moderation by perceived susceptibility and motivational orientation in a diverse sample of Americans. *Health Psychology, 34*(1), 20–29. doi:10.1037/hea0000101
- Updegraff, J. A., Sherman, D. K., Luyster, F. S., & Mann, T. L. (2007). The effects of message quality and congruency on perceptions of tailored health communications. *Journal of Experimental Social Psychology, 43*(2), 249–257. doi:10.1016/j.jesp.2006.01.007
- Uskul, A. K., Sherman, D. K., & Fitzgibbon, J. (2009). The cultural congruency effect: Culture, regulatory focus, and the effectiveness of gain- vs. loss-framed health messages. *Journal of Experimental Social Psychology, 45*(3), 535–541. doi:10.1016/j.jesp.2008.12.005

Suggested Reading

- Gallagher, K. M., & Updegraff, J. A. (2012). Health message framing effects on attitudes, intentions, and behavior: A meta-analytic review. *Annals of Behavioral Medicine, 43*, 101–116.
- Rothman, A. J., Bartels, R. D., Wlaschin, J., & Salovey, P. (2006). The strategic use of gain-and loss-framed messages to promote healthy behavior: How theory can inform practice. *Journal of Communication, 56*, S202–S220.
- Rothman, A. J., & Salovey, P. (1997). Shaping perceptions to motivate healthy behavior: The role of message framing. *Psychological Bulletin, 121*, 3–19.

Message Tailoring

Mia Liza A. Lustria and Juliann Cortese

School of Information, Florida State University, Tallahassee, FL, USA

Tailoring as a Health Communication Strategy

Much of the world's disease burden is associated with a few modifiable health behaviors like diet and physical activity and risky or unhealthy behaviors such as smoking, alcohol abuse, unsafe sex, use of firearms, and drug use. Most people recognize risks associated with these unhealthy behaviors and the potential benefits of adopting healthy behaviors in lowering their risk for chronic illness. Yet many continue to make unhealthy lifestyle choices even when they experience undesired consequences for their actions (or inaction) or even if they have a genetic predisposition toward a particular chronic disease (King, Mainous, Carnemolla, & Everett, 2009).

Health psychologists recognize that a number of factors can shape health behaviors and that these factors vary from one individual to another. These factors are driven by reciprocal influences of one's experiences, beliefs, values and attitudes, motives and expectations, environment and circumstances, personality traits, mental and emotional states, patterns of behavior, and habits, among others. Health messages primarily designed to raise awareness without considering the complexity of behavior change are deemed to have limited impact on individual actions and decision making. It is for this reason that interpersonal communication continues to be the ideal channel for promoting individual behavior change because of the ability to adjust communication on the fly and to deliver personalized recommendations. Unfortunately, the reach and accessibility of health counselors adequately trained in delivering evidence-based personalized recommendations is limited. Message tailoring, a health communication strategy for disseminating theoretically informed individualized recommendations, seeks to address this need to deliver more efficacious health behavior change strategies on a mass scale.

The Tailoring Process

Message tailoring is defined as a “multidimensional communication strategy that involves developing individualized messages based on a pre-assessment of key individual-difference

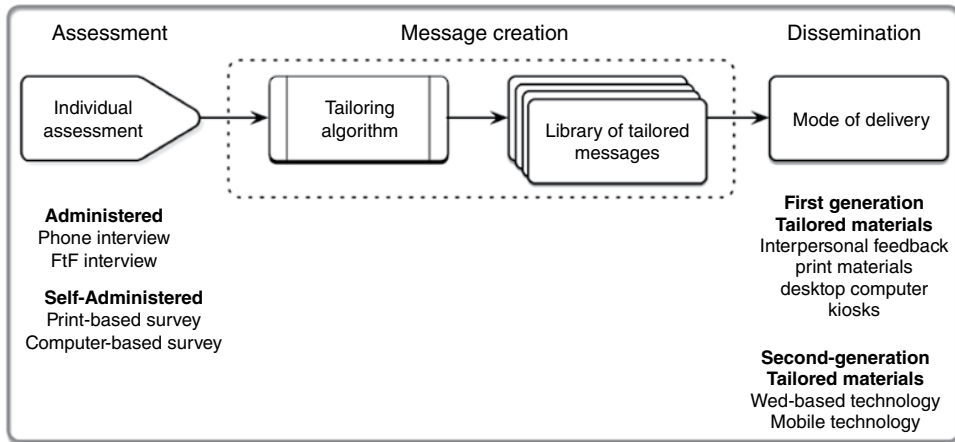


Figure 1 A basic message tailoring system.

variables or characteristics linked to the underlying model of behavior change” (Lustria et al., 2013, p. 1040). It refers to various message design strategies for creating health messages that resonate at an individual level by using knowledge about a person’s unique needs and preferences (Kreuter & Wray, 2003). Information about the individual is typically assessed through a survey of relevant factors (e.g., personal, psychosocial) and theoretically informed determinants of the targeted health behavior. This data is then evaluated by an expert system that uses tailoring algorithms to prioritize content and recommendations based on user input. At the most basic level, this process involves selecting messages most relevant for the individual from a database of tailored content. A typical tailoring system includes (a) a way to collect information about the individual (e.g., paper-based, telephone-based, or computer-based assessment), (b) a system to analyze responses (usually via computer algorithms), (c) an extensive message library, and (d) a way to disseminate the tailored messages (e.g., via print media, electronic, or web-based technologies; Figure 1).

As a communication strategy, message tailoring takes into account the heterogeneity of factors that determine an individual’s health behaviors and the variety of pathways to success. It is not to be mistaken for simple *personalization* or the inclusion of personal identifiers, the main goal of which is to draw attention to the health message (e.g., names). It is also different from *targeted messaging* or creating messages that might resonate with specific groups of people that share common characteristics (e.g., teenage mothers, dementia patients, breast cancer survivors). Rather, in *tailored messaging*, individual-level assessment enables the creation and delivery of highly personalized recommendations and plans for modifying simple to complex health behaviors based on an analysis of individual drivers for (and barriers to) change.

In contrast to generic messages, tailored messages are designed to directly address factors relevant to the individual and promote recommendations that take the individual’s context into consideration. The following is an example of a generic health message aimed at preventing weight gain found on typical websites like the Centers for Disease Control and Prevention (adapted from Centers for Disease Control and Prevention, 2015):

If you’re currently at a healthy weight, you’re already one step ahead of the game. To stay at a healthy weight, it’s worth doing a little planning now. Or maybe you are overweight but aren’t ready to lose weight yet. If this is the case, preventing further weight gain is a worthy goal. As people age, their body composition gradually shifts — the proportion of muscle decreases and the

proportion of fat increases. This shift slows their metabolism, making it easier to gain weight. In addition, some people become less physically active as they get older, increasing the risk of weight gain. The good news is that choosing a lifestyle that includes good eating habits and daily physical activity can prevent weight gain. To learn more...

In contrast, the following is an example of how a tailored health message differs from the generic message just provided:

Hi Pam! **Based on your current age, weight and height, you are slightly at risk** for obesity. This extra weight can but stress on your bones, joints, and organs, which may partly explain **your lethargy and difficulty in sustaining your daily activities**. Too much body fat also raises your **blood pressure** and cholesterol - this can be very dangerous for people like you **who have a family history of heart disease and stroke**. Here's the good news – you can achieve a healthy weight by adopting good eating habits and daily physical activity.

Being a busy career woman and mom, we understand that this is easier said than done. So we've created **a few simple steps for you to reach your goal**. We'll start by **recommending easy-to-prepare meals to help lower your daily caloric intake to ____** and **a few daily exercises that you can do in 10 minutes or less each day**. Start here...

Message Tailoring Works

A wide evidence base promotes the use of tailored messaging over one-size-fits-all generic messaging. Compared with non-tailored messages, tailored information commands greater attention and is processed more intently, recalled more readily, and perceived as more personally relevant and more positively by health consumers (Kreuter, Strecher, & Glassman, 1999). More importantly, several studies and meta-analyses provide empirical evidence for the benefits of tailored versus non-tailored interventions across a wide variety of health outcomes such as physical fitness, nutrition, mental health, immunization, early detection or screening, sexual health, alcoholism, and chronic disease (e.g., Chodosh et al., 2005; Davies, Morriss, & Glazebrook, 2014; Kessels, Ruiters, Brug, & Jansma, 2011).

Overall, various meta-analyses have pointed to small- and medium-size effects of tailoring on behavior change. Noar, Benac, and Harris (2007) conducted a meta-analytic review of 57 tailored print behavior change interventions and found a mean effect of $r = .074$ of tailoring on health behaviors. The variables that were found to significantly moderate the effect included (a) type of comparison condition, (b) health behavior, (c) type of participant population (both type of recruitment and country of sample), (d) type of print material, (e) number of intervention contacts, (f) length of follow-up, (g) number and type of theoretical concepts tailored on, and (h) whether demographics and/or behavior were tailored on.

Krebs, Prochaska, and Rossi (2010) also conducted a meta-analysis to evaluate the mean effect of 88 computer-tailored interventions focusing on smoking cessation, physical activity, eating a healthy diet, and receiving regular mammograms. They found clinically and statistically significant overall effects across each of the four behaviors. Effect sizes decreased after intervention completion, and dynamically tailored interventions were found to have increased efficacy over time as compared with those based on one assessment only.

Tailored interventions can range from simple, tailored risk assessments or tailored health messaging to more complex and highly individualized health programs, treatment plans, or recommended actions. The most basic tailored interventions involve risk assessment followed by short motivational interventions focused on promoting the targeted health behavior. These

brief tailored interventions typically involve single assessments at baseline, after which participants either immediately receive tailored advice via a webpage or print out or are scheduled to receive tailored messages via email or postal mail. For health interventions targeting more complex lifestyle changes or behavior modification, tailoring may be done iteratively or delivered in modules and may be enhanced through interactive features such as goal setting, skills building, tracking and monitoring, and expert and social support tools to help participants achieve their goals (Lustria, Cortese, Noar, & Glueckauf, 2009).

In one example, Woolford, Clark, Strecher, and Resnicow (2010) used mobile phone texting to tailor healthy behavior messages to obese adolescents. Baseline measurement was used to assess behaviors, and then text messages were sent automatically through a computer application that chose from a series of appropriate messages in a library of tailored content. Participants were enrolled in a weight management program, and the tailored reminders helped keep them on track with their weight-loss goals.

Lustria et al. (2013) conducted a meta-analysis of 40 randomized controlled trials and quasi-experimental studies comparing tailored versus non-tailored web-based interventions. The findings indicated that web-based tailored interventions lead to significantly greater improvement in health outcomes as compared with control conditions both at posttest and at follow-up. The researchers concluded that web-based interventions benefited from the inclusion of several different modalities that added an interactive element to interventions, such as audio, video, discussion, and chat, which is ideal for improving user engagement and supporting patients' self-management and skills building.

How Does Message Tailoring Work?

Hawkins, Kreuter, Resnicow, Fishbein and Dijkstra (2008) propose that tailoring improves the efficacy of health communication by enhancing (a) the cognitive preconditions for message processing or acceptance and (b) message impact by selectively modifying the behavioral determinants of desired health outcomes.

Petty and Cacioppo's elaboration likelihood model (ELM) lays the theoretical groundwork for how tailoring can enhance persuasion. ELM is a useful way of looking at how attitudes can be influenced, shaped, or changed as a result of exposure to mass-mediated persuasive messages. According to this theory, attitudes may be influenced as a result of high elaboration, one's thoughtful consideration of the merits of a persuasive message (*central route processing*), or a simple cue in the persuasion context (*peripheral route processing*; Petty & Cacioppo, 1986). When people are motivated (e.g., when the topic is interesting or perceived to be personally relevant) and have the ability to process messages actively and more thoughtfully (e.g., have prior knowledge, no distractions, message is comprehensible), they are more likely to scrutinize the information and make decisions based on its merits.

However, not all situations are conducive for thoughtful reflection, as in the case of a brief message communicated via a televised public service announcement. In situations of low elaboration like this, persuasion may still occur through the influence of heuristic cues like the appearance of the source or the source's perceived trustworthiness. Attitudes formed via the central route are thought to be more enduring, more resistant to counter-persuasion, and more predictive of behavior (Petty, Briñol, & Priester, 2009), and thus messages designed to induce higher elaboration are ideal for persuasion. While the valence of a persuasion attempt can be influenced by a number of factors, it is clear that an individual's motivation and ability to process the message are critical to the outcome. Much of the research on this topic has shown a significant

relationship between tailoring and increased attention and perceived personal relevance. Petty et al. (2009, p. 145) note that tailoring “can influence attitudes by serving as a peripheral cue when elaboration is low, by biasing thoughts when elaboration is high and by enhancing the amount of information processing when elaboration is moderate.”

The efficacy of tailored interventions is also bolstered when message tailoring is guided by research and by health behavior theories and models (Webb, Joseph, Yardley, & Michie, 2010). Theory and research can inform tailoring in a number of ways by helping prioritize relevant constructs for tailoring (e.g., perceived risk, attitudes, beliefs) and by informing the selection of specific messaging strategies and behavior change techniques (e.g., stage matching, timing, delivery mode) most effective for particular population groups. The most common theories used to guide message tailoring are described below.

Prochaska's transtheoretical model (TTM) has been used extensively and successfully in smoking cessation programs tailored based on patients' readiness to quit smoking. The TTM suggests that a patient's responsiveness to a behavioral intervention is influenced by where they are in the “stages of change”: precontemplation, contemplation, preparation, action, maintenance, and termination (J. O. Prochaska & DiClemente, 1983). The model is suited particularly well for guiding tailoring in health interventions related to areas to which people may exhibit various degrees of commitment (i.e., physical activity, nutrition, and smoking cessation; J. M. Prochaska & Prochaska, 2014). Certainly, stage-matched tailoring has proven to be effective in a number of interventions related to smoking, condom use, cancer screening, and hypertension control, among others (Stanczyk et al., 2014). Knowledge about an individual's readiness for change can be very useful in crafting and scheduling the delivery of motivational messages and reminders with the desired goal of moving someone onto latter stages or effecting more lasting change.

Another health communication theory often used to guide message tailoring is the health belief model. The health belief model is a predictive model that examines the likelihood that an individual will engage in a health-promoting behavior (Rosenstock, 1974). The theory suggests that likelihood of engagement in a health-conscious behavior can be predicted by examining an individual's perceived susceptibility, seriousness of the health concern, benefits versus barriers of the healthy choice, perceived threat, self-efficacy, and cues to action. Because all of these factors have the opportunity to affect change, presenting the individual with tailored message focused in any of these areas can result in a choice to engage in a health-promoting behavior.

Often one of the easiest ways to influence behavior is to present a behavior that can be imitated or modeled. This approach is the focus of social cognitive theory, which states that individuals can gain knowledge by observation. Though not developed in the health communication field, the theory has much to offer in this area (Bandura, 1998). Tailored messages may be created in such a way as to present scenarios that can be modeled by the viewer through stories, anecdotes, and testimonials.

Cortese and Lustria (2012) created a web-based tailored intervention to educate teens about sexual health and decision making. Message tailoring drew heavily from constructs in the health belief model and social cognitive theory. Overall, messages were created to raise perceptions of risk (*perceived severity*), address individual concerns about what it will take to adopt the recommended behavior (e.g., money, effort, time; *perceived barriers*), and build confidence that taking the recommended action can reduce risk (*perceived benefits*). Tailored testimonials were presented to model safe sex behaviors and healthy decision making and to increase perceived self-efficacy to carry out the prescribed behaviors. Results of the randomized controlled trial comparing the tailored site with a generic site showed that those in the

tailored condition had higher satisfaction with the site and elaborated on the health messages more deeply as compared with those exposed to the non-tailored site.

The theory of reasoned action and planned behavior (TRA/TPB), another predictive model, links together beliefs, attitudes, and behavioral intentions to predict behavior (Fishbein & Ajzen, 1975). A number of tailoring studies have used TRA/TPB successfully. Webb et al. (2010) conducted a meta-analysis to examine the use of theory-based message tailoring and found that studies identifying TPB as their theoretical frame (9 out of 85 studies) reported larger effect sizes compared with all other tailored interventions included in the review. To exemplify how TPB can inform tailoring, Hurling et al. (2007) conducted a randomized controlled trial to evaluate the efficacy of a tailored physical activity program using web and mobile phone technology. Participants in the real-time tailored condition received tailored feedback, tailored solutions to address their perceived barriers, a weekly exercise plan with SMS and email reminders, and access to a message board for social support. Participants in both groups were issued wrist-worn accelerometers to monitor their level of physical activity for 9 weeks. Results showed that the tailored group reported a significantly greater increase over baseline than the control group for perceived control and intention/expectation to exercise. Participants in the tailored condition also lost a greater percent of their body fat than the control group.

The Future of Message Tailoring

Thoughts about applying the process of mass customization (Davis, 1989) to health messaging was first introduced in the late 1980s and early 1990s. Kreuter, Farrell, Olevitch, and Brennan (2000) attributed the turn toward mass customization to three innovations: (a) increasing popularity of audience segmentation in developing health education materials, (b) the introduction of new models of individual health behavior change (e.g., TTM), and (c) advances in computer technology. Of these three, the application of computers for processing individual data and for generating individual reports provided the tipping point for mass production of customized content. First-generation tailored interventions were largely print based or delivered via desktop computers or kiosks and typically involved a single, simple tailoring assessment.

Over time, the sophistication of tailored interventions has greatly increased concomitant with advances in information and communication technologies. These advances have benefited message tailoring by increasing reach, improving delivery, and improving assessment. In particular, web technologies have made it possible to scale the production and delivery of tailored messages to multiple individuals at relatively low cost. Internet delivery improves the reach of effective behavior change interventions to potentially hard-to-reach population groups and provides wider access to expert care and feedback. Synchronous and asynchronous technologies (e.g., email, SMS, chat) present multiple options for the delivery of tailored content, reminders, and feedback. Users can access tailored content at any time and receive “just-in-time” information when and where they need it the most. Users also have more options for scheduling and receiving tailored reminders with the ability to personalize notification settings.

In addition to the availability of multiple delivery modes (e.g., web and mobile technologies), tailored messages may be communicated via different formats (e.g., audio, video, simulations, games, virtual reality). Multimodal options are beneficial for addressing the needs of participants with different literacy levels, learning styles, and technological competencies. Interactive technologies (e.g., e-journaling, quizzes, video, simulations, discussion forums)

provide excellent platforms for implementing effective behavior change strategies, such as tracking and monitoring, goal setting, skills building, and providing tailored feedback and social support. As an example, Stanczyk et al. (2014) conducted a randomized controlled trial comparing the efficacy of a stage-matched tailored smoking cessation intervention with tailored video or tailored text and a generic control. Video tailoring was found to be more effective in increasing 7-day point prevalence abstinence than the control condition and resulted in significantly higher prolonged abstinence rates among smokers with a low readiness to quit.

Social media also have the potential for amplifying access to tailored interventions through cross-platform communication strategies and content repurposing (e.g., re-tweeting, blogging). Web 2.0 technologies like social networks, microblogging tools, and video-sharing sites can expand messaging opportunities while also providing highly interactive, dynamic, and flexible content that may improve user engagement.

Perhaps one of the most exciting areas of development in tailored messaging is the use of mobile devices and sensing technologies. Mobile technologies are particularly suited for persuasion as people are deeply attached to their mobile devices and have them wherever they go, making it easy to deliver persuasive messages when and where they need it the most. The delivery of reminders, tailored motivational messages, and coping tips could be triggered during opportune moments—for example, when the mobile system senses a user's location as a place where temptation typically lies.

The new generation of smart phone and wearable technologies (e.g., Apple Watch, Fitbit, Nike FuelBand) have also enhanced users' ability to track and monitor progress on their health goals without requiring manual data entry. These advances are paving the way for more synchronous and interactive tailoring based on real-time tracking through ubiquitous and unobtrusive sensing (Wangberg & Psychol, 2013). Coupled with advances in artificial intelligence and data processing methods, researchers may one day be able to fully automate the tailoring process, conduct iterative, real-time tailoring, and lower the burden for user assessment.

Author Biographies

Mia Liza A. Lustria, PhD, is an associate professor and chair of the undergraduate program at the School of Information, Florida State University. She has a successful record of publications and funded research in consumer health informatics aimed at improving access to healthcare and education through the use of digital and interactive media. Her current work focuses on theory-based message tailoring and the design and evaluation of persuasive technologies for use in behavior change interventions.

Juliann Cortese, PhD, is an associate professor in the School of Communication, Florida State University. Her area of research focuses on the uses and effects of online health content, having examined online tailoring, mediated social support, and mental processes engaged in while examining online content. She has published in top ranking journals including *Patient Education and Counseling*, *Journal of Health Communication*, *Journal of the American Society for Information Science and Technology*, and *Human Communication Research*.

References

- Bandura, A. (1998). Health promotion from the perspective of social cognitive theory. *Psychology and Health*, 13(4), 623–649. doi:10.1080/08870449808407422

- Centers for Disease Control and Prevention. (2015, 15 May). Healthy weight. Retrieved from <http://www.cdc.gov/healthyweight/prevention/index.html>
- Chodosh, J., Morton, S. C., Mojica, W., Maglione, M., Suttorp, M. J., Hilton, L., ... Shekelle, P. (2005). Meta-analysis: Chronic disease self-management programs for older adults. *Annals of Internal Medicine*, 143(6), 427–438.
- Cortese, J., & Lustria, M. L. A. (2012). Can tailoring increase elaboration of health messages delivered via an adaptive educational site on adolescent sexual health and decision making? *Journal of the American Society for Information Science and Technology*, 63(8), 1567–1580. doi:10.1002/asi.22700
- Davies, E. B., Morriss, R., & Glazebrook, C. (2014). Computer-delivered and web-based interventions to improve depression, anxiety, and psychological well-being of university students: A systematic review and meta-analysis. *Journal of Medical Internet Research*, 16(5), e130. doi:10.2196/jmir.3142
- Davis, S. (1989). From future perfect: Mass customizing. *Planning Review*, 17(2), 16–21. doi:10.1108/eb054249
- Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention, and behavior: An intro to theory and research*. Reading, MA: Addison-Wesley.
- Hawkins, R. P., Kreuter, M., Resnicow, K., Fishbein, M., & Dijkstra, A. (2008). Understanding tailoring in communicating about health. *Health Education Research*, 23(3), 454–466. doi:10.1093/her/cyn004
- Hurling, R., Catt, M., De Boni, M., Fairley, B. W., Hurst, T., Murray, P., ... Barkhuus, L. (2007). Using Internet and mobile phone technology to deliver an automated physical activity program: Randomized controlled trial. *Journal of Medical Internet Research*, 9(2), e7. doi:10.2196/jmir.9.2.e7
- Kessels, L. T. E., Ruiter, R. A. C., Brug, J., & Jansma, B. M. (2011). The effects of tailored and threatening nutrition information on message attention. Evidence from an event-related potential study. *Appetite*, 56(1), 32–38. doi:10.1016/j.appet.2010.11.139
- King, D. E., Mainous, A. G., III, Carnemolla, M., & Everett, C. J. (2009). Adherence to healthy lifestyle habits in US adults, 1988–2006. *The American Journal of Medicine*, 122(6), 528–534. doi:10.1016/j.amjmed.2008.11.013
- Krebs, P., Prochaska, J. O., & Rossi, J. S. (2010). A meta-analysis of computer-tailored interventions for health behavior change. *Preventive Medicine*, 51(3–4), 214–221. doi:10.1016/j.ypmed.2010.06.004
- Kreuter, M. W., Farrell, D., Olevitch, L., & Brennan, L. (2000). *Tailoring health messages: Customizing communication with computer technology*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Kreuter, M. W., Strecher, V. J., & Glassman, B. (1999). One size does not fit all: The case for tailoring print materials. *Annals of Behavioral Medicine*, 21(4), 276–283. doi:10.1007/BF02895958
- Kreuter, M. W., & Wray, R. J. (2003). Tailored and targeted health communication: Strategies for enhancing information relevance. *American Journal of Health Behavior*, 27(Suppl. 3), S227–S232.
- Lustria, M. L. A., Cortese, J., Noar, S. M., & Glueckauf, R. L. (2009). Computer-tailored health interventions delivered over the web: Review and analysis of key components. *Patient Education and Counseling*, 74(2), 156–173. doi:10.1016/j.pec.2008.08.023
- Lustria, M. L. A., Noar, S. M., Cortese, J., van Stee, S., Glueckauf, R. L., & Lee, J. (2013). A meta-analysis of web-delivered tailored health behavior change interventions. *Journal of Health Communication*, 18(9), 1039–1069. doi:10.1080/10810730.2013.768727
- Noar, S. M., Benac, C. N., & Harris, M. S. (2007). Does tailoring matter? Meta-analytic review of tailored print health behavior change interventions. *Psychological Bulletin*, 133(4), 673–693. doi:10.1037/0033-2909.133.4.673
- Petty, R. E., Briñol, P., & Priester, J. R. (2009). Mass media attitude change: Implications of the Elaboration Likelihood Model of persuasion. In J. Bryant & M. B. Oliver (Eds.), *Media effects: Advances in theory and research* (3rd ed., pp. 125–164). New York, NY: Routledge.
- Petty, R. E., & Cacioppo, J. T. (1986). The Elaboration Likelihood Model of persuasion. In B. Leonard (Ed.), *Advances in experimental social psychology* (Vol. 19, pp. 123–205). San Diego, CA: Academic Press.
- Prochaska, J. M., & Prochaska, J. O. (2014). A stage approach to enhancing adherence to treatment. In D. I. Mostofsky (Ed.), *The handbook of behavioral medicine* (pp. 58–75). West Sussex: Wiley.

- Prochaska, J. O., & DiClemente, C. C. (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology*, 51(3), 390–395. PMID: 6863699
- Rosenstock, I. M. (1974). Historical origins of the Health Belief Model. *Health Education & Behavior*, 2(4), 328–335. doi:10.1177/109019817400200403
- Stanczyk, N., Bolman, C., van Adrichem, M., Candel, M., Muris, J., & de Vries, H. (2014). Comparison of text and video computer-tailored interventions for smoking cessation: Randomized controlled trial. *Journal of Medical Internet Research*, 16(3), 35–52. doi:10.2196/jmir.3016
- Wangberg, S. C., & Psychol, C. (2013, December 2–5). Personalized technology for supporting health behaviors. Paper presented at the 2013 IEEE 4th International Conference on Cognitive Infocommunications (CogInfoCom), Budapest, Hungary.
- Webb, L. T., Joseph, J., Yardley, L., & Michie, S. (2010). Using the Internet to promote health behavior change: A systematic review and meta-analysis of the impact of theoretical basis, use of behavior change techniques, and mode of delivery on efficacy. *Journal of Medical Internet Research*, 12(1), e4. doi:10.2196/jmir.1376
- Woolford, S. J., Clark, S. J., Strecher, V. J., & Resnicow, K. (2010). Tailored mobile phone text messages as an adjunct to obesity treatment for adolescents. *Journal of Telemedicine and Telecare*, 16(8), 458–461. doi:10.1258/jtt.2010.100207

Suggested Reading

- Hawkins, R. P., Kreuter, M., Resnicow, K., Fishbein, M., & Dijkstra, A. (2008). Understanding tailoring in communicating about health. *Health Education Research*, 23(3), 454–466. doi:10.1093/her/cyn004
- Kreuter, M. W., & Wray, R. J. (2003). Tailored and targeted health communication: Strategies for enhancing information relevance. *American Journal of Health Behavior*, 27(Suppl. 3), S227–S232.
- Lustria, M. L. A., Cortese, J., Noar, S. M., & Glueckauf, R. L. (2009). Computer-tailored health interventions delivered over the web: Review and analysis of key components. *Patient Education and Counseling*, 74(2), 156–173. doi:10.1016/j.pec.2008.08.023
- Lustria, M. L. A., Noar, S. M., Cortese, J., van Stee, S., Glueckauf, R. L., & Lee, J. (2013). A meta-analysis of web-delivered tailored health behavior change interventions. *Journal of Health Communication*, 18(9), 1039–1069. doi:10.1080/10810730.2013.768727
- Noar, S. M., Benac, C. N., & Harris, M. S. (2007). Does tailoring matter? Meta-analytic review of tailored print health behavior change interventions. *Psychological Bulletin*, 133(4), 673–693. doi:10.1037/0033-2909.133.4.673

Naturalistic Observation of Social Interactions

Angela L. Carey, Kelly E. Rentscher,
and Matthias R. Mehl

Psychology Department, University of Arizona, Tucson, AZ, USA

People's social relationships and daily social interactions can impact health in profound ways. For example, in a meta-analytic review, Holt-Lunstad, Smith, and Layton (2010) found that across 148 studies totaling 308,849 participants, individuals with strong social relationships had a 50% greater likelihood of survival relative to those with poorer social relationships. The magnitude of this effect in fact exceeded many well-known mortality risk factors (e.g., obesity, smoking), providing further evidence that social interactions not only influence quality of life but can also have important implications for human survival. It is therefore just as necessary and important to include social interactions in research on health as it is other mortality risk factors.

Interestingly, when considering existing social health research, there appears to be a discrepancy in the precision researchers tend to afford independent versus dependent variables in what might be called the “social health equation,” or the prediction of health outcomes by psychosocial variables (Mehl, Robbins, & Deters, 2012). On the criterion side, researchers measure important health outcomes using an assortment of high-fidelity methods to assess health functioning as objectively and precisely as possible (e.g., endocrinological and immunological biomarkers, markers of gene, or cellular functioning). However, on the predictor side, health-relevant psychosocial constructs tend to be measured with considerably less fidelity, objectivity, and precision. This is evidenced by a general inclination to rely heavily on self-report methods or, more specifically, what participants (can) tell about their psychological and social functioning. Self-reports are undoubtedly valuable given that they provide important information and can be obtained efficiently and at low cost; at the same time, it is clear that they do not afford a level of fidelity and precision that can rival that of objective health outcome measures. For example, the theoretical and empirical distinctness of subjective (i.e., experiential) and objective (i.e., observable) aspects of moral behaviors illustrate the need for greater precision when measuring health-relevant social constructs. With respect to

health-relevant processes, it seems clear that the subjective experience of gratitude differs in important ways from the behavioral expression of gratitude. Whereas the former is first and foremost a result of the interplay of inner cognitive processes, the latter is critically shaped by outer, situational, and cultural factors. For example, subjective experience of gratitude can promote self-esteem and reduce negative emotions such as envy, regret, and resentment, thereby affecting “downstream” constructs such as happiness and depression. Behavioral expressions of gratitude, on the other hand, including enhanced empathy toward others, reduced aggression, and further, the act itself (e.g., inviting an acquaintance to lunch to express thanks for assisting when the babysitter unexpectedly canceled) can lead to more opportunities for social interaction and development of new relationships.

Gratitude has been tied to health outcomes such as enhanced sleep quality and duration, as well as improved physiological health. It has also been found to attenuate stress responses, which in turn can improve cardiovascular function and reduce risk of heart disease. Gratitude is often assessed, however, via self-report only, which makes it challenging to truly disentangle experienced and observed gratitude and to determine how the two independently relate to health outcomes. Given this, it is important to be able to separate these two components measurement-wise, which can only be accomplished through the combination of first-person self-report and third-person observational assessments. Analyzing behavioral observations in conjunction with subjective reports of gratitude enables social scientists to achieve a deeper understanding of the interaction between what is behaviorally occurring and what is subjectively experienced in people’s daily lives, thus increasing measurement fidelity and precision in operationalizing health-relevant constructs. In short, the field of health psychology could benefit substantially from a multi-method approach to studying daily social interactions that complements self-reports with observational assessments.

Ambulatory Assessment Methods

Although the majority of research investigating social interactions and their influence on health continues to be based on self-report surveys, the last 20 years have seen a steady increase in the use of ambulatory assessment methods that can capture aspects of participants’ daily lives “in vivo.” Ambulatory assessments are defined as the use of field methods to assess the ongoing behavior, physiology, experience, and environmental aspects of people in naturalistic or unconstrained settings. Ambulatory assessment uses ecologically valid tools to understand biopsychosocial processes as they unfold naturally in time and in context (www.ambulatory-assessment.org).

Ambulatory assessments have several key advantages (Mehl & Conner, 2012). First, ambulatory assessments take place directly within participants’ everyday lives; they maximize generalizability across a wide range of real-world contexts. Second, they capture individuals’ in-the-moment or close-in-time behaviors, thoughts, and feelings and thereby bypass memory-related biases that can undermine the validity of global and retrospective self-reports (e.g., recall-related errors resulting from serial position effects, mood effects, and heuristics and estimation strategies). A third advantage of ambulatory assessments is that their typical high density of longitudinal measurements renders them uniquely suited to studying how individuals’ behaviors, thoughts, and feelings change over time in ways that cannot be adequately modeled within a laboratory setting. When experiences are assessed in real time (or as close to their occurrence as possible), participants’ responses tend to more accurately represent what is occurring in the moment and are less influenced by applicable heuristics or schemata. In this way, intensive longitudinal ambulatory assessments are ideally suited for identifying

individualized patterns of behavior and behavior change over time. Ambulatory assessment methods are also often referred to (with similar but not always identical meaning) as ecological momentary assessment, intensive longitudinal, experience sampling, and diary methods (see Mehl and Conner (2012) for further information).

Ambulatory Assessment of Physiology and Experience

Ambulatory monitoring allows for the sampling of physiological activity in daily life. Modern-day mobile technology allows for the reliable measurement of biological signals via portable signal recording devices that capture activity such as blood pressure, electrodermal activity, body temperature, and hormones, as a person goes about their everyday life.

If researchers' primary interest involves the momentary sampling of experiences in everyday life, daily diary assessments are some of the most commonly used methods. These typically involve once-a-day assessments via Internet surveys for a prolonged period of time (e.g., a few weeks) in order to assess events or experiences that can easily be recalled at the end of the day, such as emotions in response to daily stressors. With experience sampling methods (ESM), participants complete self-report assessments repeatedly throughout the day, mainly through use of mobile technology, at random (i.e., signal-contingent sampling) or predetermined (i.e., interval-contingent sampling) times, or when a target event such as an argument with a romantic partner has occurred (i.e., event-contingent sampling).

Assessment of Behavior

There is tremendous value in obtaining subjective reports of individuals' daily thoughts and experiences using each of the aforementioned ambulatory methods. Yet, one important lesson from psychometrics is that people are not always in the position to accurately report what they do. Sometimes they may actively censor information that they do not want to report; other times (and probably more often), they bias their self-perceptions in socially desirable ways for psychological self-protection. However, there are also other times when people want to honestly and accurately report information, but they simply cannot, due to limitations inherent to human information processing; for example, it is nearly impossible to recall the number of times one sighs or laughs in a day (and it would be highly impractical to be able to do so). In circumstances such as this, some of the same concerns pertaining to global and retrospective self-reports may also apply to momentary self-reports. Although ambulatory self-reports can provide more accurate representations of individuals' perceived daily lives than retrospective or global assessments in the laboratory can, they still require individuals to report on their thoughts and behaviors and therefore are inherently limited to capturing what surpasses the perceptual threshold and enters into working memory. This can be problematic given that participants' responses to self-report scales can be affected by recent events or experiences, thereby rendering them potentially susceptible to momentary influences including mood effects, socially desirable responding (i.e., impression management and self-deception), and, perhaps most importantly, lack of awareness. Another potential concern is measurement reactivity, whereby participants' behaviors and/or responses might systematically change as a result of prolonged and repeated momentary self-report assessments. For example, repeatedly reporting on one's behavioral expression(s) of gratitude might render participants mindful of their grateful behavior (or lack thereof) and consequently alter future expressions of gratitude. It is also possible that participants may lose motivation and become more careless with their responses over time leading to increased measurement error.

Naturalistic Observation of Social Interactions

Naturalistic observation methods extend the area of ambulatory assessment to real-world observational methods. Naturalistic observation methods are well suited to addressing some of the limitations inherent in momentary self-reports. Furthermore, these observational methods aim to capture individuals' behavior from the perspective of an observer and can thereby offer a complementary approach to the study of everyday social interactions through the eyes (or the mind) of the "actor." Researchers have directly observed behavior in the real world through use of small cameras placed in participants' homes, as well as live videographers. In one study at the Sloan Center on Everyday Lives of Families (CELf) at the University of California, Los Angeles, videographers with ambulatory cameras accompanied participants to work, in transit, in public, and as they reunited with their families at the end of the day. This study collected more than 1,400 hr of video recordings from 32 different dual-earner families over the course of 2 week-days and 2 weekend days, as they normally lived their lives (e.g., Ochs, Graesch, Mittman, Bradbury, & Repetti, 2006). This study has yielded several important findings, one of which is that job stressors (collected via daily diary assessments of experiences at work) appear to be linked with increases in expressed marital anger and withdrawal (derived from the video recordings). Additionally, self-reported negative mood mediated the association between experienced job stress and observed at-home marital behavior (e.g., Story & Repetti, 2006). In adopting a multi-method approach that combined self-report and observational methods, this research provided real-world evidence of "spillover effects," in which stressors experienced outside of the home impact behavior, cognitions, and emotions inside the home.

Although video-based methods produce large amounts of data that allow for detailed and nuanced investigations of participants' naturalistically observed daily social interactions, they are very labor and resource intensive and afford a certain amount of intrusiveness (i.e., having cameras and often even members of the research team present in the setting). An issue that accompanies direct observational techniques such as these is the potential that participants' awareness of being video recorded might produce evaluation apprehension and, as a consequence, behavioral reactivity, in which the participants alter their behavior in response to being observed.

The Electronically Activated Recorder (EAR)

One methodology that can alleviate some of these potential issues while preserving the ability to observationally capture social interactions during daily life is the Electronically Activated Recorder (EAR) (Mehl, Pennebaker, Crow, Dabbs, & Price, 2001). The EAR is a portable digital audio recorder designed to intermittently capture snippets of ambient sounds from individuals' momentary environments, providing acoustic logs of their daily lives as they naturally unfold. The EAR allows researchers to unobtrusively capture behavior in real time, bypassing many of the previously mentioned limitations that often accompany self-report methods. In its most recent version, the EAR is a software application (the "iEAR") that runs on iPod touch devices and can be downloaded freely from Apple's App Store. To protect the iPod device from impact, it is handed to participants in a protective case with a clip so that participants can wear the EAR by attaching it to a belt, the outside of their clothing, or a bag as they go about their normal daily lives. The EAR's sampling rate can be freely programmed, and sampling patterns that have proven useful in prior EAR research are 30 s every 12 min (or 5 times per hour; the default sampling pattern) and 50 s every 9 min. This translates roughly

into recording 5 and 10%, respectively, of the participants' day, leaving 95 and 90%, respectively, of their day private (i.e., unrecorded). In studies where there are special privacy considerations, such as using the EAR in the workplace, weekend monitoring (i.e., recording Friday evening through Sunday evening) may be preferable and the only way to obtain good compliance and valid information. In cases where there are no out-of-the-ordinary privacy concerns (e.g., among students or older, retired adults), monitoring on both weekdays and weekend days is preferable and can provide more generalizable information about participants' typical patterns of social interaction.

The current version of the EAR, that is, the iEAR app, also offers a randomization feature that allows researchers to randomize the length of the interval between each recording. At 100% randomization, recording times are fully random; at 50% randomization yields random recordings within half and one and a half times the programmed interval; for example, with a sampling rate of 30 s every 12 min, 30-s recordings will occur randomly between 6 and 18 min after the previous recording. Because of the electronic nature of the recording, participants cannot tell when the EAR is recording. The ability to set a "blackout period," which is a pre-specified period of guaranteed non-recording, is another feature of the current iEAR app. It is recommended to adjust the blackout period to participants' reported bedtime and wake-up time to maximize the obtained information and the protection of participants' privacy.

When it comes to determining a desired sampling pattern (with respect to frequency and length of recording), researchers should first consider the natural base rate of their targeted behavior (i.e., how often does it happen in a day?); low base rate behaviors (e.g., crying, conflict) can require more frequent and longer-term sampling than higher base rate behaviors (e.g., watching TV, talking to the partner) to ensure that sufficient variability in the target behavior is represented in the recordings. With respect to deciding on the length of an individual recording sample (e.g., 30, 50 s, 2 min), researchers need to consider how much information coders need to clearly identify the (presence or degree of) construct. For example, if the researcher is interested in demand-withdraw interactions, a complex or "molar" psychological construct, longer recording segments (e.g., 2–3 min) might be necessary to reliably infer the pattern from a couple's way of discussing a problem. Alternatively, if the researcher is mostly interested in relatively narrow or "molecular" behaviors, such as whether or not a person is talking, laughing, sighing, or watching TV in any given recording segment, relatively short recordings (e.g., 30 s) will likely provide sufficient information. In our research, we have successfully employed both 50- and 30-s recording segments but have found 30-s segments to be usually preferable for both pragmatic and psychometric reasons (Mehl et al., 2012). Our default sampling rate of 30-s recordings every 12 min yields quite a high sampling density (approximately 5 samples per hour) that should provide good resolution for between and many within-person analyses; and, consistent with findings from decades of thin-slice research, we have found 30-s sound bites to yield surprisingly rich information about a person's behavior and social context at a given moment.

How Can the EAR Sound Files be Analyzed?

To extract relevant information from the EAR sound files, researchers can use a psychological rating or behavior counting approach. With the psychological rating approach, trained expert raters listen to the recordings and judge the degree to which they indicate the presence of a construct of interest (e.g., social support). To date, the majority of EAR studies have utilized a behavior counting approach. Here, trained coders make binary "behavior present" versus "behavior absent" codings for each recorded sound file using a standardized coding system

referred to as the Social Environment Coding of Sound Inventory (SECSI) (Alisic, Barrett, Bowles, Conroy, & Mehl, 2015; Mehl et al., 2012). The SECSI captures a person's daily social environment, behaviors, interactions, and affect using the following four core categories: the participant's current (a) location (e.g., home, in transit, in public), (b) activity (e.g., watching TV, doing housework, eating), (c) social interaction (e.g., alone versus with others, talking to someone in person, talking on phone), and (d) emotional expression (e.g., crying, sighing, laughing). Conceptually, the EAR captures information about the types of social environments individuals are in (i.e., person–environment effects at the level of situation selection) and how they interact within those environments (i.e., person–environment effects at the level of person–situation interaction). In addition, all of the participants' utterances are transcribed verbatim, providing the ability to extract information about individuals' everyday language use from the sound files (e.g., via computerized text analysis). The language data can in turn be utilized to examine how natural language use reflects psychological states and traits; for example, individuals' personal pronoun use has been investigated as an automatic, nonconscious linguistic marker of interpersonal focus. To assess the psychometric properties of the EAR data, inter-coder reliability estimates are obtained either from double codings (i.e., independent sets of codings by two trained coders) or from codings accompanying a standard set of training EAR recordings. It is important that reliability estimates are calculated for the targeted unit of analysis. That is, if a researcher analyzes the EAR data at the aggregate level (e.g., percentage of sound files in a day in which the participant laughed), then the reliability should be reported for the aggregate measure; if a researcher analyzes each EAR sound file individually (within person), then an estimate of the coding of an individual sound file should be provided. In our lab, we first test inter-coder reliability at the level of the sound file by having all coders of a study code a standard set of training EAR recordings; this initial reliability check is part of the coder training and enables us to calibrate coders. Following this, we submit every sound file to an independent double coding and then estimate the reliability of the average measure composed of the two sets of codings. In our experience, it is reasonable to rely on single codings for narrow and highly concrete behaviors (e.g., whether or not the participant is talking or whether or not the TV is switched on); broader and more abstract psychological behaviors (e.g., disclosure, support, conflict) are more difficult to code reliably and thus tend to warrant a double-coding approach.

Ethical Considerations Pertaining to the EAR Method

Importantly, recording ambient sounds around participants raises ethical and legal concerns. EAR studies conducted within our laboratory routinely implement a series of safeguards to protect participants' privacy and ensure confidentiality of the data. First, data collection is limited to a fraction of a person's day; using the default sampling pattern (i.e., 30 s every 12 min), only 5% of a participant's total day is recorded, leaving 95% of the day private. Limiting the recordings to a brief 30 s yields sound files that are long enough to reliably extract behavioral information, yet also short enough to capture as little contextualized personal information as possible. Additionally, the latest version of the iEAR features a privacy button that, upon pressing it, guarantees participants a prespecified (by the researcher) time of non-recordings (e.g., 5 min; a countdown timer provides visual feedback for how much longer privacy is guaranteed). Another safeguard, and one of the most important, is that all participants are given the opportunity to listen to their EAR recordings and to delete any sound files that they do not want on record before investigators access any of the data.

What makes the EAR unique from other ambulatory assessment methods designed to assess behavior is that it differs in its assessment perspective. With traditional self-report-based

ambulatory assessment methods, the participant is the agent and responds with subjective experiential accounts of an event, whereas the EAR adopts the perspective of a bystander or observer, yielding corresponding objective behavioral accounts (i.e., “participant as object”). In this sense, the EAR alleviates the burden placed on participants to interrupt their day and respond to a series of questions. It does, however, place the burden of potential psychological discomfort on participants: the knowledge that they are being intermittently recorded could cause participants to feel that their privacy is being violated or feel something akin to evaluation apprehension. Most participants experience some awareness when first equipped with the EAR, but this heightened self-consciousness tends to decrease within the first couple of hours and stays relatively low after that (Mehl & Holleran, 2007).

One of the more serious challenges, however, concerns the issue of potentially recording bystanders. In the United States there are relatively few restrictions regarding the recording of behavior in public places, but some states only permit recordings of private conversations if all interactants have knowledge of the recording device. In our studies, we usually equip the EAR with a neon triangle-shaped alert sign that displays a picture of a microphone, alerting people around the participants that the device may be recording. Participants are further encouraged to openly mention the EAR in conversations with others. This safeguard is aimed at minimizing expectations of privacy for bystanders, thereby indirectly eliciting their passive consent (i.e., through bystanders “behaving” in the awareness of being recorded). For further protection, all sound files are coded by staff who are trained and certified to conduct research with human subjects, and any identifying information that is captured in the sound files is omitted from the transcripts. Participants also always have the option to delete any sound files that may contain information about bystanders. Finally, in most studies, it is recommendable to protect the sound files through a Certificate of Confidentiality that the National Institutes of Health issues (upon a simple application and at no cost) to protect study data from forced third-party disclosure.

Health and Social Interaction Research Utilizing the EAR Methodology

As an unobtrusive observational method, the EAR has the unique advantage of being able to capture health-relevant behaviors that are rarely studied, as they may reside largely outside of conscious awareness and therefore tend to evade self-report assessment. Coupled with other more traditional momentary assessment methods, the EAR provides a comprehensive representation of health-relevant social processes as they naturally occur in everyday life. For example, 13 rheumatoid arthritis patients wore the EAR for 2 separate weekends spaced approximately 1 month apart to test the degree to which naturalistically observed sighing in daily life served as a behavioral indicator of self-reported depression and physical pain symptoms (Robbins, Mehl, Holleran, & Kastle, 2011). Sighing emerged as significantly and substantially related to patients’ levels of depression but was not associated with their reported pain and number of symptom “flare” days. A second study tracked spontaneous swearing in the daily interactions of breast cancer and rheumatoid arthritis patients to investigate its potential role in coping with chronic illness (Robbins, Focella, et al., 2011). Interestingly, naturalistically observed swearing in the presence of other people, but not swearing when alone, was associated with decreases in self-reported emotional support and increases in depressive symptoms—suggesting that swearing can undermine emotional support with potential consequences for psychological adjustment. Importantly, both sighing and swearing are health-related behaviors that are difficult if not impossible to study with ambulatory self-report methods and

are, by nature, optimally assessed from an (unobtrusive) observational perspective. Together, these findings highlight the utility of the EAR method in capturing largely unconscious behaviors that have potentially important implications for health.

The EAR has also been used to assess the impact of parents' psychological health on familial environments. For example, Slatcher and Trentacosta (2011) examined the association between parental depressive symptoms and child problem behaviors. Thirty-five two-parent families with children between the ages of 3 and 5 years were recruited into the study. Both parents completed a depression questionnaire, and the EAR was administered at two separate time points to capture the children's behavior and language. The children wore the EAR for two 1-day periods that took place 1 year apart from each other. Fathers' depressive symptoms correlated positively with children's crying, acting out, and watching TV at EAR Time 1, as well as children's watching TV at EAR Time 2. Mothers' depressive symptoms correlated positively with children's crying at EAR Time 2, and both mothers' and fathers' depressive symptoms were positively associated with their children's negative behavioral symptoms at Time 2. In addition, mothers' and fathers' depressive symptoms uniquely predicted increases in children's anger word use from Time 1 to Time 2.

In more recent research, the EAR has been used to investigate family interactions as they relate to physical health, or more specifically, to examine the associations between naturalistically observed family conflict and youth asthma symptoms (Tobin et al., 2015). Fifty-four adolescents with asthma wore the EAR for a 4-day period to capture conflict with parental figures and audible symptoms of asthma (i.e., wheezing), and both adolescents and parental figures completed daily diary reports of conflict and asthma symptoms. EAR-observed family conflict was strongly associated with both self-reported asthma symptoms and coder-observed wheezing, providing evidence that everyday conflict can have important implications for health symptoms such as asthma, particularly in the context of family interactions outside of the laboratory.

Finally, the examples of research findings presented here are not intended to represent an exhaustive review of existing EAR research within the field of social health psychology. Rather, these examples serve to illustrate the utility of combining both subjective experiential reports with behavioral observations in order to gain a deeper understanding of how daily social interactions impact health and, in some cases, how health impacts social interaction.

Mobile Sensing Methods

A relatively new and rapidly developing approach to objectively documenting social interactions in daily life is mobile sensing methods. With the fast-paced development of mobile devices, that is, smartphones and wearables, coupled with the growing number of people who now own and depend on them, there is no question that objective assessment of aspects of social interactions will soon be at least partially absorbed by mobile sensing. Generally speaking, mobile sensing applications are used to make inferences about users' behavior, emotions, environments, and daily life patterns through computer integration of the data that comes from both interactions with the actual user interface of mobile devices (e.g., duration of phone calls, number of text messages) and the multiple sensors that are embedded in smartphones (e.g., GPS, Bluetooth, accelerometers). Wearable devices such as portable smart watches will also be increasingly incorporated into research on account of the behavioral (e.g., physical activity) and physiological (e.g., heart rate) information they gather. Mobile sensing applications

have much to offer in the amount of data they can collect and in the ways in which they open the field up to the objective characterization of a person's moment-to-moment social environment (e.g., location via GPS, solitary vs. social via Bluetooth, sedentary vs. active via accelerometers), and research within social and health psychology is swiftly moving in this direction. It will undoubtedly continue on its trajectory of becoming the new gold standard method for "big data collection," given that these devices can automatically store and transmit vast amounts of real-world user interaction data essentially instantaneously. Mobile sensing methods can potentially provide mass amounts of real-world data across thousands of participants, thereby critically reducing the resource burden for the researcher (i.e., studies can be run remotely at scale rather than locally in person). An example of one such mobile sensing method is EmotionSense (Rachuri et al., 2010). EmotionSense is a platform for creating mobile applications that can easily be installed onto off-the-shelf smartphones, with a focus on gathering not only data pertaining to participants' emotions, as the name would suggest, but also proximity and patterns of conversations with others. The EmotionSense apps are able to collect experience sampling self-reports as well as passive measurement of physical activity, mobility, and socializing via internal sensors and settings that can be configured for researchers' specific experience sampling studies.

Many mobile sensing applications sound ideal for conducting real-world research, but there are also limitations to utilizing mobile sensing as a methodology for naturalistic observation of social interactions. One of the most immediate concerns to address is the question of how to appropriately synthesize and interpret data derived from mobile sensing methods. Even the most advanced social sensing tools still struggle with extracting information at a level that is useful and readily interpretable for researchers. Physical activity is typically downloaded as a raw stream, meaning that artifacts have to be filtered out and activity states (e.g., sitting, standing, walking, running) have to be algorithmically computed. In a similar way, raw GPS data provides relatively little psychological information and requires post-processing to label the coordinates and make psychological sense of frequented locations (e.g., identification of home or the work place). Additionally, self-reported activity often differs from accelerometer-derived activity, and because people do not always carry their phones with them, further errors can be introduced into the existing algorithms. Intensity of physical activity also presents a crucial issue; mobile devices often struggle with basic extraction of information regarding intensity level of activity, and this is particularly important given that intensity provides significant health-relevant information, above and beyond the knowledge that someone has simply been active.

Another issue that accompanies mobile sensing methods involves some ethical concerns. Guaranteeing protection of participants' privacy is difficult; there is still the challenge of finding ways to ensure that participants' data, which largely resides online, will remain both de-identified and stored safely and securely. Moving beyond these pragmatic concerns, mobile sensing methods also differ widely from other forms of naturalistic observation in terms of what they can and cannot collect (or algorithmically produce) regarding daily social interactions. These apps can likely provide more, and potentially better quality, information regarding electronic forms of communication (e.g., text messaging, online social network use), and this increased accuracy greatly benefits the study of computer-mediated social interactions and their relation to health. However, due to privacy reasons, mobile sensing may face more challenges tracking in-person social interaction.

There is no question regarding whether smartphone sensing applications have been effectively established as proof of concept. However, for this area of research to move forward in a

way that can significantly benefit social and health psychologists, more research is needed to ensure that algorithms are producing data with a level of precision that is acceptable to health researchers. In other words, what researchers urgently need are thorough psychometric validations of sensor data as they accumulate in the context of people's natural use of their smartphones and in the context of existing mobile sensing applications.

Much work is being done to move the field of mobile sensing forward, and it will inevitably introduce impactful changes regarding the collection of naturalistic observation data and the assessment of social interactions and health. Implemented (and validated the right way), these methods can further help reduce the discrepancy that exists between the fidelity of the measures on the "social" side and the fidelity of the measures on the "health" side of the "social-health equation" (i.e., the prediction of health outcomes from psychosocial variables). Recent EAR research has demonstrated that assessing social interactions and health through use of both momentary self-report (e.g., rheumatoid arthritis "flare" days; parental depressive symptoms) and behavioral observation (e.g., swearing in the presence of others; child problem behavior in the home) can undoubtedly produce a more nuanced picture of how social and health constructs manifest and interact within the real world. Further, adopting a combined naturalistic approach affords researchers the ability to disentangle whether and when a person's experiential report of an interaction may differ from what is captured observationally and what such discrepancies might indicate. The ability to capture and make sense of any such theoretical and empirical distinctness is an example of how this combination of ambulatory assessment methods can serve to increase precision and fidelity of health-relevant psychosocial variables, ensuring a methodologically sound approach. When both sides of the "social-health equation" are held to comparable levels of precision, researchers can more comprehensively assess how real-world social interactions impact health on a variety of levels and ultimately, with the assistance of rapidly evolving mobile technologies, determine how researchers can utilize this knowledge to improve and maintain health and well-being.

Author Biographies

Angela L. Carey, MA, is a social psychology doctoral student at the University of Arizona. Her research centers around social interactions and social support influences on coping in the context of major life upheavals (e.g., divorce). Her work utilizes the Electronically Activated Recorder (EAR) in combination with other methods (e.g., daily dairies, linguistic analysis) to study how different types of real-world social interactions change over time and in relation to self-reported adjustment and well-being.

Kelly E. Rentscher, MA, is a clinical psychology doctoral candidate at the University of Arizona. Her research involves a multi-method approach to studying how couples cope with stress and chronic illness, how relationship processes affect health over time (and vice versa), and how couple-focused interventions can target these processes to improve health outcomes. Her research employs linguistic analysis, observational, and naturalistic methodologies in laboratory and real-world settings.

Matthias R. Mehl, PhD, is associate professor of psychology at the University of Arizona. He has developed the Electronically Activated Recorder (EAR) as a methodology for the unobtrusive naturalistic observation of daily life and explores other novel methods for studying daily life. In 2012, he coedited the *Handbook of Research Methods for Studying Daily Life* (Guilford Press). He is a founding member of the Society for Ambulatory Assessment.

References

- Alisic, E., Barrett, A., Bowles, P., Conroy, R., & Mehl, M. R. (2015). Topical review: Families coping with child trauma: A naturalistic observation methodology. *Journal of Pediatric Psychology*, 41(1), 117–127.
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7(7), e1000316. doi:10.1371/journal.pmed.1000316
- Mehl, M. R., & Conner, T. S. (Eds.) (2012). *Handbook of research methods for studying daily life*. New York, NY: Guilford Press.
- Mehl, M. R., & Holleran, S. E. (2007). An empirical analysis of the obtrusiveness of and participants' compliance with the electronically activated recorder (EAR). *European Journal of Psychological Assessment*, 23(4), 248–257. doi:10.1027/1015-5759.23.4.248
- Mehl, M. R., Pennebaker, J. W., Crow, D. M., Dabbs, J., & Price, J. H. (2001). The electronically activated recorder (EAR): A device for sampling naturalistic daily activities and conversations. *Behavior Research Methods, Instruments, & Computers*, 33, 517–523. doi:10.3758/BF03195410
- Mehl, M. R., Robbins, M. L., & Deters, F. G. (2012). Naturalistic observation of health-relevant social processes: The electronically activated recorder methodology in psychosomatics. *Psychosomatic Medicine*, 74(4), 410–417. doi:10.1097/PSY.0b013e3182545470
- Ochs, E., Graesch, A. P., Mittmann, A., Bradbury, T., & Repetti, R. L. (2006). Video ethnography and ethnoarchaeological tracking. In M. Pitt-Catsouphes, E. E. Kossek, & S. Sweet (Eds.), *The work and family handbook: Multi-disciplinary perspectives, methods, and approaches* (pp. 387–409). Mahwah, NJ: Erlbaum.
- Rachuri, K. K., Musolesi, M., Mascolo, C., Rentfrow, P. J., Longworth, C., & Aucinas, A. (2010). EmotionSense: A mobile phones based adaptive platform for experimental social psychology research. *Proceedings of the 12th ACM international conference on Ubiquitous Computing*, (pp. 281–290). doi: 10.1145/1864349.1864393
- Robbins, M. L., Focella, E. S., Kasle, S., López, A. M., Weihs, K. L., & Mehl, M. R. (2011). Brief report: Naturalistically observed swearing, emotional support and depressive symptoms in women coping with illness. *Health Psychology*, 30(6), 789–792. doi:10.1037/a0023431
- Robbins, M. L., Mehl, M. R., Holleran, S. E., & Kasle, S. (2011). Naturalistically observed sighing and depression in rheumatoid arthritis patients: A preliminary study. *Health Psychology*, 30(1), 129–133. doi:10.1037/a0021558
- Slatcher, R. B., & Trentacosta, C. J. (2011). A naturalistic observation study of the links between parental depressive symptoms and preschoolers' behaviors in everyday life. *Journal of Family Psychology*, 25(3), 444–448. https://doi.org/10.1037/a0023728
- Story, L. B., & Repetti, R. L. (2006). Daily occupational stressors and marital behavior. *Journal of Family Psychology*, 20, 690–700. doi:10.1037/0893-3200.20.4.690
- Tobin, E. T., Kane, H. S., Saleh, D. J., Naar-King, S., Poowuttikul, P., Secord, E., ... Slatcher, R. B. (2015). Naturalistically observed conflict and youth asthma symptoms. *Health Psychology*, 34(6), 622–631. doi:10.1037/hea0000138

Suggested Reading

- Conner, T. S., & Mehl, M. R. (2015). Ambulatory assessment: Methods for studying everyday life. In R. A. Scott, S. M. Kosslyn, & M. Stephen (Eds.), *Emerging trends in the social and behavioral sciences: An interdisciplinary, searchable, and linkable resource* (pp. 1–15). Thousand Oaks, CA: SAGE Publications. doi:10.1002/9781118900772.etrds0010
- Mehl, M. R., & Conner, T. S. (Eds.) (2012). *Handbook of research methods for studying daily life*. New York, NY: Guilford Press.
- Mehl, M. R., Robbins, M. L., & Deters, F. G. (2012). Naturalistic observation of health-relevant social processes: The electronically activated recorder methodology in psychosomatics. *Psychosomatic Medicine*, 74(4), 410–417. doi:10.1097/PSY.0b013e3182545470

Optimism and Physical Health

Maria G. Mens¹, Michael F. Scheier¹,
and Charles S. Carver²

¹*Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA*

²*Department of Psychology, University of Miami, Coral Gables, FL, USA*

Optimists are people who expect good things to happen to them. Pessimists are people who expect bad things to happen to them. Folk wisdom has long held that this difference among people is important in many aspects of living. In this case, folk wisdom appears to be right. A substantial body of research suggests that optimists experience better physical health than pessimists.

Defining Optimism

Defining optimism in terms of expectancies links the optimism construct to a long tradition of expectancy-value theories of motivation. These theories hold that people pursue only goals they both value and expect to obtain. Expectancies reflect a person's confidence that sought after goals will be attained and are particularly important when impediments arise in goal pursuit. If a person is confident about eventual success, he or she will persist despite difficulties. However, if confidence wanes, the person is likely to disengage effort, sometimes disengaging from the goal itself.

Expectancies exist at many levels of generality. A student can have expectancies about her ability to get to class on time, expectancies about her ability to learn a topic, and expectancies about her likelihood of finding a fulfilling career. The principles of expectancy-value theories pertain equally well to expectancies that are specific and expectancies that are more general. They should even apply to the most broad or general kinds of expectancies, which characterize optimists and pessimists. The "confidence" that is at issue in optimism is simply broader in scope, pertaining to most situations in life rather than just one or two.

Measuring Optimism

Research on the effects of optimism has flourished over the past 30 years. This research has taken several different routes to assessing optimism, leading to somewhat distinct literatures. The most common approach asks people whether they think that good or bad things will generally happen to them in the future. In this method, individuals are asked to respond to statements such as “I am optimistic about my future” (e.g., Scheier, Carver, & Bridges, 1994). Most of the research linking optimism to physical health has used this approach to measuring expectancies.

Researchers who study optimism often refer to optimists and pessimists as though they were distinct groups. However, this usage is often a verbal convenience. All approaches to the measurement of optimism provide scores that vary continuously across large numbers of people. Thus, as often conceived, people actually range from very optimistic to very pessimistic, with most falling somewhere between.

Interestingly, research exists (reviewed later in the chapter) that suggests that optimism and pessimism might not represent opposite poles of the same dimension, but actually might reflect two separate but related constructs. Use of separate terms in this context reflects more than a verbal convenience, but even here people vary in how optimistic or pessimistic they are. For now, we will treat optimism and pessimism as bipolar end points on a single dimension. This is how most of the researchers treated optimism and pessimism in the work that we review.

Effects of Optimism on Physical Health

Early Research on Optimism

Initial research on the benefits of optimism focused on relationships between optimism and subjective well-being. This research clearly demonstrated that optimists experience less distress when faced with various stressors, such as undergoing coronary artery bypass surgery (e.g., Scheier et al., 1989) or being diagnosed with cancer (e.g., Carver et al., 1993). Over time, researchers became increasingly interested in whether optimism might also predict better physical health outcomes. Early research on optimism and physical health focused on the effects of optimism on short-term health outcomes, as the main motivation was simply to test as efficiently as possible whether such relationships exist. These studies consistently found that optimists have better physical health than pessimists.

An example of this type of short-term health outcome is rehospitalization after coronary artery bypass surgery. Rehospitalization after bypass surgery is fairly common for a variety of reasons (e.g., wound infection, myocardial infarction, need for subsequent surgery), but it is quite costly in terms of healthcare expenditures and patients' well-being. Scheier et al. (1999) investigated whether optimists might respond better to surgery and be less likely than pessimists to need rehospitalization. To test this idea, they administered a measure of optimism to over 300 men and women who were scheduled to undergo bypass surgery. Six months later, they found that optimists were less likely to have been rehospitalized.

Larger Epidemiological Studies

More recently, a number of epidemiological studies have substantially extended the research linking optimism and physical health. Typically, these studies enroll samples of initially healthy

participants and measure the incidence (number of new cases) of disease over many years. Long periods of follow-up time are necessary, as many diseases, such as cardiovascular disease and cancer, take years and even decades to develop. Furthermore, because most people do not develop the disease, studies have to recruit large numbers of participants in order to have adequate statistical power to detect associations. Participant numbers in these studies have ranged from the 1,000s to upward of 100,000.

One of the largest epidemiological studies to investigate the effects of optimism was conducted by Tindle et al. (2009). They administered a measure of optimism to over 95,000 initially healthy Caucasian and African American women. Eight years later, they found that optimists were less likely to have developed coronary heart disease and were less likely to have died from any cause, as compared with pessimists.

Pathways to Cardiovascular Disease

Research on disease incidence and mortality strongly suggests that optimism plays a role in susceptibility to cardiovascular diseases, such as coronary heart disease and stroke. Research on various biological markers further supports this idea, demonstrating that optimism is related to markers that may represent physiological pathways to the development of future cardiovascular diseases.

One physiological pathway implicated in a number of cardiovascular diseases is atherosclerosis, or the development of plaque, which narrows and hardens the arteries. Atherosclerosis is a progressive condition, normally increasing with age. Matthews et al. (2004) investigated whether optimism might slow this progression. They followed a sample of middle-aged women for 10–13 years, measuring the intima-media thickness of the carotid artery as a metric of atherosclerosis. They found that optimists had a slower progression of atherosclerosis. In fact, the individuals highest in optimism showed almost no progression of atherosclerosis at all.

Another important risk factor for cardiovascular disease is high blood pressure, which can damage the heart and the arteries over time. Several studies have shown that pessimists have higher systolic and diastolic blood pressure throughout the day (e.g., Räikkönen & Matthews, 2008) and tend to have larger blood pressure responses to stressors (e.g., Terrill, Ruiz, & Garofalo, 2010).

In addition to atherosclerosis and blood pressure, optimism has also been linked to a number of other risk factors for cardiovascular disease such as obesity, cholesterol, and triglyceride levels (Boehm, Williams, Rimm, Ryff, & Kubzansky, 2013b), as well as metabolic syndrome, which is a cluster of conditions (i.e., high blood pressure, high blood sugar levels, excess abdominal fat, and high cholesterol levels) that put an individual at risk for cardiovascular diseases and diabetes (Cohen, Panguluri, Na, & Whooley, 2010).

Non-cardiovascular Disease Outcomes and Immune Function

Although optimism has shown strong associations with cardiovascular outcomes, evidence linking optimism to other diseases, particularly cancer and HIV, has been more equivocal. Some studies have found that optimism is related to lower mortality in patients with cancer (Allison, Guichard, & Gilain, 2000) and slower disease progression in patients with HIV (e.g., Ironson et al., 2005). However, other studies have not found that optimism predicts cancer and HIV-related outcomes (e.g., Tindle et al., 2009), and a recent meta-analysis failed to find a significant association between optimism and cancer (Rasmussen, Scheier, & Greenhouse, 2009).

The inconsistent relationship between optimism and cancer/HIV outcomes may be partially attributable to the nuanced relationship between optimism and various aspects of immunity. The immune system consists of multiple interacting components, and psychological factors can have positive and negative effects on different aspects of the immune system. These effects can further be moderated by specific contexts and situations.

In terms of the positive effects of optimism on immune outcomes, multiple studies have demonstrated that optimism is associated with lower levels of circulating pro-inflammatory cytokines and indicators of the inflammatory process, such as interleukin-6, homocysteine, fibrinogen, and intercellular adhesion molecule 1 (e.g., Roy et al., 2010). High levels of circulating inflammatory markers are considered an index of systemic or chronic low-grade inflammation. Such systemic inflammation is thought to be involved in a number of diseases, including cardiovascular disease, arthritis, and diabetes (e.g., Libby & Theroux, 2005). Thus, chronic inflammation may be another biological pathway by which optimism influences the development and progression of cardiovascular diseases, as well as other diseases.

In contrast to the effects on inflammation, optimism has shown more qualified relationships with indicators of cell-mediated immunity (e.g., circulating levels of T cells, natural killer cell cytotoxicity, delayed-type hypersensitivity skin test). Cell-mediated immunity is particularly important for combating viruses that enter human cells, such as the HIV virus, and for the removal of cells that have become cancerous. Research suggests that optimism may be related to better cell-mediated immunity under some conditions, namely, when individuals face brief, controllable stressors. Optimism may be related to diminished levels of cell-mediated immunity, though, when people face more prolonged and uncontrollable stressors (for a review, see Segerstrom, 2005).

One of the first studies to identify this qualified relationship was conducted by Cohen et al. (1999). They followed a sample of young women for 3 months, assessing the experience of stressors weekly. Among women who experienced only a brief stressor, lasting 1 week or less, optimism was associated with higher levels of circulating T cells. The opposite pattern was found among women who experienced a stressor for multiple weeks. For these women, optimism was associated with lower levels of circulating T cells and lower natural killer cell cytotoxicity.

Explanations for this pattern have varied. Some authors have argued that optimists are particularly vulnerable to disappointment when their positive expectations are disconfirmed (Tennen & Affleck, 1987). This disappointment could then explain the negative relationship between optimism and immunity when stressors prove difficult to resolve or control (e.g., Cohen et al., 1999). Although possible, this explanation is inconsistent with a large body of research suggesting that optimists experience less distress when they encounter negative outcomes, for example, when they are diagnosed with breast cancer (Carver et al., 1993).

Segerstrom and colleagues proposed an alternative explanation for the negative relationship between optimism and immune response in some circumstances (Segerstrom, Castañeda, & Spencer, 2003). Specifically, they argued that optimists are more likely to engage actively in trying to resolve stressors and to persist in these efforts despite difficulties. During a prolonged stressor, this persistence strategy may have costs reflected in a sustained physiological stress response and reduced cell-mediated immunity. However, this strategy likely has long-term benefits in terms of eventual stressor resolution. Pessimists, who are more likely to give up dealing with a stressor, may not incur these physiological costs during a prolonged stressor, but they may also be less likely to deal with the stressor adaptively.

Subsequent research has generally supported this second explanation (Segerstrom et al., 2003). For example, Solberg Nes, Segerstrom, and Sephton (2005) gave optimists a difficult

task while monitoring physiological indicators of stress. Optimists were more likely to persist at the task, but this persistence was associated with higher levels of cortisol and greater skin conductance.

If the negative effects of optimism on immunity are attributable to greater persistence, more research will still be needed to determine whether this association explains the inconsistent relationship between optimism and cancer/HIV outcomes. Similarly, more research will be needed to demonstrate which of the multiple plausible physiological pathways actually underlie the effect of optimism on cardiovascular outcomes. In the future, research will likely need to move beyond simply demonstrating associations between optimism and physical health. Instead, research should focus on documenting how optimism influences specific physiological pathways, which ultimately result in the development and progression of specific diseases.

Another fruitful area for future research will be to investigate the effect of optimism on a wider array of physical health outcomes. The effects of optimism on various physiological pathways (e.g., metabolic syndrome, inflammatory markers) strongly indicate that optimism contributes to other diseases. New research is beginning to support this idea. Initial research has found that optimism may play a role in diabetes management, being associated with better glycemic control among individuals with diabetes (Brody, Kogan, Murry, Chen, & Brown, 2008). Additionally, studies have shown that optimism is related to several pregnancy-related outcomes, such as birthweight and gestational age (Lobel, DeVincent, Kaminer, & Meyer, 2000), as well as in vitro fertilization success (Bleil et al., 2012).

Remaining Questions

Psychological and Behavioral Mechanisms

We have discussed several physiological pathways that might underlie the relationship between optimism and specific disease outcomes. What these physiological pathways do not explain is how optimism exerts physiological effects in the first place. In this regard, optimism has a number of effects on behavioral and psychological factors, which may account for the relationship between optimism and health. However, little research has actually tested whether these effects mediate the association between optimism and health.

One of the most likely reasons optimists experience better health is they engage in more positive health behaviors, while pessimists tend to engage in health-damaging behaviors. A number of studies have shown that optimists have a better diet (e.g., more fruits and vegetables), are more physically active, and are more likely to consume alcohol in moderation (e.g., Steptoe, Wright, Kunz-Ebrecht, & Iliffe, 2006). In contrast, pessimists are more likely to be smokers (e.g., Steptoe et al., 2006) and to suffer from substance abuse problems (e.g., Carvajal, Garner, & Evans, 1998). Few studies have formally tested whether these health behaviors mediate the relationship between optimism and physical health, but some evidence suggests that these health behaviors have physiological effects. Boehm, Williams, Rimm, Ryff, and Kubzansky (2013a) found that the tendency of optimists to eat a better diet and smoke less had a beneficial impact on their serum levels of antioxidants (see also Steptoe et al., 2006; Tindle et al., 2009).

The tendency of optimists to engage in proactive efforts to maintain their health is thought to be a manifestation of a broader tendency to engage in active, approach-oriented coping strategies. A number of studies have found that optimists utilize coping strategies that directly address the stressor (e.g., seeking information about the problem) or directly alter cognitions about the

stressor (e.g., positively reframing a stressful situation). Pessimists, in contrast, utilize avoidance coping strategies such as denying that a problem exists (for a meta-analytic review of the different coping strategies used by optimists and pessimists, see Solberg Nes & Segerstrom, 2006).

These coping strategies have a pervasive influence on how optimists and pessimists experience stressors. As mentioned previously, optimists experience less distress and higher levels of subjective well-being when confronted with a variety of stressors, perhaps especially so for those that are acute in nature. Decades of research have shown that stress impairs physical health, while subjective well-being is associated with better health and longevity (e.g., Diener & Chan, 2011). The tendency of optimists to engage in adaptive methods of coping that minimize distress may ultimately contribute to their physical health as well.

Another potential mechanism concerns the effects of optimism on social relationships. Optimism is associated with greater social network size and to ties with others that cross age, educational, and racial boundaries (Andersson, 2012). Optimists are also more satisfied with their relationships and report greater social support than pessimists (e.g., Assad, Donnellan, & Conger, 2007). As both social network size and perceived social support predict physical health (for review see Cohen, 2004), these factors may also contribute to the relationship between optimism and health.

Dimensionality of Optimism

Throughout this chapter, we have been referring to optimism as a single bipolar dimension ranging from optimism to pessimism. Many people working in the field construe optimism and pessimism in this fashion and analyze their studies accordingly, but as noted earlier on, researchers have also explored the possibility that optimism and pessimism may be somewhat distinct constructs. This view is consistent with the fact that factor analysis of generalized optimism scales often yields two separate components—one measuring expectancies for positive outcomes and one measuring expectancies for negative outcomes. Psychometrically, it remains unclear whether the distinction between optimism and pessimism is meaningful or merely an artifact of how optimism is measured.

Recent research on physical health has contributed to the discussion of dimensionality, suggesting that when analyzed separately, optimism and pessimism may have differential effects on physical health outcomes. In fact, most of the research linking optimism to inflammatory outcomes suggests that it is mainly pessimism driving this effect, not optimism (e.g., Roy et al., 2010). Pessimism was also found to be a stronger predictor than optimism of in vitro fertilization success (Bleil et al., 2012). These findings raise the possibility that the detrimental impact of pessimism on health is stronger than any salutary effects of optimism, but much more research is needed to answer this question definitively.

Conclusion

A substantial body of research suggests that optimism has a pervasive influence on physical health. The effects of optimism have been observed on short-term health outcomes, such as rehospitalization after surgery, and long-term health outcomes, such as mortality. This research clearly demonstrates that optimism is related to the development and progression of cardiovascular diseases, as well as a number of plausible physiological pathways to the development of these diseases. Optimism has shown a less consistent relationship with HIV/cancer outcomes, which may be due to the qualified relationship between optimism and immunity.

More recent research has begun to investigate a wider array of physical health outcomes as well. Several questions remain concerning the relationship between optimism and physical health, including what psychological and behavioral factors allow optimism to create health-related physiological responses, as well as how best to conceptualize optimism. As time passes, the answers to these and other questions are likely to be resolved. Research on optimism and physical health has flourished for decades. There is no reason to expect that the tide of research will recede anytime soon.

Acknowledgment

Preparation of this chapter was facilitated by support from the National Center for Complementary and Alternative Medicine (AT007262) and the Templeton Foundation. We would like to thank Nabila Fahin Jahan for reading an earlier draft of the paper.

Author Biographies

Maria G. Mens is a graduate student at Carnegie Mellon University. She is interested in the health effects of optimism and pessimism and the processes that underlie a sense of purpose in life and the links between purpose in life and well-being.

Michael F. Scheier is a professor of psychology at Carnegie Mellon University. His work falls at the intersection of personality, social, and health psychology. He does research on the associations between personality and health, including optimism and pessimism, as well as work on coping with stress, adjustment to chronic disease, and goal adjustment processes and well-being.

Charles S. Carver is a professor of psychology at the University of Miami. His work at various times has addressed topics in personality, social psychology, health psychology, and experimental psychopathology. His current research mostly examines individual differences in impulsiveness versus constraint, how those differences are produced, and what their consequences are for effective self-regulation.

References

- Allison, P. J., Guichard, C., & Gilain, L. (2000). A prospective investigation of dispositional optimism as a predictor of health-related quality of life in head and neck cancer patients. *Quality of Life Research*, 9, 951–960. <http://dx.doi.org/10.1023/A:1008931906253>
- Andersson, M. A. (2012). Dispositional optimism and the emergence of social network diversity. *The Sociological Quarterly*, 53(1), 92–115. <http://dx.doi.org/10.1111/j.1533-8525.2011.01227.x>
- Assad, K. K., Donnellan, M. B., & Conger, R. D. (2007). Optimism: An enduring resource for romantic relationships. *Journal of Personality and Social Psychology*, 93(2), 285–297. <http://dx.doi.org/10.1037/0022-3514.93.2.285>
- Bleil, M. E., Pasch, L. A., Gregorich, S. E., Millstein, S. G., Katz, P. P., & Adler, N. E. (2012). Fertility treatment response: Is it better to be more optimistic or less pessimistic? *Psychosomatic Medicine*, 74(2), 193–199. <http://dx.doi.org/10.1097/PSY.0b013e318242096b>
- Boehm, J. K., Williams, D. R., Rimm, E. B., Ryff, C., & Kubzansky, L. D. (2013a). The association between optimism and serum antioxidants in the Midlife in the United States Study. *Psychosomatic Medicine*, 75(1), 2–10. <http://dx.doi.org/10.1097/PSY.0b013e31827c08a9>

- Boehm, J. K., Williams, D. R., Rimm, E. B., Ryff, C., & Kubzansky, L. D. (2013b). Relation between optimism and lipids in midlife. *The American Journal of Cardiology*, 111(10), 1425–1431. <http://dx.doi.org/10.1016/j.amjcard.2013.01.292>
- Brody, G. H., Kogan, S. M., Murry, V. M., Chen, Y. F., & Brown, A. C. (2008). Psychological functioning, support for self-management, and glycemic control among rural African American adults with diabetes mellitus type 2. *Health Psychology*, 27(1S), S83–S90. <http://dx.doi.org/10.1037/0278-6133.27.1.S83>
- Carvajal, S. C., Garner, R. L., & Evans, R. I. (1998). Dispositional optimism as a protective factor in resisting HIV exposure in sexually active inner-city minority adolescents. *Journal of Applied Social Psychology*, 28(23), 2196–2211. <http://dx.doi.org/10.1111/j.1559-1816.1998.tb01367.x>
- Carver, C. S., Pozo, C., Harris, S. D., Noriega, V., Scheier, M. F., Robinson, D. S., ... Clark, K. C. (1993). How coping mediates the effect of optimism on distress: A study of women with early stage breast cancer. *Journal of Personality and Social Psychology*, 65(2), 375–390. <http://dx.doi.org/10.1037/0022-3514.65.2.375>
- Cohen, B. E., Panguluri, P., Na, B., & Whooley, M. A. (2010). Psychological risk factors and the metabolic syndrome in patients with coronary heart disease: Findings from the Heart and Soul Study. *Psychiatry Research*, 175(1-2), 133–137.
- Cohen, F., Kearney, K. A., Zegans, L. S., Kemeny, M. E., Neuhaus, J. M., & Stites, D. P. (1999). Differential immune system changes with acute and persistent stress for optimists vs pessimists. *Brain, Behavior, and Immunity*, 13(2), 155–174. <http://dx.doi.org/10.1006/brbi.1998.0531>
- Cohen, S. (2004). Social relationships and health. *American Psychologist*, 59(8), 676–684. <http://dx.doi.org/10.1037/0003-066X.59.8.676>
- Diener, E., & Chan, M. Y. (2011). Happy people live longer: Subjective well-being contributes to health and longevity. *Applied Psychology: Health and Well-Being*, 3(1), 1–43. <http://dx.doi.org/10.1111/j.1758-0854.2010.01045.x>
- Ironson, G., Balbin, E., Stuetzle, R., Fletcher, M. A., O'Cleirigh, C., Laurenceau, J.-P., ... Solomon, G. (2005). Dispositional optimism and the mechanisms by which it predicts slower disease progression in HIV: Proactive behavior, avoidant coping, and depression. *International Journal of Behavioral Medicine*, 12(2), 86–97. http://dx.doi.org/10.1207/s15327558ijbm1202_6
- Libby, P., & Theroux, P. (2005). Pathophysiology of coronary artery disease. *Circulation*, 111(25), 3481–3488. <http://dx.doi.org/10.1161/CIRCULATIONAHA.105.537878>
- Lobel, M., DeVincent, C. J., Kaminer, A., & Meyer, B. A. (2000). The impact of prenatal maternal stress and optimistic disposition on birth outcomes in medically high-risk women. *Health Psychology*, 19(6), 544–553. <http://dx.doi.org/10.1037/0278-6133.19.6.544>
- Matthews, K. A., Raikonen, K., Sutton-Tyrrell, K., & Kuller, L. H. (2004). Optimistic attitudes protect against progression of carotid atherosclerosis in healthy middle-aged women. *Psychosomatic Medicine*, 66(5), 640–644. <http://dx.doi.org/10.1097/01.psy.0000139999.99756.a5>
- Räikkönen, K., & Matthews, K. A. (2008). Do dispositional pessimism and optimism predict ambulatory blood pressure during schooldays and nights in adolescents? *Journal of Personality*, 76(3), 605–630. <http://dx.doi.org/10.1111/j.1467-6494.2008.00498.x>
- Rasmussen, H. N., Scheier, M. F., & Greenhouse, J. B. (2009). Optimism and physical health: A meta-analytic review. *Annals of Behavioral Medicine*, 37(3), 239–256. <http://dx.doi.org/10.1007/s12160-009-9111-x>
- Roy, B., Diez-Roux, A. V., Seeman, T., Ranjit, N., Shea, S., & Cushman, M. (2010). The association of optimism and pessimism with inflammation and hemostasis in the Multi-Ethnic Study of Atherosclerosis (MESA). *Psychosomatic Medicine*, 72(2), 134–140. <http://dx.doi.org/10.1097/PSY.0b013e3181cb981b>
- Scheier, M. F., Carver, C. S., & Bridges, M. W. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A re-evaluation of the Life Orientation Test. *Journal of Personality and Social Psychology*, 67, 1063–1078.
- Scheier, M. F., Matthews, K. A., Owens, J. F., Magovern, G. J., Lefebvre, R. C., Abbott, R. A., & Carver, C. S. (1989). Dispositional optimism and recovery from coronary artery bypass surgery:

- The beneficial effects on physical and psychological well-being. *Journal of Personality and Social Psychology*, 57(6), 1024–1040. <http://dx.doi.org/10.1037/0022-3514.57.6.1024>
- Scheier, M. F., Matthews, K. A., Owens, J. F., Schulz, R., Bridges, M. W., Magovern, G., Sr., & Carver, C. S. (1999). Optimism and re-hospitalization following coronary artery bypass graft surgery. *Archives of Internal Medicine*, 159(8), 829–835. <http://dx.doi.org/10.1001/archinte.159.8.829>
- Segerstrom, S. C. (2005). Optimism and immunity: Do positive thoughts always lead to positive effects? *Brain, Behavior, and Immunity*, 19(3), 195–200. <http://dx.doi.org/10.1016/j.bbi.2004.08.003>
- Segerstrom, S. C., Castañeda, J. O., & Spencer, T. E. (2003). Optimism effects on cellular immunity: Testing the affective and persistence models. *Personality and Individual Differences*, 35(7), 1615–1624. [http://dx.doi.org/10.1016/S0191-8869\(02\)00384-7](http://dx.doi.org/10.1016/S0191-8869(02)00384-7)
- Solberg Nes, L., & Segerstrom, S. C. (2006). Dispositional optimism and coping: A meta-analytic review. *Personality and Social Psychology Review*, 10(3), 235–251. http://dx.doi.org/10.1207/s15327957pspr1003_3
- Solberg Nes, L., Segerstrom, S. C., & Sephton, S. E. (2005). Engagement and arousal: Optimism's effects during a brief stressor. *Personality and Social Psychology Bulletin*, 31(1), 111–120. <http://dx.doi.org/10.1177/0146167204271319>
- Steptoe, A., Wright, C., Kunz-Ebrecht, S. R., & Iliffe, S. (2006). Dispositional optimism and health behavior in community-dwelling older people: Associations with healthy ageing. *British Journal of Health Psychology*, 11(1), 71–84. <http://dx.doi.org/10.1348/135910705X42850>
- Tennen, H., & Affleck, G. (1987). The costs and benefits of optimistic explanations and dispositional optimism. *Journal of Personality*, 55(2), 377–392. <http://dx.doi.org/10.1111/j.1467-6494.1987.tb00443.x>
- Terrill, A. L., Ruiz, J. M., & Garofalo, J. P. (2010). Look on the bright side: Do the benefits of optimism depend on the social nature of the stressor? *Journal of Behavioral Medicine*, 33(5), 399–414. <http://dx.doi.org/10.1007/s10865-010-9268-6>
- Tindle, H. A., Chang, Y. F., Kuller, L. H., Manson, J. E., Robinson, J. G., Rosal, M. C., ... Matthews, K. A. (2009). Optimism, cynical hostility, and incident coronary heart disease and mortality in the Women's Health Initiative. *Circulation*, 120(8), 656–662. <http://dx.doi.org/10.1161/CIRCULATIONAHA.108.827642>

Suggested Reading

- Carver, C. S., & Scheier, M. F. (2014). Dispositional optimism. *Trends in Cognitive Sciences*, 18, 293–299. doi:10.1016/j.tics.2014.02.003
- Carver, C. S., & Scheier, M. F. (2017). Optimism, coping, and well-being. In C. Cooper & J. C. Quick (Eds.), *Wiley handbook of stress and health: A guide to research and practice* (pp. 401–414). Chichester, UK: Wiley-Blackwell.
- Scheier, M. F., Carver, C. S., & Armstrong, G. H. (2012). Behavioral self-regulation, health, and illness. In A. S. Baum, T. A. Revenson, & J. E. Singer (Eds.), *Handbook of health psychology* (2nd ed., pp. 79–98). New York, NY: Psychology Press.
- Scheier, M. F., Carver, C. S., & Bridges, M. W. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A re-evaluation of the Life Orientation Test. *Journal of Personality and Social Psychology*, 67, 1063–1078.
- Segerstrom, S. C., Carver, C. S., & Scheier, M. F. (2017). Optimism. In M. Eid & M. D. Robinson (Eds.), *The happy mind: Cognitive contributions to well-being* (pp. 195–214). New York, NY: Springer.

Patient Adherence

Kelly B. Haskard-Zolnierек and Briana Cobos

Department of Psychology, Texas State University, San Marcos, TX, USA

Introduction and Definition

The primary factor in achieving optimal health and the effectiveness of a medical treatment plan is determined by the patient's adherence toward their treatment regimen. Adherence is defined as the extent to which a patient's behavior corresponds with the agreed-upon treatment guidelines set forth by a healthcare provider. The terms "compliance" and "persistence" are essentially synonymous with the term "adherence"; however, adherence is preferred because compliance suggests that the patient is passively obeying the guidelines of the medical provider. Persistence describes the degree to which the patient follows the guidelines or behavior modifications recommended by a provider for a predetermined duration of time. Adherence includes various health-related behaviors such as taking medication, filling prescriptions, attending follow-up appointments, self-management of a chronic and/or acute disease, receiving immunizations, and/or modifying one's lifestyle behaviors. These behaviors are intended to provide optimal health when followed.

The terms "adherence" and "concordance" similarly describe a situation in which a treatment plan is negotiated and collectively determined between the patient and physician. The primary objective of concordance is to establish a therapeutic relationship between the patient and physician. However, "adherence" is the preferred term because it suggests an agreement between a patient and a healthcare provider, that is, negotiated, planned, and discussed with a collaborative and therapeutic effort to determine a treatment plan and/or behavior modifications aimed at providing optimal health for the patient.

In contrast, nonadherence constitutes the patient failing to follow the recommended treatment regimen. An individual can be nonadherent by not taking their medication, discontinuing their medications before the recommended time set forth by their healthcare provider, not taking their medication dosage correctly, and/or disregarding certain behavior modifications that are part of the treatment plan. Furthermore, an individual can be nonadherent by not filling a prescription, which is referred to as "nonfulfillment."

Intentional nonadherence is another form of nonadherence in which an individual actively decides not to follow a healthcare provider's treatment recommendations or guidelines. Within this type of nonadherence, the individual usually takes an active approach in deciding whether to adhere or not adhere to their treatment plan. Individuals' beliefs and knowledge of their health condition and treatment regimen play an important role in their decision. For example, the individual may actively decide to discontinue medication due to aversive symptoms subsiding.

Alternatively, an individual can be unintentionally nonadherent toward treatment. This type of nonadherence can occur when the individual forgets to take a medication or lacks knowledge or understanding about how to take medication or adhere to a treatment plan. This has less to do with the individual's belief or knowledge. Instead, this type of nonadherence is due to unforeseen circumstances, such as the complexity of a treatment plan causing the individual to forget certain behavior modifications or medication dosages. Therefore, nonadherence could ultimately lead the individual to not experience the benefits of their treatment plan and can further worsen an individual's well-being.

Rates of Nonadherence

Rates of nonadherence vary depending on regimen, disease, disease category (i.e., acute versus chronic), and other factors. On average, rates of nonadherence range from 25 to 50%, with greater rates of nonadherence in lifestyle change regimens (e.g., making major dietary changes or maintaining a consistent exercise regimen). Rates of nonadherence tend to be higher for more complex regimens such as those that require multiple doses per day or have more complicated dosing instructions, such as associated dietary restrictions. In addition, rates of nonadherence to chronic diseases can be greater due to the long-term nature of these regimens as compared with adherence to short-term regimens for acute diseases. A meta-analysis of 569 studies published from 1948 through 1998 reported the highest rates of adherence in HIV and cancer and the lowest rates in pulmonary diseases, diabetes, and sleep disorders (DiMatteo, 2004a). Other meta-analytic work has shown that across 116 studies, patients with serious illnesses such as end-stage renal disease or heart disease who are in poorer health (either objectively or according to self-ratings) are less adherent (DiMatteo, Haskard, & Williams, 2007). These variations in rates of adherence across diseases could be due to the complex nature of some disease regimens, other factors such as adverse side effects, and other restrictions on one's activities and lifestyle. This nonadherence could also be due to personal factors such as depression, cognitive deficits, and pessimism that can accompany serious illnesses.

Consequences of Nonadherence

There are various causes of nonadherence to treatment regimens. For example, contributing factors toward nonadherence can include the following: adverse medication side effects, a long duration of treatment, frequency of expected intake of medication, or the complexity of the treatment. Nonadherence is an important healthcare issue because of the associated financial, health, and other consequences. Patients who are nonadherent are more likely to utilize healthcare resources, thus increasing healthcare costs.

The economic burden of nonadherence in the United States is estimated to be between \$290 and \$300 billion dollars each year. This burden is caused by individuals who are unable

to modify health or lifestyle behaviors, patients unable to attend medical visits, secondary illnesses that arise due to uncontrolled chronic health conditions, and unnecessary hospitalizations or emergency room visits because of uncontrolled health conditions (DiMatteo, Haskard Zolnierak, & Martin, 2012). Nonadherence could also cause either additional economic burden in the US workforce by loss of productivity at work or an increased rate of employee absenteeism. Nonadherence has been associated with higher costs in various diseases, wherein patients face greater annual healthcare costs because of their nonadherence (Iuga & McGuire, 2014).

Conversely, increased rates of adherence provide economic benefits. An increase in adherence rates would reduce the use and cost of healthcare resources needed during relapses, emergencies, and/or hospitalizations that could have been prevented by the patient following their treatment regimen. Therefore, nonadherence represents a serious issue for healthcare professionals, patients, and the healthcare industry, in which optimal health outcomes are exclusively determined by the individual's adherence toward a treatment regimen.

Nonadherence toward a treatment regimen can have further adverse effects on an individual's health outcomes by causing additional health complications or secondary illnesses. The consequences of nonadherence on an individual's health vary based on the type of illness and the treatment regimen. For example, types of health conditions that require strict adherence toward their treatment regimen and medication include kidney transplantation, end-stage renal disease, and HIV. The primary cause of kidney transplant rejection for patients is nonadherence toward their immunosuppressant medication (Cukor, Rosenthal, Jindal, Brown, & Kimmel, 2009). Individuals who are nonadherent toward their medication are at an increased risk of rejecting their organ or experiencing kidney failure. Furthermore, for individuals with end-stage renal disease, adherence to hemodialysis regimens is crucial in order to decrease the individual's risk of earlier mortality or further complications. Individuals diagnosed with HIV face an increased rate of drug resistance to their antiretroviral medication if they are not strictly adherent toward their medication regimen as well. As a result, nonadherence causes an increased risk of mortality and future adverse effects such as faster progression of the virus.

Health conditions that are not solely dependent on medication have been shown to have suboptimal adherence rates. For example, treatment regimens for individuals with diabetes or primary hypertension usually include a complex treatment regimen that includes medication, as well as diet and lifestyle modifications in order to obtain optimal health. Nonadherence to treatment regimens for these health conditions can have an adverse effect on an individual's health over time. Individuals diagnosed with diabetes and are nonadherent can experience adverse complications such as ketoacidosis (diabetic coma), stroke, kidney disease, blindness, damage to the nerves, the development of heart disease, and an increased risk of early mortality. Furthermore, individuals diagnosed with primary hypertension have a treatment plan that usually consists of antihypertensive medication as well as lifestyle change. Nonadherence to antihypertensive medication and behavior change can lead to the development of heart disease, kidney damage, congestive heart failure, and stroke. Therefore, adherence to the agreed-upon treatment plan is critical to decrease an individual's risk of developing secondary illnesses or adverse complications and to increase the likelihood of optimal health outcomes.

Another consequence of nonadherence is erosion of the physician-patient relationship. Ideally, a patient and physician work together in a collaborative manner using open communication to reach the common goal of good health outcomes for the patient. If a patient does not admit to struggles with adherence, it may be difficult for the physician to trust the patient, and the quality of their relationship can be negatively affected.

Measurements of Adherence

There are multiple methods of measuring adherence to treatment, but unfortunately no “gold standard” measurement method exists. Numerous measurement approaches can be used based on the setting and context of the measurement (e.g., clinical settings versus research contexts). Measurement approaches are typically grouped into two categories: direct and indirect methods. Direct methods include direct observation of patients taking their medication and measurement of levels of the medication or metabolite in blood or urine. Direct approaches can be very accurate but may also be more invasive, expensive, and time consuming. Indirect measures include patient self-reports either via interview or questionnaire, family member reports, pill counts, pharmacy refill records, or electronic monitors (e.g., measurement via special pill bottle caps such as MEMScap™ that record the time and date each time a pill bottle is opened). Each of these measures has both benefits and drawbacks. Pharmacy refill records, for example, can be inaccurate because they do not directly show ingestion of medication or behavior such as incorrect timing of doses. Electronic monitors not only can be very accurate but also are a more expensive way to measure adherence and also do not indicate ingestion of medication. Although self-report measures can be subject to biases in memory or the desire to present oneself in the best possible light, they do tend to be the simplest, most common, and least expensive approach to adherence measurement. In general, a multimethod approach to measuring adherence is recommended.

Predictors of Nonadherence

Researchers have attempted to understand the factors that influence patient nonadherence and the factors that may promote adherence to treatment. Predictors of nonadherence have been grouped into several categories: patient-related factors, regimen-related factors, provider–patient interaction level factors, and healthcare system-related factors (Osterberg & Blaschke, 2005).

Patient-related factors that predict nonadherence include such elements as mental health, beliefs and attitudes, understanding, motivation, and social support. Patients with poor mental health (particularly depression) tend to be less adherent. Meta-analytic work reveals a strong relationship between nonadherence and depression across 31 studies of patients with chronic diseases (Grenard et al., 2011). This association may be due to a pessimistic attitude, forgetfulness, or lack of motivation that often accompanies depression. Furthermore, patients who do not believe in the benefit of their regimen or have negative attitudes toward it may be less adherent. Such patients make a conscious choice not to follow their treatment regimen, as they are not convinced that it will help them or alleviate their symptoms. In addition, patients who do not understand their regimen or how to follow it correctly may be unintentionally nonadherent. When patients first receive instructions on how to take medications, they may not understand the instructions due to poor health literacy (i.e., an inability or struggle to understand and process health-related information). Also, patients who are lacking in social support, particularly that who comes from a loving family, may also be less adherent (DiMatteo, 2004b). A patient’s support network may play many roles in promoting adherence, including giving reminders to the patient, encouraging the patient in their health-related goals, and providing tangible support such as driving the patient to medical appointments.

There are several regimen-related factors that may be associated with nonadherence. These include side effects, frequency of dosing, and complexity of the regimen. A review of 61

studies reported that in diseases such as diabetes and HIV, greater dose frequency and more complex regimens (e.g., more medications, special requirements associated with medication taking) are associated with poorer adherence (Ingersoll & Cohen, 2008).

Physician–patient communication is also associated with patient adherence. A meta-analysis of 106 studies (Zolnierek & DiMatteo, 2009) reported that patients of physicians who communicated effectively had 19% higher adherence. Mutual trust, open communication, sharing in the process of making medical decisions, and partnership are all interpersonal factors in the medical provider–patient relationship that are central to achievement of patient adherence.

Healthcare system factors may also play a role in nonadherence. Numerous studies have indicated that the cost of medications may predict nonadherence, as patients delay refills or take their medication less frequently due to cost issues (Briesacher, Gurwitz, & Soumerai, 2007).

The information–motivation–strategy model has been proposed to explain three basic factors that are central to achievement of adherence (DiMatteo et al., 2012). First, patients do not understand their regimen and how to follow it. They lack the information and understanding needed to adhere. Communication with their physician may have been ineffective, or they may have difficulty understanding the regimen due to poor health literacy or forgetting the instructions they were given. Second, patients may not be motivated and committed to adherence to their treatment regimen. This lack of motivation or commitment could be due to their beliefs about the treatment regimen or their negative attitudes about it or to a lack of support that encourages the patient's adherence. Third, patients may lack the resources and strategies to support adherence. These strategies could include tools to help them remember to take their medication or a plan for getting to the pharmacy for refills. Understanding the predictors of adherence is helpful in designing interventions to improve adherence.

Interventions to Improve Adherence

As discussed, adherence is influenced by multiple factors, and researchers have proposed a variety of interventions in an effort to reduce rates of nonadherence. Interventions have been proposed on multiple levels, including patient, provider, and system-related levels. For example, a policy-level intervention might involve reducing out-of-pocket costs of medications. Unfortunately, fewer than half of published interventions have been found to improve adherence (Haynes, Ackloo, Sahota, McDonald, & Yao, 2008; van Dulmen et al., 2007). Evidence suggests that multifaceted interventions that address multiple barriers to adherence may be most effective. Strategies that have been found to be effective in multicomponent interventions include more intensive communication with and counseling of patients, providing reminders, and providing closer follow-up (Haynes et al., 2008). A recent review of 182 randomized clinical trials of interventions to improve medication adherence reported that the most successful interventions were complex and personalized and involved several approaches to improving adherence (Nieuwlaat et al., 2014). Examples of these approaches included increased support from family members or personalized care, education, and communication from healthcare providers such as pharmacists.

Educational interventions with monitoring, support, and follow-up were most strongly associated with improvements in adherence in a systematic review (Viswanathan et al., 2012). These authors compared interventions in various categories of chronic disease and stratified by patient, provider, and system-level interventions. Specific examples of successful interventions included asthma self-management and case management interventions for depression

(Viswanathan et al., 2012). Other techniques that have been successful include reducing daily dosing frequency and providing reminders in the form of alarms or alerts, which can be helpful with the unintentional aspect of nonadherence.

In promoting patient adherence, it will continue to be important to be aware of and address barriers at the patient, provider, and system levels. In addition, future research should continue to study the most successful components of interventions and improve these interventions to enhance adherence and health outcomes.

Author Biographies

Kelly B. Haskard-Zolnieriek is an associate professor of psychology at Texas State University. Her research involves two main areas: patients' adherence to medical recommendations and medical visit communication in the provider–patient relationship. Her current research involves evaluating mobile health interventions to improve patient adherence. Dr. Haskard-Zolnieriek has coauthored a book on patient adherence, published in 2010 by Oxford University Press, and a 2009 meta-analysis of the relationship between physician communication skills and patient adherence.

Briana Cobos completed her master of arts degree at Texas State University in psychological research. Her general research area is in health psychology. Ms. Cobos' research interests include predictors of nonadherence in individuals diagnosed with both a mental health condition and a chronic illness. She has coauthored various papers with a focus in health psychology and mental health conditions.

References

- Briesacher, B. A., Gurwitz, J. H., & Soumerai, S. B. (2007). Patients at-risk for cost-related medication nonadherence: A review of the literature. *Journal of General Internal Medicine*, 22(6), 864–871. doi:10.1007/s11606-007-0180-x
- Cukor, D., Rosenthal, D., Jindal, R., Brown, C., & Kimmel, P. (2009). Depression is an important contributor to low medication adherence in hemodialyzed patients and transplant recipients. *Kidney International*, 75(11), 1223–1229. doi:10.1038/ki.2009.51
- DiMatteo, M. R. (2004a). Variations in patients' adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care*, 42(3), 200–209. doi:10.1097/01.mlr.0000114908.90348.f9
- DiMatteo, M. R. (2004b). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology*, 23(2), 207–218. doi:10.1037/0278-6133.23.2.207
- DiMatteo, M. R., Haskard, K. B., & Williams, S. L. (2007). Health beliefs, disease severity, and patient adherence: A meta-analysis. *Medical Care*, 45(6), 521–528.
- DiMatteo, M. R., Haskard Zolnieriek, K. B., & Martin, L. R. (2012). Improving patient adherence: A three-factor model to guide practice. *Health Psychology Review*, 6, 74–91. doi:10.1080/17437199.2010.537592
- Grenard, J. L., Munjas, B. A., Adams, J. L., Suttrop, M., Maglione, M., McGlynn, E. A., & Gellad, W. F. (2011). Depression and medication adherence in the treatment of chronic diseases in the United States: A meta-analysis. *Journal of General Internal Medicine*, 26(10), 1175–1182. doi:10.1007/s11606-011-1704-y
- Haynes, R. B., Ackloo, E., Sahota, N., McDonald, H. P., & Yao, X. (2008). Interventions for enhancing medication adherence. *Cochrane Database of Systematic Reviews*, (2), CD000011. doi:10.1002/14651858.CD000011.pub3

- Ingersoll, K. S., & Cohen, J. (2008). The impact of medication regimen factors on adherence to chronic treatment: A review of literature. *Journal of Behavioral Medicine*, 31(3), 213–224. doi:10.1007/s10865-007-9147-y
- Iuga, A. O., & McGuire, M. J. (2014). Adherence and health care costs. *Risk Management Healthcare Policy*, 7, 35–44. doi:10.2147/RMHP.S19801
- Nieuwlaat, R., Wilczynski, N., Navarro, T., Hobson, N., Jeffery, R., ... Haynes, R. B. (2014). Interventions for enhancing medication adherence. *Cochrane Database of Systematic Reviews*, (11), CD000011. doi:10.1002/14651858.CD000011.pub4
- Osterberg, L., & Blaschke, T. (2005). Adherence to medication. *New England Journal of Medicine*, 353(5), 487–497. doi:10.1056/NEJMr050100
- van Dulmen, S., Sluijs, E., van Dijk, L., de Ridder, D., Heerdink, R., & Bensing, J. (2007). Patient adherence to medical treatment: A review of reviews. *BMC Health Services Research*, 7, 55. doi:10.1186/1472-6963-7-55
- Viswanathan, M., Golin, C. E., Jones, C. D., Ashok, M., Blalock, S. J., Wines, R. C., et al. (2012). Interventions to improve adherence to self-administered medications for chronic diseases in the United States: A systematic review. *Annals of Internal Medicine*, 157(11), 785–795. doi:10.7326/0003-4819-157-11-201212040-00538
- Zolnieriek, K. B., & DiMatteo, M. R. (2009). Physician communication and patient adherence to treatment: A meta-analysis. *Medical Care*, 47(8), 826–834. doi:10.1097/MLR.0b013e31819a5acc

Suggested Reading

- Atreja, A., Bellam, N., & Levy, S. R. (2005). Strategies to enhance patient adherence: Making it simple. *Medscape General Medicine*, 7(1), 4.
- DiMatteo, M. R., Haskard Zolnieriek, K. B., & Martin, L. R. (2012). Improving patient adherence: A three-factor model to guide practice. *Health Psychology Review*, 6, 74–91.
- Martin, L. R., Williams, S. L., Haskard, K. B., & DiMatteo, M. R. (2005). The challenge of patient adherence. *Therapeutics and Clinical Risk Management*, 1(3), 189–199.
- Osterberg, L., & Blaschke, T. (2005). Adherence to medication. *New England Journal of Medicine*, 353(5), 487–497.

Patient Satisfaction

Summer L. Williams¹ and Tricia A. Miller²

¹*Department of Psychology, Westfield State University, Westfield, MA, USA*

²*Department of Psychology, University of California, Riverside, CA, USA*

Introduction

The field of patient satisfaction is extensive as an area of empirical research and covers many constructs and facets of the patient experience in medical care. Literature reviews and meta-analytic studies have explored patient satisfaction in relation to a multitude of external variables such as patient health outcomes and domains within the clinical care context. Many aspects of healthcare delivery have focused on the patient as a consumer of health services and likewise an essential evaluator of the quality of that healthcare experience, wherein the satisfaction of the patient becomes a critical element in the quest for improved healthcare delivery and health outcomes. Patients' perceptions of care have become increasingly more important and valued as key indicators of outcome measures. Health researchers are keen to the importance of patient satisfaction, and thus they are engaged in ongoing efforts toward quality patient satisfaction measurement and translating patient satisfaction findings to inform health policy and make system-level changes.

Assessment and Measurement of Patient Satisfaction

Patient satisfaction as a healthcare outcome is typically measured via self-report, in which patients report their preferences, expectations, and level of satisfaction with the medical care they received within a variety of domains, including access to and cost of care, empathy, competence, attention and information given by the provider, continuity of care, attention to psychosocial issues by the provider, and overall quality of care (Hall & Dornan, 1988).

Typically patients report their perceptions of care immediately after their visit or some time later by phone, mail, online survey, or an in-person interview. One of the most widely used measures of patient satisfaction is the Patient Satisfaction Questionnaire, developed by the

RAND corporation, to assess general or global satisfaction with care as well as patient satisfaction with a number of individual domains such as technical quality, interpersonal manner, communication, financial aspects, time spent with doctor, and accessibility and convenience (Ware, Snyder, & Wright, 1976). This 50-item measure has been shortened in subsequent versions to include 18 items, known as the Patient Satisfaction Questionnaire-18 (PSQ-18), for ease of use in healthcare settings (Marshall & Hays, 1994).

Later measures of patient satisfaction extended to include more detailed aspects of the medical interview and include the Medical Interview Satisfaction Scale (MISS-29) and refinements of it, developed for use in British general practice consultations (Meakin & Weinman, 2002; Wolf, Putnam, James, & Stiles, 1978). One of the most recent and widely used measures of patient satisfaction is the Consumer Assessment of Healthcare Providers and Systems—Clinician and Group Surveys (CG-CAHPS) developed by Dyer, Sorra, Smith, Cleary, and Hays (2012). This measure extends questions of patient satisfaction beyond the clinician level and into the healthcare system level by looking at aspects of care such as access.

One of the major early concerns with assessing patient satisfaction was the lack of standardization across studies in its measurement, with some measures yielding low reliability and the validity of the measuring instruments generally unknown. Additional challenges to measuring patient satisfaction include recent concerns over whether patients have the expertise, medical knowledge, and health literacy to provide an accurate assessment of their medical care (Manary, Boulding, Staelin, & Glickman, 2013) and questions as to the accuracy of patients' recall of their medical experiences (Aharony & Strasser, 1993).

Perhaps though one of the toughest challenges to measuring patient satisfaction has been concern over the high reported levels of satisfaction across studies (Williams, Coyle, & Healy, 1998). The concern lies in whether patients are accurately reporting their levels of satisfaction and, if so, what factors might be driving such high satisfaction scores across studies. Social psychological concepts have been applied to understanding high satisfaction scores. Cognitive consistency theory asserts that patients are likely to report satisfaction with their care as a way of justifying the time and effort they have spent seeking that care (LeVois, Nguyen, & Attkisson, 1981). Patients especially prone to this bias are those who continue their care despite adverse situations in the healthcare setting. It may be that dissatisfaction with care is only reported when the patient experiences an extreme negative event (Williams, 1994).

Key Research Findings in Patient Satisfaction

Due to the sheer number of empirical studies on patient satisfaction, the literature and findings can often be unclear. Meta-analytic studies within the area of patient satisfaction have become paramount in cutting through the diffuse nature of the literature in order to document the important role that patient satisfaction plays in the medical care forum and in the quality of care provided.

Early research on patient satisfaction suggested that it may be hard to discern patients' true attitudes toward their care because patients often fear unfavorable treatment in the future if their provider learns that they expressed dissatisfaction (e.g., through self-report) (Ley, 1972). In one of the first meta-analytic studies conducted addressing patient satisfaction, Hall and Dornan (1988) uncovered a bit of reluctance on the part of patients to evaluate their healthcare providers and to report on satisfaction with their medical care. The authors determined that patients' reluctance to report on satisfaction was related to patients' viewing their care as subpar in some regard yet not wanting to criticize their providers. Of the 221 studies included in the

meta-analysis, patients were overall more satisfied with their care when their physicians were still in training. This finding begs the question, what is it about physicians in training that makes their patients more satisfied? It is within the physician–patient relationship that the research has focused on correlates of patient satisfaction to gain a better understanding of the mediators involved; one key mediator of patient satisfaction is provider–patient communication.

The importance of physician–patient communication skills is now, more than ever, a central component in medical school curricula and residency training programs thanks to a multitude of research demonstrating that beneficial patient outcomes, including patient satisfaction, are linked to good communication skills in providers (Williams, Weinman, & Dale, 1998). Aspects of physician communication such as instrumental communication (i.e., task-directed skill) and discussion of psychosocial issues (i.e., socio-emotional communication) with the patient have been found to be most related to and predictive of patient satisfaction (Inui, Carter, Kukull, & Haigh, 1982; Roter, Hall, & Katz, 1987). Other communication barriers such as lack of warmth and friendliness from the provider, failure to take the patient’s concerns into account, unclear expectations and explanations, and ambiguous diagnosis and causation of illness have been found to decrease satisfaction (Jackson, Chamberlin, & Kroenke, 2001).

Additionally, providers who use medical jargon or fail to consider a patient’s level of health literacy may also be inadvertently contributing to their patient’s decreased satisfaction (Roter et al., 1997). Providers who take the time to educate their patients in a comprehensible manner will likely result in many positive clinical outcomes, including not only patient satisfaction but also reduced patient anxiety, patients’ enhanced ability to cope with symptoms, enhanced recovery after surgery, and increased patient adherence (Sitzia & Wood, 1997). It stands to reason that providers in training, who are practicing, developing, and honing their communication skills that involve the aforementioned aspects of communication, would produce more satisfied patients.

However, provider communication alone does not speak to the entire relationship between aspects of the care visit and patient satisfaction. Other studies have linked increased patient satisfaction to the sheer amount of time spent in the clinical encounter, specifically during the physical exam as opposed to the history-taking portion of the visit (Robbins et al., 1993). These physician practice behaviors have an impact on patient satisfaction that goes beyond just how “good” of a communicator the provider is. Patients have entered into more collaborative partnerships with their providers and expect discussion of health education and treatment effects, among other information on which they might base their future health behaviors and decisions (Robbins et al., 1993).

Other Correlates of Patient Satisfaction

Patient adherence to treatment guidelines is one health outcome that has been positively correlated with satisfaction (Jha, Orav, Zheng, & Epstein, 2008). This makes sense: a patient is more likely to adhere when a common understanding of treatment and self-care expectations has been reached, and this association likely goes back to effective provider communication and establishment of collaboration with the patient (Manary et al., 2013).

Other researchers have explored various patient demographics and health status (i.e., age, gender, socioeconomic, mental health, etc.) and their possible influence on a patient’s satisfaction. The most consistent determinant characteristic is age, such that older patients are generally more satisfied than younger patients (Hall & Dornan, 1990; Zahr, William, & El-Hadad, 1991). Part of the reasoning behind this finding may hinge upon the expectations of older and

younger patients. Cartwright and Anderson (1981) found that older patients expect less information from their physicians. With the advent of the Internet and accessibility of medical information, younger patients today might expect more information from their providers especially when dealing with cancer; younger patients may even be going into the interactions with more knowledge than older patients (Norum, Grev, Moen, Balteskard, & Holthe, 2003). A mediator of this finding might be the education level of the patient. Greater satisfaction is associated with lower levels of education (Hall & Dornan, 1990). On the other hand, demographics such as sex and race seem to reveal mixed results in relation to patient satisfaction. Pascoe and Attkisson (1983) demonstrated that US Whites, as a whole, tend to be more satisfied with their care than non-Whites, yet the authors indicate that the interaction of ethnicity and socioeconomic status can confuse the results. Hall, Irish, Roter, Ehrlich, and Miller (1994) reported that within routine medical care visits, lower satisfaction was associated with younger female physicians and the least amount of satisfaction was found among male patients who were examined by younger female physicians. Other findings show no significant relation with sex and race (Marple, Lucey, Kroenke, Wilder, & Lucas, 1997).

It is clear that the relationship between demographic variables and patient satisfaction is complex. Social and cultural issues in healthcare have pointed to the need for further examination of the various social inequalities involved in patient care and the recognition that patient distrust of providers, discrimination, mental health disparities, diversity in the clinical encounter, limited English proficiency, stereotypes, and bias operate within the bounds of the clinical encounter and very likely affect patient satisfaction in a multitude of ways (Smedley, Stith, & Nelson, 2003).

Emerging as another patient factor related to satisfaction is the health status, both mental and physical, of the patient. Jackson et al. (2001) report various studies that have shown psychological distress, depression, and personality disorders to be associated with lower levels of satisfaction. Often these psychological factors are comorbid with chronic physical illness (Linn & Greenfield, 1982). Providers may be quite unaware of the expectations patients bring with them to their visits and may likewise not be comfortable addressing patients' psychosocial concerns in general (Jackson et al., 2001; Roter et al., 1987).

Future Directions in the Patient Satisfaction Literature

Although quantitative studies have provided much in the way of concrete correlates and outcomes in the field of patient satisfaction research, there is an ever-pressing need for more qualitative research as well. In their review of the literature, Aharony and Strasser (1993) point to the void in studies looking at patients' qualitative comments about their care. The authors suggest that there is still much to be done in content analyzing the written remarks of patients and those verbalized during interviews and focus groups. Such qualitative research might bring about a better understanding of the cognitive and affective processes that a patient moves through in evaluating his/her care, as well as allowing for more ethnographically accurate and representative data on the patient experience (Aharony & Strasser, 1993).

There are also ways in which the methodology of patient satisfaction research could be improved. Moving from correlational designs to those that would allow for more methodological and statistical control is essential. The research paradigm should progress into theory-driven hypotheses, larger sample sizes, and factor analysis and regression models that would examine the direct and indirect impact of dependent variables (e.g., overall satisfaction; Aharony & Strasser, 1993).

To address the concern over patients being inaccurate reporters of their satisfaction, survey instruments should focus on the patient–provider interactions, as well as on the overall care team of the patient, and evaluate all interactions within that framework. This is where patient-reported measures seem to be the most credible (Manary et al., 2013).

Still the largest barrier remaining for this area of research is agreement on a common definition of “patient satisfaction.” The underpinnings of this research need cohesive grounding in theory in order to drive accurate and reliable data comparisons across studies (Manary et al., 2013).

Author Biographies

Summer Williams is an assistant professor of psychology at Westfield State University in Western Massachusetts. Her areas of research interest include health communication, patient satisfaction and adherence, health outcomes and health inequities, and social and psychological determinants of health. She has recently published chapters in *Assessment in Health Psychology* and the *Handbook of Health Behavior Change*.

Tricia A. Miller received her PhD in psychology from the University of California at Riverside. Her research focuses on the social psychological process of health and medical care delivery including patients’ health literacy. She also studies the effect of provider–patient communication on the promotion and maintenance of treatment adherence and patients’ overall health outcomes.

References

- Aharony, L., & Strasser, S. (1993). Patient satisfaction: What we know about and what we still need to explore. *Medical Care Review*, 50(1), 49–79.
- Cartwright, A., & Anderson, R. (1981). *General practice revisited*. London, UK: Tavistock.
- Dyer, N., Sorra, J. S., Smith, S. A., Cleary, P. D., & Hays, R. D. (2012). Psychometric properties of the Consumer Assessment of Healthcare Providers and Systems (CAHPS®) clinician and group adult visit survey. *Medical Care*, 50, S28–S34. doi:10.1097/MLR.0b013e31826cbc0d
- Hall, J. A., & Dornan, M. C. (1988). What patients like about their medical care and how often they are asked: A meta-analysis of the satisfaction literature. *Social Science and Medicine*, 27(9), 935–939. doi:10.1016/0277-9536(88)90284-5
- Hall, J. A., & Dornan, M. C. (1990). Patient sociodemographic characteristics as predictors of patient satisfaction with medical care. *Social Science and Medicine*, 6, 811–818. doi:10.1016/0277-9536(90)90205-7
- Hall, J. A., Irish, J. T., Roter, D. L., Ehrlich, C. M., & Miller, L. H. (1994). Satisfaction, gender, and communication in medical visits. *Medical Care*, 32, 1216–1231.
- Inui, T. S., Carter, W. B., Kukull, W. A., & Haigh, V. H. (1982). Outcome-based doctor-patient interaction analysis: I. Comparison of techniques. *Medical Care*, 20(6), 535–549.
- Jackson, J. L., Chamberlin, J., & Kroenke, K. (2001). Predictors of patient satisfaction. *Social Science and Medicine*, 52, 609–620. doi:10.1016/S0277-9536(00)00164-7
- Jha, A. K., Orav, E. J., Zheng, J., & Epstein, A. M. (2008). Patients’ perception of hospital care in the United States. *New England Journal of Medicine*, 359, 1921–1931. doi:10.1056/NEJMsa0804116
- LeVois, M., Nguyen, T. D., & Attkisson, C. C. (1981). Artifact in client satisfaction assessment experience in community mental health settings. *Evaluation and Program Planning*, 4, 139–150. doi:10.1016/0149-7189(81)90004-5

- Ley, P. (1972). Complaints made by hospital staff and patients: A review of the literature. *Bulletin of British Psychologists*, 25, 115–120.
- Linn, L. S., & Greenfield, S. (1982). Patient suffering and patient satisfaction among the chronically ill. *Medical Care*, 20, 425–431.
- Manary, M. P., Boulding, W., Staelin, R., & Glickman, S. W. (2013). The patient experience and health outcomes. *New England Journal of Medicine*, 368(3), 201–203. doi:10.1056/NEJMp1211775
- Marple, R., Lucey, C., Kroenke, K., Wilder, J., & Lucas, C. (1997). A prospective study of concerns and expectations in patients presenting with common symptoms. *Archives of Internal Medicine*, 157, 1482–1488.
- Marshall, G. N., & Hays, R. D. (1994). *The Patient Satisfaction Questionnaire short-form (PSQ-18)*. Santa Monica, CA: RAND.
- Meakin, R., & Weinman, J. (2002). The 'Medical Interview Satisfaction Scale' (MISS-21) adapted for British general practice. *Family Practice*, 19(3), 257–263. doi:10.1093/fampra/19.3.257
- Norum, J., Grev, A., Moen, M., Balteskard, L., & Holthe, K. (2003). Information and communication technology in oncology. Patients' and relatives' experiences and suggestions. *Supportive Care in Cancer*, 11(5), 286–293. doi:10.1007/s00520-002-0437-1
- Pascoe, G., & Attkisson, C. (1983). The evaluation ranking scale: A new methodology for assessing satisfaction. *Evaluation and Program Planning*, 6, 335–347. doi:10.1016/0149-7189(83)90013-7
- Robbins, J. A., Bertakis, K. D., Helms, L. J., Azari, R., Callahan, E. J., & Creten, D. A. (1993). The influence of physician practice behaviors on patient satisfaction. *Family Medicine*, 25(1), 17–20.
- Roter, D. L., Hall, J. A., & Katz, N. R. (1987). Relations between physicians' behaviors and analogue patients' satisfaction, recall, and impressions. *Medical Care*, 25(5), 437–451.
- Roter, D. L., Stewart, M., Putnam, S. M., Lipkin, M., Jr., Stiles, W., & Inui, T. S. (1997). Communication patterns of primary care physicians. *Journal of the American Medical Association*, 277, 350–356. doi:10.1001/jama.1997.03540280088045
- Sitzia, J., & Wood, N. (1997). Patient satisfaction: A review of issues and concepts. *Social Science and Medicine*, 45(12), 1829–1843. doi:10.1016/S0277-9536(97)00128-7
- Smedley, B. D., Stith, A. Y., & Nelson, A. R. (Eds.) (2003). *Unequal treatment: Confronting racial and ethnic disparities in healthcare*. Washington, DC: National Academy Press.
- Ware, J. E., Snyder, M. K., & Wright, W. R. (1976). *Development and validation of scales to measure patient satisfaction with medical care services*. Springfield, VA: National Technical Information Service.
- Williams, B. (1994). Patient satisfaction: A valid concept? *Social Science and Medicine*, 38, 509–516. doi:10.1016/0277-9536(94)90247-X
- Williams, B., Coyle, J., & Healy, D. (1998). The meaning of patient satisfaction: An explanation of high reported levels. *Social Science and Medicine*, 47(9), 1351–1359. doi:10.1016/S0277-9536(98)00213-5
- Williams, S., Weinman, J., & Dale, J. (1998). Doctor-patient communication and patient satisfaction: A review. *Family Practice*, 15(5), 480–492.
- Wolf, M. H., Putnam, S. M., James, S. A., & Stiles, W. B. (1978). The medical interview satisfaction scale: Development of a scale to measure patient perceptions of physician behavior. *Journal of Behavioral Medicine*, 1(4), 391–401.
- Zahr, L. K., William, S. G., & El-Hadad, A. (1991). Patient satisfaction with nursing care in Alexandria, Egypt. *International Journal of Nursing Studies*, 28, 337–342. doi:10.1016/0020-7489(91)90060-G

Suggested Reading

- Hall, J. A., & Dornan, M. C. (1988). What patients like about their medical care and how often they are asked: A meta-analysis of the satisfaction literature. *Social Science and Medicine*, 27(9), 935–939. doi:10.1016/0277-9536(88)90284-5

- Hall, J. A., Roter, D. L., & Katz, N. R. (1988). Meta-analysis of correlates of provider behavior in medical encounters. *Medical Care*, 26(7), 657–675.
- Haskard, K. B., Williams, S. L., DiMatteo, M. R., Rosenthal, R., White, M. K., & Goldstein, M. G. (2008). Physician and patient communication training in primary care: effects on participation and satisfaction. *Health Psychology*, 27(5), 513–522. doi:10.1037/0278-6133.27.5.513
- Sitzia, J., & Wood, N. (1997). Patient satisfaction: A review of issues and concepts. *Social Science and Medicine*, 45(12), 1829–1843. doi:10.1016/S0277-9536(97)00128-7
- Williams, S., Weinman, J., & Dale, J. (1998). Doctor-patient communication and patient satisfaction: A review. *Family Practice*, 15(5), 480–492.

Personality and Coping

Jamie M. Jacobs¹ and Charles S. Carver^{2,3}

¹*Department of Psychiatry, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA*

²*Department of Psychology, University of Miami, Coral Gables, FL, USA*

³*Center for Advanced Study in the Behavioral Sciences, Stanford University, Stanford, CA, USA*

How do people deal with threats and challenges pertaining to acute and chronic diseases? Answers to this question generally invoke the concept of coping: attempts to remove or diminish a stressor or limit its impact. Coping responses vary widely, based in part on the duration of the disease (acute vs. chronic), its severity and prognosis, and the person's overall interpretation, or appraisal, of the illness and its implications. Coping can also be affected by personality, such that a given stressor elicits different coping responses from different people. In this way, personality and situational coping both play roles in subsequent mental and physical health outcomes (Sharpe, Martin, & Roth, 2011; also see Kern & Friedman, 2011; Carver & Connor-Smith, 2010). This chapter outlines several aspects of personality and coping, describes how they relate to each other, and suggests how they may relate to outcomes in the context of physical health and illness.

Personality

The psychology of personality includes a very broad range of ideas (see Carver & Scheier, 2012), but two themes stand out. One is that personality incorporates some internal processes or dynamics that influence the person's thoughts, feelings, and actions. The other is that, despite having the same basic functions, people differ from one another. There are many viewpoints on the nature of the underlying systems and also on what individual differences are most important. Here we outline two viewpoints that have been influential in health psychology.

The Five-Factor Model

What is generally termed the five-factor model is a structural framework for describing individual differences. It places personality on five broad trait dimensions, commonly labeled extraversion, neuroticism, agreeableness, conscientiousness, and openness to experience (e.g., McCrae & John, 1992). Each person is assumed to occupy a position on each of the five traits, though that “position” is less a fixed point, perhaps, than a central tendency with variability around it (e.g., Fleeson, 2001). This model was not only developed mainly through studies of how people are described in natural language, but links have also developed between this view of personality and analyses of childhood temperament (e.g., Hambrick & McCord, 2010; McCrae et al., 2000).

Extraversion is a dimension of approach and engagement with incentives, as reflected in descriptors such as active, assertive, energetic, and enthusiastic (McCrae & John, 1992). Some views of extraversion also emphasize qualities of sociability (Ashton, Lee, & Paunonen, 2002), and virtually all views incorporate an element of positive affect (Ozer & Benet-Martinez, 2006).

Neuroticism is the tendency to respond to threat, frustration, and loss with negative emotions (McCrae & John, 1992). Individuals high in neuroticism are vulnerable to anxiety, moodiness, and general emotional distress (see Carver & Connor-Smith, 2010). Another term for neuroticism is emotional instability (McCrae & John, 1992).

Agreeableness refers to investment in establishing and maintaining social relationships (Jensen-Campbell & Graziano, 2001). Agreeableness facilitates compassion (Ozer & Benet-Martinez, 2006), and people high on this trait are more forgiving and trusting than those low on it (McCrae & John, 1992).

Conscientiousness concerns thoroughness and trustworthiness. It includes qualities such as planfulness, persistence, and organization (McCrae & John, 1992), competence, self-efficacy, and self-discipline (Bartley & Roesch, 2011). Conscientiousness implies being perseverant in goals and tasks (Ozer & Benet-Martinez, 2006). People with this trait act in ways that facilitate achievement striving (McCrae & John, 1992).

The final factor generally termed openness to experience incorporates qualities such as curiosity and imagination (McCrae & John, 1992). Persons who are high on openness are flexible and willing to involve themselves in new and atypical experiences (McCrae, 1996). Openness to experience often overlaps with qualities such as creativity and inspiration (Ozer & Benet-Martinez, 2006).

Optimism and Expectancy-Value Process Models

Another individual difference variable that figures prominently in the coping literature is optimism (see Carver, Scheier, & Segerstrom, 2010 for a review). Optimism, which does not fit neatly into the five-factor model, derives from a different research tradition, following from expectancy-value models of motivation. Optimism consists of a generalized favorable versus unfavorable expectancy for one's future. As is true of all expectancy-value models, this construct assumes that a favorable expectancy promotes greater motivation, resulting in greater persistence and generally better outcomes.

Optimism forms a lens, or cognitive filter, that influences life at multiple levels. It affects people's appraisals of challenging or stressful situations and their subsequent reactions (both behavioral and physiological) to those situations. Optimism is not the view that adversity will not occur; rather, it incorporates the belief that adversity and threat can be overcome.

(Fowler & Geers, 2015). Whether people are optimistic or pessimistic influences the extent to which they experience distress in the context of adversity, and it influences the manner in which people cope.

Optimism and the Five-Factor Model

Although standing somewhat apart from the five-factor model, optimism shares qualities with four of the five traits of that model. Optimism is positively associated with extraversion, agreeableness, and conscientiousness and negatively associated with neuroticism. The one factor with which optimism is not associated is openness to experience. In general, neuroticism and extraversion are the most correlated of the big five traits with optimism. However, optimism is not simply low neuroticism and high extraversion; rather, it reflects a more complex profile (Sharpe, Martin, & Roth, 2011).

Coping

Coping is generally defined as the cognitive and behavioral efforts made by an individual to manage or reduce external or internal demands associated with a stressor that seem to tax or exceed the individuals' resources (Lazarus & Folkman, 1987). Coping takes many forms, and a variety of distinctions have been made within the realm of coping. Coping may be cognitive or behavioral. It may be effective or ineffective. Some would say that coping consists of intentional use of strategies, but others would say that some coping consists of involuntary reactions. Several distinctions are elaborated upon in the following sections (for more detail, see Carver & Connor-Smith, 2010).

Problem-Focused Versus Emotion-Focused Coping

A distinction was made early in the literature on coping between problem-focused and emotion-focused coping (Lazarus & Folkman, 1987). Problem-focused coping comprises active efforts to remove the stressor or reduce its impact by engaging in such activities as problem solving, conflict resolution, information seeking, and decision making. Emotion-focused coping comprises a set of efforts to manage and reduce the emotional distress provoked by the stressor (see Gross, 2014). Problem-focused behaviors are most commonly used, and are thought to be more useful, in situations where the stressor is controllable. Emotion-focused coping is more common in situations of low controllability.

Emotion-focused coping is more diverse than problem-focused coping. Included in this category are activities as diverse as relaxation, meditation, music therapy, physical exercise, massage, cognitive restructuring, and humor. Also included in this category are avoidant strategies such as self-distraction, shopping, wishful thinking, and retreating into substance use. These responses are all intended to regulate stress-induced emotions.

Many emotion-focused responses are quite useful. Even distraction away from a threat can be useful in the short term, especially if a person has already taken suitable action. Some emotion-focused coping responses, however—particularly those with avoidant properties—are not effective in the long term and can have adverse consequences for longer-term health and well-being.

Problem-focused and emotion-focused coping categories are distinguishable in principle, but they often are used jointly (Lazarus, 2006). They can also facilitate one another. Although

problem-focused coping is aimed at removing the stressor or blunting its impact, a secondary consequence of effective problem-focused coping is a reduction in distress. Although emotion-focused coping is aimed at emotion regulation, the reduction in distress can also make it easier to turn to problem-focused coping.

Engagement Versus Disengagement Coping

A particularly important distinction is the one made between engagement and disengagement coping. Engagement coping, also known as approach coping, is geared toward dealing with the stressor and its emotional consequences (Skinner, Edge, Altman, & Sherwood, 2003). Disengagement coping involves trying to avoid or escape from dealing with the threat. Engagement coping encompasses both problem-focused and some of the emotion-focused techniques. Emotion-focused techniques that would be viewed as engagement coping would include support seeking, emotion regulation, acceptance of the reality of the challenge, and cognitive restructuring.

Disengagement techniques are avoidant in nature, including reactions such as denial, wishful thinking, and substance use. Often people use disengagement coping to distance themselves from the stressor. While this can be effective for avoiding distress in the short term, it does not address the situation that is producing the distress. More important, it could worsen long-term outcomes. For instance, a person with a cancer diagnosis who avoids scheduling an appointment for a scan may evade distress temporarily. But that evasion ultimately allows an opportunity for the cancer to progress. This may result in more aggressive treatment and greater overall stress and threat to life. Certain disengagement coping mechanisms can cause other problems, such as drugs, alcohol, and overeating. Disengagement coping may ultimately lead to an abandonment of the goals with which the stressor is interfering in order to avoid experiencing or exacerbating any associated negative emotions (Carver, Scheier, & Weintraub, 1989).

Proactive Coping

A final variation in the concept of coping we will note here is what is termed proactive coping. Proactive coping is anticipating and preparing for obstacles or stressful situations before they arise (Schwarzer & Taubert, 2002). Proactive coping is goal oriented and adaptive, and those who cope proactively are motivated to succeed. This tendency to expect and prepare for challenges plays a large role in the reduction of associated emotional distress and may be dictated by personality traits (Hambrick & McCord, 2010).

Personality and Coping

How do aspects of personality relate to aspects of coping? Personality is not the same as coping, but personality can influence what sorts of coping a person engages in, at several stages in the stress process. Most fundamentally, personality can influence how people appraise and interpret potential stressors. A person who perceives a great threat will turn to coping responses more quickly than a person who perceives a lesser threat. Since appraisals also influence choice of coping response, differences in appraisal affect what actions follow. People also vary in which coping responses come naturally to them. Thus, even given the same appraisal, people may cope in different ways.

The following sections address this issue from the perspective of the two views on personality that were described earlier in the chapter. First we consider the five-factor model.

Five-Factor Model and Coping

A recent meta-analysis of personality traits and coping (see Connor-Smith & Flachsbart, 2007) showed a number of associations between coping tendencies and traits from the five-factor model. For example, the trait of extraversion was fairly consistently related to use of problem-focused coping strategies and cognitive restructuring. These findings are consistent with the view that extraversion is a dimension of engagement with incentives and pursuit of goals.

In contrast to this, the meta-analysis found that neuroticism related inversely to problem solving and cognitive restructuring. Neuroticism also was positively associated with several maladaptive emotion-focused coping strategies, such as wishful thinking and withdrawal. On the positive side, neuroticism was also found to predict greater support seeking, which can be helpful in coping with health difficulties and chronic illness.

Interestingly, persons higher in neuroticism report greater emotional reactivity to negative life events and are more likely to develop anxiety and depression in response to such events (Lahey, 2009). Coping style probably plays a role in this relationship, given evidence that persons high in neuroticism use more disengagement-related strategies than problem-focused coping behaviors (Watson & Hubbard, 1996). Consistent with this pattern, people high in neuroticism also tend not to engage in proactive coping.

The trait of conscientiousness is associated with properties such as self-discipline, competence, self-efficacy, and striving toward achievement. Not surprisingly, then, it is strongly associated with the use of problem-focused, approach coping (Bartley & Roesch, 2011), including proactive coping. People high in conscientiousness also report greater positive affect. This relationship is likely mediated by the use of problem-focused coping in situations of daily stress (Bartley & Roesch, 2011).

Although the term proactive coping is generally reserved for cases of preparation for a particular stressor, there is a sense in which general efforts to live healthy lives represent proactive coping. Such health-promoting behaviors are an important part of managing well-being. Persons who are emotionally stable, introverted, and conscientious are more likely to engage in health-promoting behaviors such as physical activity, a healthy diet, immunizations, safe driving, sleep, weight control, and not smoking (Korotkov, 2008).

Optimism and Coping

Another personality dimension that has been linked to coping is optimism. Solberg Nes and Segerstrom (2006) conducted a meta-analysis of optimism effects. Optimism is systematically associated with greater use of approach coping tendencies, strategies that are active and problem focused. Optimism is also related to the use of social support, both for emotional reasons and for instrumental assistance. In contrast to these tendencies, pessimism is associated with avoidance strategies, essentially efforts to escape the stressor and sometimes to escape even acknowledging the stressor (e.g., Solberg Nes & Segerstrom, 2006).

As one example, in a sample of HIV-positive men and women coping with the loss of their partners to AIDS, optimism was positively associated with reports of using active coping strategies and negatively associated with reports of using avoidant coping strategies and falling into hopelessness (Rogers, Hanson, Levy, Tate, & Sikkema, 2005).

In a study of newly diagnosed breast cancer patients (Carver et al., 1993), optimism was associated with greater use of a range of engagement coping at time-appropriate points in treatment. That is, correlations of optimism with report of planning and active coping emerged prior to surgery but not afterward. Correlations with acceptance and use of humor were present across the first 6 months after treatment, and with positive reframing across the first 3 months. Optimism was also inversely associated with disengagement coping—denial and behavioral disengagement—across the full year after treatment.

Although similar effects have emerged in diverse contexts (Carver et al., 2010), there do appear to be boundaries on such effects of optimism on coping. Very recent research has investigated the role of what has been termed comparative optimism, which statistically is nearly independent of trait optimism as discussed here. Comparative optimism is assessed by items asking about the likelihood that respondents will experience problems in a given life domain compared with other people who are otherwise like them. Comparative optimists say those events are less likely than do other people. Trait optimists who were also comparative optimists minimized the degree of threat posed by particular health challenges; those who were trait optimists but not comparative optimists did not (Fowler & Geers, 2015).

Adjustment and Health

Coping, Adjustment, and the Role of Personality

Coping is associated with the health perceptions and quality of life of patients dealing with chronic illness. Across many diseases and health conditions, coping tendencies are associated with both mental and physical outcomes. There are two ways in which coping can have beneficial effects on well-being. First, it does so to the extent that it removes or eliminates the threat (thereby reducing distress). Second, it does so to the extent that it directly reduces distress.

Which of these classes of coping is more appropriate depends on the context. In the context of a controllable stressor, problem-focused behaviors such as active problem solving and changing one's circumstances are helpful (see Carver & Connor-Smith, 2010 in further readings). In the context of an uncontrollable stressor, these responses are less useful; emotion regulation, use of social support resources, and controlled emotional expression are most beneficial (Penley, Tomaka, & Wiebe, 2002). Reliance on an emotion-focused strategy in the context of a controllable stressor may be less productive, and vice versa. Behaviors that are either pointlessly ruminative or avoidant in nature—e.g., venting, denial, and self-blame—tend to predict poorer adjustment regardless of context (e.g., Moskowitz, Hult, Bussolari, & Acree, 2009; also see Austenfeld & Stanton, 2004 in further readings). This selection and matching of the most beneficial coping strategy (or strategies) to a given stressor is one way to think about how coping influences adjustment. Within this viewpoint, the role of personality is principally to influence choice of coping responses.

A second way in which personality may play a role here is to moderate the effect of coping, such that once a coping response is selected, personality may influence how well the individual implements that response and how well it works for that individual. For example, problem-solving behaviors may be more successfully implemented by people who are conscientious (see Carver & Connor-Smith, 2010 in further readings).

Coping, Personality, and Health

Regardless of whether the role of personality is to influence selection of coping response or to influence the execution of the response, there are at least two pathways by which coping *per se* may have an effect on health. One path involves the level of wear and tear created on physical systems as a result of ineffective coping with threats and challenges. There is little dispute over the fact that chronic, elevated stress can affect physical health and disease processes via a range of physiological pathways such as the hypothalamic–pituitary–adrenocortical axis and the sympathetic nervous system (e.g., Schneiderman, Ironson, & Siegel, 2005). Effective coping can dampen physiological stress responses, proactive coping can prevent stress responses altogether, and dysfunctional coping can worsen stress responses (Carver & Vargas, 2011). With repeated or prolonged physiological activation, people become physically exhausted and more susceptible to illness.

The key issue here may be the extent to which the stress is invoked repeatedly or chronically. Meta-analyses suggest that persons coping with conditions such as HIV, prostate cancer, and diabetes who implement engagement coping have better physical and mental health than those who do so to a lesser degree (e.g., Moskowitz et al., 2009). On the other hand, poorer physical health outcomes are observed among persons who opt for disengagement coping (Moskowitz et al., 2009; also see Austenfeld & Stanton, 2004).

A second pathway by which coping affects health is a behavioral mechanism. This pathway follows from the fact that some kinds of effective coping involve engaging in health-promoting behaviors and avoiding health-damaging behaviors. In this case, the effect on health is intrinsic to the behavior itself. This pathway, of course, is also influenced by personality. The likelihood of engaging in healthy lifestyle behaviors is greater among persons who are optimistic than pessimistic (e.g., Carver et al., 2010). Of the five-factor model, conscientiousness, agreeableness, and extraversion are also linked to health-promoting behaviors (e.g., Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007).

Concluding Comments

This review is by no means complete or comprehensive. Coping in a health context is a multi-dimensional process, influenced by the healthcare environment, family factors, and life changes, along with physical demands such as pain and treatment side effects. Variation in these demands illustrates how different coping responses may be required for various components of a stressor. In part for this reason, it is challenging to predict coping responses from personality dispositions alone.

There is much more to be said about coping and personality in the context of different health stressors, as well as the inevitable variation based on demographic differences such as age, gender, ethnicity, socioeconomic status, and stage of disease. More research is needed on the extent to which coping is stable across situations. The concept of proactive coping is another area in need of further investigation, for example, to understand whether proactive coping follows from particular personality profiles, or whether it develops over time in response to experiences of adversity. Finally, it would seem desirable to continue to hone programs to teach effective coping, thereby helping patients enhance their use of engagement coping techniques. Enhanced coping would, in turn, increase positive health behaviors and improve health outcomes from morbidity to mortality in the context of clinical settings.

Author Biographies

Dr. Jamie M. Jacobs is a clinical psychologist at the Massachusetts General Hospital Cancer Center and Behavioral Medicine Service and an Assistant Professor of Psychology in the Department of Psychiatry at Harvard Medical School. Her expertise includes effects of stress on health outcomes and the role of coping and stress management in buffering physiological stress and disease processes in the context of cancer. She is also interested in psychological factors that predict nonadherence to cancer treatment regimens, in order to guide intervention development to improve adherence and symptom management.

Charles S. Carver is distinguished professor of psychology at the University of Miami. His background is in personality and social psychology, but he has also been active in health psychology and experimental psychopathology. One aspect of his research has focused on coping processes and optimism as a personality disposition, in the context of major health threats.

References

- Ashton, M. C., Lee, K., & Paunonen, S. V. (2002). What is the central feature of extraversion? Social attention versus reward sensitivity. *Journal of Personality and Social Psychology*, 83, 245–252. doi:10.1037/0022-3514.83.1.245
- Austenfeld, J. L., & Stanton, A. L. (2004). Coping through emotional approach: A new look at emotion, coping, and health-related outcomes. *Journal of Personality*, 72, 1335–1364. doi:10.1111/j.1467-6494.2004.00299.x
- Bartley, C. E., & Roesch, S. C. (2011). Coping with daily stress: The role of conscientiousness. *Personality and Individual Differences*, 50, 79–83. doi:10.1016/j.paid.2010.08.027
- Carver, C., Scheier, M., & Segerstrom, S. (2010). Optimism. *Clinical Psychology Review*, 30, 879–889. doi:10.1016/j.cpr.2010.01.006
- Carver, C. S., & Connor-Smith, J. (2010). Personality and coping. *Annual Review of Psychology*, 61, 679–704. doi:10.1146/annurev.psych.093008.100352
- Carver, C. S., Pozo, C., Harris, S. D., Noriega, V., Scheier, M. F., Robinson, D. S., ... Clark, K. C. (1993). How coping mediates the effect of optimism on distress: A study of women with early stage breast cancer. *Journal of Personality and Social Psychology*, 65, 375–390. doi:10.1037/0022-3514.65.2.375
- Carver, C. S., & Scheier, M. F. (2012). Cybernetic control processes and the self-regulation of behavior. In R. M. Ryan (Ed.), *The Oxford handbook of human motivation* (pp. 28–42). New York, NY: Oxford University Press.
- Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, 56, 267–283. doi:10.1037/0022-3514.56.2.267
- Carver, C. S., & Vargas, S. (2011). Stress, coping, and health. In H. S. Friedman (Ed.), *The Oxford handbook of health psychology* (pp. 165–191). New York, NY: Oxford University Press.
- Connor-Smith, J. K., & Flachsbart, C. (2007). Relations between personality and coping: A meta-analysis. *Journal of Personality and Social Psychology*, 93, 1080–1107. doi:10.1037/0022-3514.93.6.1080
- Fleeson, W. (2001). Toward a structure- and process-integrated view of personality: Traits as density distributions of states. *Journal of Personality and Social Psychology*, 80, 1011–1027. doi:10.1037/0022-3514.80.6.1011
- Fowler, S. L., & Geers, A. L. (2015). Dispositional and comparative optimism interact to predict avoidance of a looming health threat. *Psychology & Health*, 30, 456–474. doi:10.1080/08870446.2014.977282
- Gross, J. J. (Ed.) (2014). *Handbook of emotion regulation* (2nd ed.). New York, NY: Guilford Press.

- Hambrick, E. P., & McCord, D. M. (2010). Proactive coping and its relation to the five-factor model of personality. *Individual Differences Research*, 8, 67–77.
- Jensen-Campbell, L. A., & Graziano, W. G. (2001). Agreeableness as a moderator of interpersonal conflict. *Journal of Personality*, 69, 323–362. doi:10.1111/1467-6494.00148
- Kern, M. L., & Friedman, H. S. (2011). Personality and pathways of influence on physical health. *Social and Personality Psychology Compass*, 5, 76–87. doi:10.1111/j.1751-9004.2010.00331.x
- Korotkov, D. (2008). Does personality moderate the relationship between stress and health behavior? Expanding the nomological network of the five-factor model. *Journal of Research in Personality*, 42, 1418–1426. doi:10.1016/j.jrp.2008.06.003
- Lahey, B. B. (2009). Public health significance of neuroticism. *American Psychologist*, 64, 241. doi:10.1037/a0015309
- Lazarus, R. S. (2006). Emotions and interpersonal relationships: Toward a person-centered conceptualization of emotions and coping. *Journal of Personality*, 74, 9–46. doi:10.1111/j.1467-6494.2005.00368.x
- Lazarus, R. S., & Folkman, S. (1987). Transactional theory and research on emotions and coping. *European Journal of Personality*, 1, 141–169. doi:10.1002/per.2410010304
- McCrae, R. R. (1996). Social consequences of experiential openness. *Psychological Bulletin*, 120, 323–337. doi:10.1037/0033-2909.120.3.323
- McCrae, R. R., Costa, P. T., Jr., Ostendorf, F., Angleitner, A., Hřebíčková, M., Avia, M. D., ... Smith, P. B. (2000). Nature over nurture: Temperament, personality, and life span development. *Journal of Personality and Social Psychology*, 78, 173–186. doi:10.1037/0022-3514.78.1.173
- McCrae, R. R., & John, O. P. (1992). An introduction to the five-factor model and its applications. *Journal of Personality*, 60, 175–215. doi:10.1111/j.1467-6494.1992.tb00970.x
- Moskowitz, J. T., Hult, J. R., Bussolari, C., & Acree, M. (2009). What works in coping with HIV? A meta-analysis with implications for coping with serious illness. *Psychological Bulletin*, 135, 121–141. doi:10.1037/a0014210
- Nes, L. S., & Segerstrom, S. C. (2006). Dispositional optimism and coping: A meta-analytic review. *Personality and Social Psychology Review*, 10, 235–251. doi:10.1207/s15327957pspr1003_3
- Ozer, D. J., & Benet-Martinez, V. (2006). Personality and the prediction of consequential outcomes. *Annual Review of Psychology*, 57, 401–421. doi:10.1146/annurev.psych.57.102904.190127
- Penley, J. A., Tomaka, J., & Wiebe, J. S. (2002). The association of coping to physical and psychological health outcomes: A meta-analytic review. *Journal of Behavioral Medicine*, 25, 551–603. doi:10.1023/A:1020641400589
- Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, 2, 313–345. doi:10.1111/j.1745-6916.2007.00047.x
- Rogers, M. E., Hanson, N. B., Levy, B. R., Tate, D. C., & Sikkema, K. J. (2005). Optimism and coping with loss in bereaved HIV-infected men and women. *Journal of Social and Clinical Psychology*, 24, 341–360.
- Schneiderman, N., Ironson, G., & Siegel, S. D. (2005). Stress and health: Psychological, behavioral, and biological determinants. *Annual Review of Clinical Psychology*, 1, 607–628. doi:10.1146/annurev.clinpsy.1.102803.144141
- Schwarzer, R., & Taubert, S. (2002). Tenacious goal pursuits and striving toward personal growth: Proactive coping. In E. Frydenberg (Ed.), *Beyond coping: Meeting goals, visions and challenges* (pp. 19–35). London: Oxford University Press.
- Sharpe, J. P., Martin, N. R., & Roth, K. A. (2011). Optimism and the big five factors of personality: Beyond neuroticism and extraversion. *Personality and Individual Differences*, 51, 946–951. doi:10.1016/j.paid.2011.07.033
- Skinner, E. A., Edge, K., Altman, J., & Sherwood, H. (2003). Searching for the structure of coping: A review and critique of category systems for classifying ways of coping. *Psychological Bulletin*, 129, 216–269. doi:10.1037/0033-2909.129.2.216

Watson, D., & Hubbard, B. (1996). Adaptational style and dispositional structure: Coping in the context of the five-factor model. *Journal of Personality*, 64, 737–774. doi:10.1111/j.1467-6494.1996.tb00943.x

Suggested Reading

Austenfeld, J. L., & Stanton, A. L. (2004). Coping through emotional approach: A new look at emotion, coping, and health-related outcomes. *Journal of Personality*, 72, 1335–1364. doi:10.1111/j.1467-6494.2004.00299.x

Carver, C. S., & Connor-Smith, J. (2010). Personality and coping. *Annual Review of Psychology*, 61, 679–704. doi:10.1146/annurev.psych.093008.100352

Carver, C. S., & Scheier, M. F. (2012). Cybernetic control processes and the self-regulation of behavior. In R. M. Ryan (Ed.), *The Oxford handbook of human motivation* (pp. 28–42). New York, NY: Oxford University Press.

Connor-Smith, J. K., & Flachsbart, C. (2007). Relations between personality and coping: A meta-analysis. *Journal of Personality and Social Psychology*, 93, 1080–1107. doi:10.1037/0022-3514.93.6.1080

Kern, M. L., & Friedman, H. S. (2011). Personality and pathways of influence on physical health. *Social and Personality Psychology Compass*, 5, 76–87. doi:10.1111/j.1751-9004.2010.00331.x

Personality and Health Behaviors

Christopher S. Nave^{1,2} and Michelle R. Dixon²

¹*Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA*

²*Department of Psychology, Rutgers University Camden, Camden, NJ, USA*

Introduction

Health behaviors are positive or negative in valence and are associated with an individual's overall health. There are two types of health behaviors: health-promoting behaviors and health-compromising behaviors. Health-promoting behaviors include all behaviors that enhance or promote one's health (Sarafino & Smith, 2011). Health-promoting behaviors are considered beneficial for individuals to engage in and are frequently recommended by physicians, dietitians, and nutritionists in addition to being used in health behavior interventions. Examples of health-promoting behaviors include maintaining a healthy diet, exercising on a regular basis, and engaging in preventative health habits. Preventative health habits include wearing sunscreen daily, regularly visiting the doctors, drinking adequate amounts of water, and reducing salt intake (Sarafino & Smith, 2011). Individuals who engage in health-promoting behaviors can enhance one's quality of life, increase confidence, decrease illness, and even increase lifespan.

Health-compromising behaviors are harmful or detrimental to one's health, are considered destructive for people to engage in, and should be eliminated from daily life or minimized as much as possible (Sarafino & Smith, 2011). Examples of health-compromising behaviors include smoking tobacco, eating foods high in sugar, using illegal drugs, engaging in excessive alcohol use or binge drinking, and participating in risky sexual behavior. There are many factors that can influence health behaviors. One influence on health behaviors is personality.

Personality is described as a pattern of thought, emotions, and behavior that each individual possesses, while personality traits are any individual difference variable that can be used to predict behavior. Personality traits are a key predictor of important life outcomes like divorce, occupational attainment, mortality at comparable or even greater levels than IQ, and socioeconomic status (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). Personality traits show a remarkable amount of stability throughout the lifespan (Ferguson, 2010) though there are opportunities for personality change and growth (Srivastava, John, Gosling, & Potter, 2003).

A thorough, systematic lexical and empirical approach to studying personality traits has yielded five broad factors, known as the Big Five (McCrae & Costa, 1987). The Big Five includes extraversion, agreeableness, conscientiousness, neuroticism and openness, or intellect/culture (McCrae & Costa, 1987). Extraversion refers to individuals who are outgoing, talkative, and social. Agreeableness refers to individuals who are tolerant and kind. Conscientiousness refers to individuals who are highly organized and achievement orientated. Neuroticism refers to individuals who are anxious and moody, though many researchers study the positive end of this trait and call it emotional stability. Lastly, openness refers to individuals who are open to new experiences and curious. Other researchers (e.g., Goldberg, 1990) believe the fifth factor is best described as intellect/culture, with more emphasis on cognitive ability and IQ.

The Big Five and Health Behaviors

An abundance of research has demonstrated reliable associations between personality traits and health behaviors, many of which that can be organized around the Big Five. Support exists for a health behavior model of personality, where specific health behaviors have increasingly been demonstrated to mediate the relationship between personality and health outcomes (Hampson, Edmonds, Goldberg, Dubanoski, & Hillier, 2015). The following subsections break down a sampling of what we know about personality and its relationship with health behaviors.

Conscientiousness

Conscientious individuals are those who show self-discipline, plan their behaviors, aim for achievement, and practice impulse control. A variety of meta-analyses, large longitudinal studies, surveys, and experimental studies convincingly show that conscientiousness is related to a large number of health-promoting and health-compromising behaviors (Bogg & Roberts, 2013). Previous research has shown that conscientiousness is related to individuals maintaining a healthy diet by consistently eating foods like fruits, vegetables, lean meats, and whole grains and avoiding foods that are high in sugar and unhealthy fats (Bogg & Roberts, 2004). Engaging in frequent exercise is related to conscientiousness although the relationship is not very strong. In a meta-analysis that looked at conscientiousness and activity levels, the results showed that activity level was least related to conscientiousness in comparison with the other health behaviors, though conscientious individuals tend to possess better fitness levels and lower body mass indices than those who are low in conscientiousness (Bogg & Roberts, 2004). Another health-promoting behavior conscientious people engage in is more periodic health screening and greater attendance at scheduled doctor appointments (Armon & Toker, 2013). In addition to going to doctors more often, conscientious people are also more likely to engage in medication adherence, an effect that nearly doubles in magnitude when examining younger conscientious individuals (Molloy, O'Carroll, & Ferguson, 2014). Medication adherence is a particularly important health-promoting behavior because medication is not effective if the patient is not taking it.

Conscientiousness is also related to individuals avoiding health-compromising behaviors. People who are high in conscientiousness are less likely to smoke cigarettes or use drugs (Bogg & Roberts, 2004). Even conscientious ratings of children assessed by elementary school teachers predict low or no usage of tobacco later in life (Hampson, Goldberg, Vogt, & Dubanoski, 2006). Conscientious individuals are also less likely to consume alcohol to excess (Bogg &

Roberts, 2013) and are unlikely to engage in risky driving as measured by number of accidents, driving violations, speeding tickets, DUIs, and not using seatbelts (Bogg & Roberts, 2004; Raynor & Levine, 2009). Individuals who are high in conscientiousness tend not to engage in risky sexual behaviors (e.g., not using contraceptives or having a large number of sexual partners) and tend to not participate in behaviors that lead to incarceration, conviction, or detention (Bogg & Roberts, 2004). A seminal meta-analysis on conscientiousness and health behaviors found that out of all the healthy behaviors tested, frequency in alcohol consumption and driving while under the influence of alcohol were the most negatively correlated with conscientiousness (Bogg & Roberts, 2004). In summary, conscientiousness is related to the engagement in almost all health-promoting behaviors and the nonengagement in nearly all health-compromising behaviors. People who are high in conscientiousness are more likely to practice healthy behaviors and avoid unhealthy behaviors.

Extraversion

Extraverted individuals tend to be energetic and assertive and take pleasure in talking with others. Findings for how extraversion and sociability influence health behaviors are unclear, particularly with respect to health-promoting behaviors. One area where there is a consistent pattern of results for health-promoting behaviors is in coping with stressful life events. Extraverted individuals tend to manage stress better than introverted individuals and use a variety of coping mechanisms to manage stress (DeLongis & Holtzman, 2005; McCrae & Costa, 1986).

Extraversion is related to a number of health-compromising behaviors. In two large longitudinal studies, individuals high in extraversion tend to drink more at midlife and participate in excessive alcohol use such as binge drinking and frequent drinking (Hampson et al., 2006). Other research shows that those high in extraversion are less likely to limit consumption of alcoholic drinks or use designated drivers (Raynor & Levine, 2009). Tobacco use has also been shown to be moderately positively associated with extraversion (Raynor & Levine, 2009). One possible explanation for the relationships between extraversion and alcohol and tobacco use may be because highly extraverted people are social and enjoy going to social gatherings that involve alcohol or tobacco use. In a large survey study examining college students, extraversion was the strongest predictor of risky sexual practices including number of sexual partners and not using condoms in the last 30 days. Extraverted college students also slept less compared with those lower in extraversion (Raynor & Levine, 2009). Those high in extraversion tend to get less sleep than those who are low in extraversion, perhaps due to their social life and various campus activities (Raynor & Levine, 2009). Highly extraverted people are also less likely to participate in periodic health screenings (Armon & Toker, 2013). Lack of health screenings is a health-compromising behavior because regular health checkups allow individuals to prevent illness rather than treat illness after it has already been contracted. Overall, the evidence for extraversion shows the trait to be related to engaging in various health-compromising behaviors.

Agreeableness

Individuals who are high in agreeableness are those who tend to trust others, show kindness, and express affection. Agreeableness is related to a number of different health behaviors. Agreeableness is related to possessing a high body mass index (Sutin, Ferrucci, Zonderman, & Terracciano, 2011). Body mass index is a measurement that primary care physicians use to determine if an individual's weight is healthy or not. Individuals who have a higher body mass

index than normal are considered overweight (Sarafino & Smith, 2011). An individual becomes overweight by engaging in health-compromising behaviors such as not maintaining a healthy diet or regularly exercising. One study found that people high in agreeableness are slightly more likely to use drugs and alcohol (Turiano, Whiteman, Hampson, Roberts, & Mroczek, 2012). In a large survey study of college students, agreeableness was not related to alcohol or tobacco use but was slightly negatively correlated to binge drinking and number of sexual partners (Raynor & Levine, 2009). Another study found that agreeableness is related to the avoidance of risky driving (Booth-Kewley & Vickers, 1994), which makes conceptual sense given that agreeableness is the opposite of hostility and aggression. Overall, agreeableness is related to engagement in health-promoting behaviors and avoiding health-compromising behaviors. Research is unclear on the relationship between agreeableness and substance use. More research on agreeableness needs to be done in order to find conclusive evidence with respect to health-promoting and health-compromising behaviors.

Openness and Culture/Intellect

Openness refers to an individual's ability to be highly imaginative and insightful. Out of all the Big Five personality traits, openness seems to be the least related to health behaviors. There are, however, a few health behaviors that previous research has found to be related to openness. Those who are highly open tend to practice healthy eating habits. Conversely, individuals high in openness are less likely to participate in periodic health screenings (Armon & Toker, 2013). One study found that substance use is related to openness (Booth-Kewley & Vickers, 1994). Another study involving college students found that openness was not related to any health behaviors (Raynor & Levine, 2009). The relationships between openness and the above stated health behaviors are very weak relationships. One explanation for the slight correlations may be due to openness being related to creativity. Creative people may be more open to unique healthy foods, holistic medicine, and recreational drug use such as marijuana. Nevertheless the relationship between openness and health behaviors is not very strong.

Other researchers consider intellect, not openness, to be a major factor of the Big Five (e.g., Goldberg, 1990). Research examining childhood IQ has shown associations between lower IQ and smoking later in adulthood (Hart et al., 2004) and higher IQ and smoking cessation in midlife (Taylor et al., 2003). Other research from a prospective longitudinal study shows that highly intelligent individuals often engage in many of the same unhealthy behaviors compared with individuals of average intelligence (Friedman & Markey, 2003).

Neuroticism and Emotional Stability

Neuroticism refers to an individual's anxiety levels, emotional instability, and sadness. Neuroticism is only related to health-compromising behaviors. Individuals high in neuroticism have been shown to be more likely to engage in substance use (Turiano et al., 2012). One reason for this relationship could be that the anxiety neurotic people experience influences their substance use. Highly neurotic individuals are not only more likely to smoke tobacco, but they are also more inclined to smoke large quantities of cigarettes (Booth-Kewley & Vickers, 1994). When neuroticism is paired with being conscientious, however, research suggests that this combination, described as healthy neuroticism, predicts lower levels of smoking behavior (Weston & Jackson, 2015). One study found that low emotional stability (i.e., neuroticism) is related to alcohol use when combined with high levels of extraversion. This study found that children who rated high in emotional instability and extraversion predicted greater

alcohol use 40 years later (Hampson et al., 2006). The relationship between alcohol use and neuroticism only occurs when combined with extraversion. Another study of college students found no relationship between neuroticism and alcohol use (Raynor & Levine, 2009). Highly neurotic individuals are also more likely to experience fluctuations with their weight (Sutin et al., 2011). This relationship may be related to the conception that people who are emotionally unstable may be more likely to eat unhealthy foods as a coping mechanism. Weight fluctuations are considered unhealthy because it usually means that the individuals are not consistently engaging in health-promoting behaviors related to eating and exercising. Neurotics demonstrate higher levels of daily stress and negative affect (Mroczek & Almeida, 2004). Hostility, a subcomponent to neuroticism, has been related to smoking, insufficient physical activity, and overeating (Niaura et al., 2000). Finally, those who are very high or very low in neuroticism are less likely to participate in periodic health screening. Individuals that fall right in the middle on the neuroticism scale are more likely to participate in periodic health screening (Armon & Toker, 2013). Overall, neuroticism has a wide-ranging set of associations with health-promoting behaviors (negative associations) and with compromising behaviors.

Other Personality Traits

Although the Big Five encompasses a variety of personality traits, there are personality and individual variables not captured by the Big Five that are related to health behaviors. In the Dunedin birth cohort study, high negative emotionality and low constraint at age 18 predicted various health risk behaviors (i.e., unsafe sexual behavior, alcohol dependence, dangerous driving habits, violence conviction) at age 21 (Caspi et al., 1997). Honesty-humility is one of six factors of the HEXACO model of personality and refers to an individual who is honest, loyal, and sincere (Ashton & Lee, 2009). One study found that honesty-humility was a significant negative predictor of exercise habits such that those who rated lower in honesty-humility reported more frequent physical exercise (MacCann, Todd, Mullan, & Roberts, 2015). Other traits related to health habits include perfectionism, which is the tendency to strive for flawlessness. One study found that perfectionism is related to engaging in preventative health behaviors (Williams & Cropley, 2014). Dispositional optimism is related to less smoking and alcohol use and more physical activity among older participants (Steptoe, Wright, Kunz-Ebrecht, & Iliffe, 2006). Finally, aggression has been associated with smoking habits such that individuals who tend to express hostility also tend to smoke more (Welch & Poulton, 2009).

Conclusion

Research has shown that personality traits, particularly the Big Five, are in fact predictors of health behaviors. The association between personality and health behaviors does not mean that it is guaranteed that an individual with a certain personality trait will definitely engage in a specific health behavior. It simply means that the individual is more inclined to behave a certain way if they possess a specific personality trait. Recent health behavior models look to use personality and health behaviors to help understand important health outcomes like general health, cardiovascular risk, and mortality (Hampson et al., 2015). Some correlations between health behaviors and personality traits are strong although many are weak. Health behaviors are influenced by a number of diverse factors, with personality as just one of these factors. With that being said, it is likely important to consider personality when implementing health interventions. People with varying levels of a personality trait may mean that a “one size

fits all” for altering health behaviors is inadequate. Individuals may find it difficult to change their personality, but they can certainly take their personality into consideration when trying to change their health behaviors. As such, knowing associations between personality and health behaviors could improve intervention-based research by allowing researchers to tailor programs better designed for those with certain personality characteristics. For example, group-based therapies to change behavior (e.g., diet, weight, addiction) may be better suited to patients high in extraversion and sociability compared with highly introverted or highly neurotic individuals. Therapies that involve complicated adherence strategies would likely benefit from including participants with high conscientiousness compared with those with low conscientiousness. Future research is needed to understand and unpack the processes and mechanisms behind associations of personality and health behaviors (Hampson, 2012). Understanding the relationship between personality and health behaviors is important because health behaviors (e.g., smoking, drinking, wearing a seatbelt) can be analyzed as the mechanism by which personality is related to many important health outcomes (e.g., lung cancer, alcoholism, longevity; Hampson, 2012). Personality traits seem to be important individual difference variables to help understand *who* engages in particular health behaviors, which in turn leads to particular health outcomes (e.g., conscientious individuals wear seatbelts more, which in turn may help predict why conscientious people live longer, on average, than individuals low in conscientiousness). As such, it is important for researchers to continue to examine how personality relates to a variety of health-promoting and health-compromising behaviors.

Author Biographies

Christopher S. Nave, PhD, Associate Director, Master of Behavioral and Decision Sciences, University of Pennsylvania. Major research interests include examining associations between personality, health, and behavior across the lifespan, using multi-method approaches to studying individual differences, person perception and impression management, and situational assessment.

Michelle R. Dixon, MA Student, Rutgers University, Camden. Interests include health psychology and the relationships between health, personality, and behavior.

References

- Armon, G., & Toker, S. (2013). The role of personality in predicting repeat participation in periodic health screening. *Journal of Personality*, 81, 452–464. doi:10.1111/jopy.12021
- Ashton, M. C., & Lee, K. (2009). The HEXACO-60: A short measure of the major dimensions of personality. *Journal of Personality Assessment*, 91, 340–345. doi:10.1080/00223890902935878
- Bogg, T., & Roberts, B. W. (2004). Conscientiousness and health-related behaviors: A meta-analysis of the leading behavioral contributors to mortality. *Psychological Bulletin*, 130, 887–919. doi:10.1037/0033-2909.130.6.887
- Bogg, T., & Roberts, B. W. (2013). The case for conscientiousness: Evidence and implications for a personality trait marker of health and longevity. *Annals of Behavioral Medicine*, 45, 278–288. doi:10.1007/s12160-012-9454-6
- Booth-Kewley, S., & Vickers, R. R., Jr. (1994). Associations between major domains of personality and health behavior. *Journal of Personality*, 62, 281–298. doi:10.1111/j.1467-6494.1994.tb00298.x
- Caspi, A., Begg, D., Dickson, N., Harrington, H., Langley, J., Moffitt, T. E., & Silva, P. A. (1997). Personality differences predict health-risk behaviors in young adulthood: Evidence from a

- longitudinal study. *Journal of Personality and Social Psychology*, 73, 1052–1063. doi:10.1037/0022-3514.73.5.1052
- DeLongis, A., & Holtzman, S. (2005). Coping in context: The role of stress, social support, and personality in coping. *Journal of Personality*, 73, 1–24. doi:10.1111/j.1467-6494.2005.00361.x
- Ferguson, C. J. (2010). A meta-analysis of normal and disordered personality across the life span. *Journal of Personality and Social Psychology*, 98, 659–667. doi:10.1037/a0018770
- Friedman, H. S., & Markey, C. N. (2003). Paths to longevity in the highly intelligent Terman cohort. In C. E. Finch, J.-M. Robine, & Y. Christen (Eds.), *Brain and longevity* (pp. 165–175). New York: Springer.
- Goldberg, L. R. (1990). An alternative “description of personality”: The big-five factor structure. *Journal of Personality and Social Psychology*, 59, 1216–1229. doi:10.1037/0022-3514.59.6.1216
- Hampson, S. E. (2012). Personality processes: Mechanisms by which personality traits “get outside the skin”. *Annual Review of Psychology*, 63, 315–339. doi:10.1037/0022-3514.59.6.1216
- Hampson, S. E., Edmonds, G. W., Goldberg, L. R., Dubanoski, J. P., & Hillier, T. A. (2015). A life-span behavioral mechanism relating childhood conscientiousness to adult clinical health. *Health Psychology*, 34, 887–895. doi:10.1037/hea0000209
- Hampson, S. E., Goldberg, L. R., Vogt, T. M., & Dubanoski, J. P. (2006). Forty years on: Teachers’ assessments of children’s personality traits predict self-reported health behaviors and outcomes at midlife. *Health Psychology*, 25, 57–64. doi:10.1037/0278-6133.25.1.57
- Hart, C. L., Taylor, M. D., Davey Smith, G., Whalley, L. J., Starr, J. M., Hole, D. J., ... Deary, I. J. (2004). Childhood IQ and cardiovascular disease in adulthood: Prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies. *Social Science and Medicine*, 59, 2131–2138. doi:10.1016/j.socscimed.2004.03.016
- MacCann, C., Todd, J., Mullan, B. A., & Roberts, R. D. (2015). Can personality bridge the intention-behavior gap to predict who will exercise? *American Journal of Health Behavior*, 39, 140–147. doi:10.5993/AJHB.39.1.15
- McCrae, R. R., & Costa, P. T., Jr. (1986). Personality, coping, and coping effectiveness in an adult sample. *Journal of Personality*, 54, 385–405. doi:10.1111/j.1467-6494.1986.tb00401.x
- McCrae, R. R., & Costa, P. T., Jr. (1987). Validation of the five-factor model of personality across instruments and observers. *Journal of Personality and Social Psychology*, 52, 81–90. doi:10.1037/0022-3514.52.1.81
- Molloy, G. J., O’Carroll, R. E., & Ferguson, E. (2014). Conscientiousness and medication adherence: A meta-analysis. *Annals of Behavioral Medicine*, 47, 92–101. doi:10.1007/s12160-013-9524-4
- Mroczek, D. K., & Almeida, D. M. (2004). The effect of daily stress, personality, and age on daily negative affect. *Journal of Personality*, 72, 355–378. doi:10.1111/j.0022-3506.2004.00265.x
- Niaura, R., Banks, S. M., Ward, K. D., Stoney, C. M., Spiro, A., III, Aldwin, C. M., ... Weiss, S. T. (2000). Hostility and the metabolic syndrome in older males: The normative aging study. *Psychosomatic Medicine*, 62, 7–16.
- Raynor, D. A., & Levine, H. (2009). Associations between the five-factor model of personality and health behaviors among college students. *Journal of American College Health*, 58, 73–82. doi:10.3200/JACH.58.1.73-82
- Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, 2(4), 313–345.
- Sarafino, E., & Smith, T. (2011). *Health psychology: Biopsychosocial interactions* (7th ed.). New York, NY: Wiley.
- Srivastava, S., John, O. P., Gosling, S. D., & Potter, J. (2003). Development of personality in early and middle adulthood: Set like plaster or persistent change? *Journal of Personality and Social Psychology*, 84, 1041–1053. doi:10.1037/0022-3514.84.5.1041
- Stephoe, A., Wright, C., Kunz-Ebrecht, S. R., & Iliffe, S. (2006). Dispositional optimism and health behavior in community-dwelling older people: Associations with health ageing. *British Journal of Health Psychology*, 11, 71–84. doi:10.1348/135910705X42850

- Sutin, A. R., Ferrucci, L., Zonderman, A. B., & Terracciano, A. (2011). Personality and obesity across the adult life span. *Journal of Personality and Social Psychology*, 101, 579–592. doi:10.1037/a0024286
- Taylor, M. D., Hart, C. L., Davey Smith, G., Starr, J. M., Hole, D. J., Whalley, L. J., ... Deary, I. J. (2003). Childhood mental ability and smoking cessation in adulthood: Prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies. *Journal of Epidemiology and Community Health*, 57, 464–465. doi:10.1136/jech.57.6.464
- Turiano, N. A., Whiteman, S. D., Hampson, S. E., Roberts, B. W., & Mroczek, D. K. (2012). Personality and substance use in midlife: Conscientiousness as a moderator and the effects of trait change. *Journal of Research in Personality*, 46, 295–305. doi:10.1016/j.jrp.2012.02.009
- Welch, D., & Poulton, R. (2009). Personality influences on change in smoking behavior. *Health Psychology*, 28, 292–299. doi:10.1037/a0013471
- Weston, S. J., & Jackson, J. J. (2015). Identification of the healthy neurotic: Personality traits predict smoking after disease onset. *Journal of Research in Personality*, 54, 61–69. doi:10.1016/j.jrp.2014.04.008
- Williams, C. J., & Cropley, M. (2014). The relationship between perfectionism and engagement in preventive health behaviours: The mediating role of self-concealment. *Journal of Health Psychology*, 19, 1211–1221. doi:10.1177/1359105313488971

Suggested Reading

- Bogg, T., & Roberts, B. W. (2013). The case for conscientiousness: Evidence and implications for a personality trait marker of health and longevity. *Annals of Behavioral Medicine*, 45, 278–288. doi:10.1007/s12160-012-9454-6
- Friedman, H. S., & Kern, M. L. (2014). Personality, well-being, and health. *Annual Review of Psychology*, 65, 719–742. doi:10.1146/annurev-psych-010213-115123
- Hampson, S. E., & Friedman, H. S. (2008). Personality and health: A lifespan perspective. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 770–794). New York, NY: Guilford.

Personality and Health Outcomes

Dietlinde Heilmayr¹ and Howard S. Friedman²

¹*Psychology Department, Moravian College, Bethlehem, PA, USA*

²*Psychology Department, University of California, Riverside, CA, USA*

Introduction

The fundamental question in the study of personality and health is who gets sick and who stays well. This question of connections between individual differences and physical health dates back to Hippocrates and Galen, who proposed that four bodily humors—phlegm, black bile, yellow bile, and blood—were the underlying components in health and individuality and excess or deficiency of any humor would have negative consequences for both personality and health. Though replaced by modern physiology, the ancient Greeks' observations of the connections between psychosocial and physical health influence science to this day.

At the beginning of the twentieth century, Walter Cannon's work on the "fight-or-flight" response linked perceptions, emotions, and physiological reactions. Subsequent research at Johns Hopkins University found that "irregular and uneven" people were more prone to morbidity and mortality than wary and self-reliant types, or cool and clever types (Betz & Thomas, 1979). Such efforts combined with the developing field of psychosomatic medicine to set the stage for modern research.

Attention was piqued in the broader scientific community in the 1970s, with research on Type A behavior pattern. A Type A person is hasty, competitive, impulsive, and impatient and is hypothesized to be prone to coronary disease. However, imprecise conceptualization and inadequate assessment hindered rigorous scientific advances; the field of personality and health needed a more comprehensive approach. Such an approach involves multifactor predictors, multifactor outcomes, and models of the specific, multiple pathways to health across time (Friedman & Kern, 2014). We now know that there is at best a weak and non-unique connection between Type A and disease proneness (Booth-Kewley & Friedman, 1987) although elements of Type A behavior (especially hostility) are indeed part of a disease-prone pattern.

Some people tend to select into (put themselves into) stressful, hostile environments and evoke negative reactions from others. This cyclical process of selecting into and evoking

negative interactions and associated unhealthy behaviors shifts people onto a trajectory toward illness and is known as “disease proneness.” Alternatively, someone may have a good match with his or her environment, creating psychological and physiological homeostasis, and a virtuous circle of behaviors. Such a person is said to have a *self-healing personality*, and tends to be conscientious, secure, and sociable, and self-selects into healthy environments and elicits positive reactions from others (see Friedman, 1991). This type of person is not necessarily stress-free, but views difficulties as challenges rather than threats, and tends to have good coping patterns and psychosocial resources. The self-sustaining appraisals, decisions, and actions of a person with the self-healing personality set him or her on a healthy pathway to long life (see Friedman, 1991; Friedman & Martin, 2011).

Key Considerations in Defining Outcomes and Contexts

The constructs of “disease-prone” and “self-healing” personalities—with their emphases on multiple predictors and multiple outcomes—highlight important considerations when studying personality and health. Many flawed studies rely solely on self-reported health as the measure of health, sometimes boldly (and invalidly) drawing conclusions about objective health. Self-reported health is highly correlated with subjective well-being, and so people who tend to be cheerful are more likely to report good health than individuals who have a more serious or cautious view of life. Although self-reported personality predicts self-reported health, which in turn predicts mortality risk (Idler & Benyamini, 1997), studying self-reported personality and health is not at all the same thing as studying validly assessed personality and objective health; this confusion has led to many problems in this field (Friedman & Kern, 2014). The most revealing studies avoid such problems by measuring multiple aspects of personality, assessing stress and coping, monitoring health behaviors, and tracking disease and mortality across time. For example, using the Type A classification, a hardworking corporate businesswoman who has climbed her way up the ranks with determination and drive would likely be told (incorrectly) that she is at risk of coronary disease. However, knowing that she has a supportive social network, relishes the challenges she faces at work, and has the resources to prevail over challenges, we can place her in the broader context of her surroundings and life path; research suggests she is on a pathway to good health (see Friedman & Martin, 2011).

Six Pathways to Health or Illness

The self-healing and disease-prone personalities encapsulate broadly defined trajectories of health. For instance, one unhealthy trajectory may involve an imprudent adventurer camping in a lightning storm while another may involve an impulsive neurotic coping with stress by binging on TV and junk food, but both pathways predict premature mortality as a function of personality. Differentiating multiple pathways yields a clearer understanding of causes across time. In general, there are six ways of conceptualizing the pathways from personality to health or disease.

First, personality may disrupt the body’s physiological regulation through emotional instability and chronic negative affect. Stress and the long-term breakdown of homeostasis are linked with disease, though the pathways are not yet fully specified. Personality is a predictor and a moderator of stress perceptions, appraisals, and coping mechanisms: some personalities are not only more likely to encounter stressful challenges but are also more likely to both

appraise situations as especially stressful and to lack adequate coping responses. Importantly, challenge in and of itself is not necessarily bad. Indeed, challenge, curiosity, and novelty are established elements of thriving. Rather, it is the appraisal of and reaction to challenge and the degree and length of disruption that puts some people at higher risk of disease.

The second, and perhaps the most direct pathway, involves health behaviors. Certain personalities are more likely to engage in healthy behaviors—everything from eating well, not smoking, and being active to wearing a seatbelt and keeping a regular sleep cycle. Other individuals are more likely to engage in unhealthy behaviors. A key question is whether changing a behavior will change long-term disease risk. Altering some behaviors, such as smoking, will have a big impact on health (as long as other unhealthy behaviors are not adopted instead), while others are erroneously thought to be causal but are simply correlational. Often, predictors are not key elements of causal processes (e.g., see Friedman & Kern, 2014).

Third, personality predicts (and often plays a causal role in) social support, social networks, quality of relationships, and amount of interpersonal conflict, all of which are relevant to health. Good social relations are healthy, but poor relationships can cause stress and influence unhealthy behaviors—for example, divorce and marital conflict predict mortality risk (Sbarra, Law, & Portley, 2011). Separating from a spouse may bring about financial burdens, emotional challenges, the loss of social support, and a need to restructure one's identity and social network. These challenges may pass quickly if coped with appropriately but may also have deleterious long-term consequences. Though the precise mechanisms through which social interactions affect health are not yet fully specified, close, high-quality relationships are usually important to a healthy life trajectory.

A fourth pathway linking personality and health involves biological third variables, including genetics, neurological differences, and central nervous system changes due to early environments. These biological variables predispose individuals toward certain personalities and physiological reactions. For example, people born with Down syndrome not only are at increased risk of early mortality and congenital heart disease but also differ predictably in personality from individuals without Down syndrome. In such cases, personality is correlated with health and longevity, but personality is not a causal element in this association (and so changing the personality will not affect health).

Fifth is the key pathway of situation selection and evocation. Certain individuals gravitate toward certain situations and evoke responses from others that fit with their personalities. This pathway reinforces trajectories toward health or disease, where personality facilitates entering healthy or unhealthy situations. The processes of situation selection and evocation suggest that many events that seem to be bad fortune—such as being bullied—are actually predictable to some degree. For example, a hostile or socially unskilled individual may create trouble and thus elicit an unhealthy environment.

Lastly, we can reverse the causal arrow and look at how health predicts—and sometimes alters—personality. Illness can cause changes relevant to depression, anger, worrying, and more. For example, the immune response to infection can cause lethargy. Similarly, some medications or treatment regimens change personality. Although this pathway is not currently well studied, being treated for or coping with chronic illnesses or handicaps may shift personality. A vicious circle of negative outcomes may result.

These multiple pathways between personality and health are not mutually exclusive; they function together, making it important to understand the full picture. Without a broad understanding of how these pathways work across time, interventions may be misguided and may eventually fail. For instance, early research on Type A led the American Heart Association to recommend screening and treatment for depression in patients with cardiovascular disease, but

treating these patients for depression does not by itself decrease mortality risk (Friedman & Kern, 2014). Caution is needed to study the full causal pathways to ensure that interventions target the true causal elements of illness. Making people happier will not necessarily make them healthier, and worrying is health protective for some individuals in some circumstances (e.g., Weston & Jackson, 2015). It depends on the situation and the life trajectory of an individual.

Personality Trajectories

What are the pathways to health? How do individuals who thrive and stay healthy live their lives? In particular, what is the relevance of the five broad domains used by researchers to describe personality: conscientiousness, extraversion, agreeableness, openness, and neuroticism?

Conscientiousness

Conscientiousness is the best-established trait predicting health. People who are orderly, achievement motivated, prudent, responsible, and planful are more likely to stay on healthy pathways to long life (Friedman & Martin, 2011; Goodwin & Friedman, 2006). The original finding that conscientiousness in childhood predicts risk of dying at any age (Friedman et al., 1993) has been supported by many other subsequent studies (e.g., Chapman, Fiscella, Kawachi, & Duberstein, 2010).

Conscientiousness predicts, and often affects, health through several pathways that work together to produce a cycle of healthy habits, appraisals, and coping responses. People high in conscientiousness are more likely to engage in protective health behaviors and are less likely to engage in riskier behaviors such as smoking (see Bogg & Roberts, 2004). However, behavioral pathways do not fully explain the healthy outcomes of conscientious people (Martin, Friedman, & Schwartz, 2007).

Highly conscientious people often find their way to less stressful lives. For example, the most conscientious children in the Terman lifespan study (a nine-decade study of personality and health) were the least likely later to be sent to the most difficult US frontline during World War II as adults (Duggan & Friedman, 2014), avoiding the subsequent disruption in family life and career that combat often brings. When they do encounter stress, they may have better coping abilities. The difference in trajectories means that conscientious people are more likely to be productively engaged in society, with better careers, educations, and incomes than their less conscientious counterparts (Hampson, Goldberg, Vogt, & Dubanoski, 2007).

When conscientious people do experience stress, such as a career setback, their personality is more likely to attenuate the negative effects, perhaps because they are more likely to engage in problem-focused coping rather than through substance abuse, problem-denying, or expressing negative emotions (Connor-Smith & Flachsbart, 2007). Taken together, these studies suggest that the pathway from conscientiousness to health occurs across the lifespan through selection into stable situations (marriages, careers), evocation of positive responses from others, and healthy habits that maintain homeostasis.

Extraversion

Extraverts tend to be dominant, sociable, and cheerful and are inclined to elicit positive social interactions. Extraversion provides a good example of why considering the facets (or subtraits) of the Big Five is informative in understanding personality and health; the facets

(dominance, sociability, and positivity) relate to health differentially—part of the reason why extraversion is inconsistently linked to physical health (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007).

The facet of dominance predicts positive job outcomes, but its relation to health depends on the situation. When combined with positive affect and agreeableness, dominance plays an important role in good leadership, but when combined with hostility, dominance may increase risk of heart disease, unstable relationships, and poor mental health. Further, in stable environments, where dominance can be established, it may be adaptive to take the lead; however, being dominant when the hierarchy is easily threatened can be risky (for a study of monkeys: Manuck, Kaplan, Adams, & Clarkson, 1988). These findings illustrate how the link between personality and health is tied to environmental contexts and other personality variables.

When considering the importance of social relationships to health, one might assume that the sociability facet of extraversion will be predictive of good health. However, although sociability is generally linked with good subjective health and social outcomes, there are mixed results for objective health outcomes. For example, in the Terman sample, extraversion predicted more social ties and higher social competence, but not physical health or mortality (Friedman, Kern, & Reynolds, 2010). Sociability may be linked to health through better coping strategies, social relationships, approach-oriented temperament, and positive engagement with others (Connor-Smith & Flachsbart, 2007). However, these extraverts may also be drawn toward unhealthy situations. Extraverted college students, for instance, are more likely to smoke, drink, get poor sleep, and have multiple sex partners, thus increasing risk of morbidity and mortality (Munafo, Zetteler, & Clark, 2007).

A highly debated topic is that of positive affect (good feelings) and health. There is a small positive correlation of trait positive affect with self-reported health, symptom control, and survival of people with chronic diseases (Howell, Kern, & Lyubomirsky, 2007), but the causal link is unclear, and it would therefore be unwise to intervene with positivity interventions in an attempt to save lives at this stage of understanding. Although individuals with high positive affect are more likely to exercise, to have healthy behaviors and healthy relationships, and to perceive stressors as challenging rather than threatening, these are correlations. Being optimistic in the face of challenge may increase someone's willingness to persist with treatment or attain a goal, but there is no scientific evidence that positive thinking will heal broken bones, unclog arteries, fight cancer, or undo a diagnosis. Positivity interventions may promote health so far as they encourage healthy behaviors or decrease chronic stress but may raise the risks of blaming the victim and encouraging unproductive or irrelevant responses.

Being too positive may put one at increased risk by underestimating one's susceptibility to morbidity or mortality. In the Terman study, children rated as cheerful (sense of humor, cheery, optimistic) were at greater risk of dying at any age, partially due to risky behaviors in adulthood (Friedman et al., 1993). Dispositional optimism may steer individuals toward unrealistic thoughts, or the "above average effect" (illusory superiority), as is reflected in a study of individuals trying to lose weight, where dispositional optimism predicted confidence in ability to lose weight but did not predict actual weight loss (Benyamini & Raz, 2007). Taken together, it appears that optimism combined with conscientiousness is protective as it drives individuals toward achievement, but optimism combined with impulsivity and unrealistic expectations may become detrimental.

Agreeableness

Agreeableness is the tendency to be cooperative, trusting, kind, and generous. People who are agreeable are more likely to use adaptive coping mechanisms (Connor-Smith & Flachsbart,

2007). However, research on the relationship between agreeableness and health is mixed. Some studies suggest that agreeable people have better self-reported health and live longer lives, but other studies have found no relationship (Iwasa et al., 2008).

Though agreeableness does not reliably predict morbidity or mortality (Martin et al., 2007), the combination of agreeableness with other traits may have explanatory power. For instance, hostility involves both high neuroticism and low agreeableness, which predicts poor health and increased mortality risk (Booth-Kewley & Friedman, 1987), likely through chronic stress, and poor health behaviors and social skills. In a meta-analysis of 27 studies, high hostility was associated with higher BMI, more alcohol use, smoking, and markers of heart disease (Bunde & Suls, 2006).

Conscientiousness may also work in tandem with agreeableness in predicting health. High agreeableness and low conscientiousness may be risky, as the individual may be considered a “pushover” and fails to achieve personal goals in exchange for doing what others want them to do (Chapman et al., 2010). As previously discussed, though, low agreeableness is also risky. Therefore, agreeableness may be most predictive of good health when it is paired with protective traits and social contexts.

Openness

Openness involves intellectual curiosity, willingness to experiment, and creativity. People who are open are more likely to cope resourcefully and effectively (Connor-Smith & Flachsbart, 2007). In the MIDUS study (a longitudinal study of health and well-being in the United States), it is found that low levels of openness in patients relate to stroke, hernia, tuberculosis, and bone problems (Goodwin & Friedman, 2006). A subsequent study found that high levels of openness relate to decreased odds of a stroke, heart condition, blood pressure, and arthritis diagnosis (Weston, Hill, & Jackson, 2014).

The intellect facet may play a particularly strong role in predicting health. For instance, higher intellect predicts health status and educational attainment (Hampson et al., 2007). However, some studies of personality and longevity have found no relation between openness and mortality (Maier & Smith, 1999). Openness is the least understood trait in its relation to health, as the pathways are not yet clearly specified, and the objective outcomes from study to study are at odds with one another.

Neuroticism

Neuroticism, the tendency to experience the world as distressful, is frequently studied and further reveals the complex relationship of personality and health. Neurotics are prone to anxiety and depression and are emotionally unstable. It has long been known that people with higher levels of hostility, anxiety, and depression are more likely to be ill, but this is not necessarily a causal pathway, and prospective studies do not always show the same relationship; the commonsense view that neuroticism (“worrying”) leads to disease and premature mortality requires significant qualification (Friedman & Kern, 2014).

Consider the strong relationship between neuroticism and subjective well-being. This association makes it difficult to disentangle the effects of neuroticism on objective health outcomes because subjective well-being is related to self-rated health, symptom report, and medical visits. In other words, individuals high on neuroticism are more likely to feel and report symptoms, which can lead to further distress even when there are no objective health differences (Costa & McCrae, 1987). Though neuroticism may sometimes predict increased risk of

mortality, various studies find no relation between neuroticism and mortality (e.g., Iwasa et al., 2008). Given these conflicting findings, it is likely that the effect of neuroticism on objective health is moderated by other personality or environmental factors (e.g., Taga, Friedman, & Martin, 2009). There is also little evidence that intervening on neuroticism increases longevity. In fact, it may even be risky to intervene in some situations, such as what Friedman terms “healthy neuroticism” (Friedman, 2000), or the combination of high neuroticism with high conscientiousness.

Healthy neurotics may be particularly vigilant about their health (Friedman, 2000). Though healthy neurotics likely have lower subjective well-being, more doctor visits, and more psychosomatic symptoms, they may be more willing to engage in health protective behaviors. Healthy neuroticism may also have a particularly profound effect in stressful situations. For example, conscientiously neurotic individuals diagnosed with diseases most related to smoking were more likely to quit smoking after disease diagnosis, but did not show any smoking differences before disease onset (Weston & Jackson, 2015). In the Terman sample, neuroticism predicted lower health and increased mortality risk for women, but decreased risk for widowed men (Taga et al., 2009). That is, men who lost their wives and were particularly anxiety prone lived longer than the carefree men who lost their wives. Together, these studies suggest that when experiencing a stressful situation, high levels of neuroticism may induce increased anxiety, and conscientiousness may make one more successful at channeling that anxiety into health-promoting behaviors.

Neuroticism is a good example of the importance of using a lifespan perspective to study the relationship between personality and health, as neuroticism can also be the outcome of illness. For instance, neurological conditions and inflammatory responses may cause depression. When depression is the outcome of disease, high neuroticism may cause a negative spiral when the individual fails to enact adaptive coping mechanisms.

There is limited evidence that decreasing neuroticism reaps objective health gains. Recall that though treating depression decreases the risk of a secondary cardiac event, it does not reduce mortality risk (Rutledge, Redwine, Linke, & Mills, 2013). The association between depression and mortality may often be confounded by hostility (Lemogne et al., 2010). Treating depression may only deter or delay disease so far as it motivates individuals to adopt healthier behaviors (such as increased physical activity), social networks, or coping skills. As neuroticism is health protective for some individuals, intervening on neuroticism may be risky, thus highlighting the importance of targeted interventions that understand the full causal chain.

Interventions and Future Directions

Interventions that address personality to improve health should simultaneously target individual, community, and societal levels in order to launch people onto healthy paths and maintain their adherence to these healthy pathways. Because early character affects social relationships, careers, and environmental choice, all of which relate to health in later years, we need further understanding of how to shift people onto these healthy trajectories early and how to maintain these pathways, rather than intervening on short-term stressors at any instance throughout life. Such interventions need to be rigorously tested over time to determine their impact on well-being, disease, and, ultimately, mortality.

The most promising trait for health intervention is conscientiousness. We know that having a high level of conscientiousness is healthy, and we know many key pathways from high

conscientiousness to longevity. Can we increase conscientiousness and sustain these changes? Personality is relatively stable but shows patterns of meaningful change across the lifespan, suggesting that change is possible. Different facets of conscientiousness may change differentially, as well. For instance, industriousness has been found to change early in life, impulse control and reliability changes across the lifespan, and orderliness is relatively stable (Jackson et al., 2009). Whether change is possible may also depend on contextual factors, such as age or stage of life. It may, for instance, be most effective to intervene during transitional phases of life, when a person is shifting habits and behaviors.

Conclusion

Understanding who gets sick and who stays well is an integral part of healthcare. By identifying which individuals are more susceptible to illness, we may be more likely to prevent illness and intervene in a more targeted manner appropriate to a specific demographic. After people fall ill, understanding personality influences can be important in predicting who will cooperate with treatment and stay on pathways that maximize chances for recovery.

In disentangling the effects of personality on health and longevity, it is important to remember that personality interacts with psychosocial contexts. We cannot say that, for everyone, neuroticism is bad or that agreeableness is good. We must consider each individual as embedded in his or her environment across time and the ways in which personality traits interact with each other. In doing so, we can address how to shift people onto healthy life trajectories.

Author Biographies

Dietlinde Heilmayr is an assistant professor of health psychology at Moravian College. She earned her BA from Hendrix College and her PhD from the University of California, Riverside. Her service experiences with AmeriCorps, Fulbright, and Bike and Build inform her applied scholarly interests broadly focused on nature-based health interventions. In particular, Heilmayr focuses on the rigorous assessment and exploration of community gardening as a health intervention. She teaches a variety of courses including health psychology and experimental methods and data analysis and was the recipient of the Distinguished Graduate Teaching Award in 2017.

Howard S. Friedman is a distinguished professor of psychology at the University of California, Riverside. His book, *The Longevity Project: Surprising Discoveries for Health and Long Life from the Landmark Eight-Decade Study*, summarizes his 25-year scientific study of the pathways to health and long life. His major scientific awards include the James McKeen Cattell Fellow career award from the Association for Psychological Science and the “Outstanding Contributions to Health Psychology” senior award from the American Psychological Association (Div. 38).

References

- Benyamini, Y., & Raz, O. (2007). “I can tell you if I’ll really lose all that weight”: Dispositional and situated optimism as predictors of weight loss following a group intervention. *Journal of Applied Social Psychology*, 37(4), 844–861. doi:10.1111/j.1559-1816.2007.00189.x

- Betz, B. J., & Thomas, C. B. (1979). Individual temperament as a predictor of health or premature disease. *The Johns Hopkins Medical Journal*, 144(3), 81–89.
- Bogg, T., & Roberts, B. W. (2004). Conscientiousness and health-related behaviors: A meta-analysis of the leading behavioral contributors to mortality. *Psychological Bulletin*, 130(6), 887–919.
- Booth-Kewley, S., & Friedman, H. S. (1987). Psychological predictors of heart disease: A quantitative review. *Psychological Bulletin*, 101(3), 343–362. doi:10.1037//0033-2909.101.3.343
- Bunde, J., & Suls, J. (2006). A quantitative analysis of the relationship between the Cook-Medley Hostility Scale and traditional coronary artery disease risk factors. *Health Psychology*, 25(4), 493–500. doi:10.1037/0278-6133.25.4.493
- Chapman, B. P., Fiscella, K., Kawachi, I., & Duberstein, P. R. (2010). Personality, socioeconomic status, and all-cause mortality in the United States. *American Journal of Epidemiology*, 171(1), 83–92. doi:10.1093/aje/kwp323
- Connor-Smith, J. K., & Flachsbart, C. (2007). Relations between personality and coping: A meta-analysis. *Journal of Personality and Social Psychology*, 93(6), 1080–1107. doi:10.1037/0022-3514.93.6.1080
- Costa, P. T., & McCrae, R. R. (1987). Neuroticism, somatic complaints, and disease: Is the bark worse than the bite? *Journal of Personality*, 55(2), 299–316. doi:10.1111/j.1467-6494.1987.tb00438.x
- Duggan, K. A., & Friedman, H. S. (2014). Lifetime biopsychosocial trajectories of the Terman gifted children: Health, well-being, and longevity. In D. K. Simonton (Ed.), *The Wiley handbook of genius* (pp. 488–507). Oxford, UK: Wiley-Blackwell.
- Friedman, H. S. (1991). *The self-healing personality. Why some people achieve health and others succumb to illness*. New York, NY: Henry Holt.
- Friedman, H. S. (2000). Long-term relations of personality and health: Dynamisms, mechanisms, tropisms. *Journal of Personality*, 68(6), 1089–1107.
- Friedman, H. S., & Kern, M. L. (2014). Personality, well-being, and health. *The Annual Review of Psychology*, 65(18), 1–24. doi:10.1146/annurev-psych-010213-115123
- Friedman, H. S., Kern, M. L., & Reynolds, C. A. (2010). Personality and health, subjective well-being, and longevity. *Journal of Personality*, 78(1), 179–216. doi:10.1111/j.1467-6494.2009.00613.x
- Friedman, H. S., & Martin, L. R. (2011). *The longevity project: Surprising discoveries for health and long life from the landmark eight decade study*. London: Hay House, Inc.
- Friedman, H. S., Tucker, J. S., Tomlinson-Keasey, C., Schwartz, J. E., Wingard, D. L., & Criqui, M. H. (1993). Does childhood personality predict longevity? *Journal of Personality and Social Psychology*, 65(1), 176–185. doi:10.1037/0022-3514.65.1.176
- Goodwin, R. D., & Friedman, H. S. (2006). Health status and the five-factor personality traits in a nationally representative sample. *Journal of Health Psychology*, 11(5), 643–654. doi:10.1177/13591053060066610
- Hampson, S. E., Goldberg, L. R., Vogt, T. M., & Dubanoski, J. P. (2007). Mechanisms by which childhood personality traits influence adult health status: Educational attainment and healthy behaviors. *Health Psychology*, 26(1), 121–125. doi:10.1037/0278-6133.26.1.121
- Howell, R. T., Kern, M. L., & Lyubomirsky, S. (2007). Health benefits: Meta-analytically determining the impact of well-being on objective health outcomes. *Health Psychology Review*, 1(1), 83–136. doi:10.1080/17437190701492486
- Idler, E. L., & Benyamini, Y. (1997). Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior*, 38, 21–37.
- Iwasa, H., Masui, Y., Gondo, Y., Inagaki, H., Kawaai, C., & Suzuki, T. (2008). Personality and all-cause mortality among older adults dwelling in a Japanese community: A five-year population-based prospective cohort study. *The American Journal of Geriatric Psychiatry*, 16(5), 399–405. doi:10.1097/JGP.0b013e3181662ac9
- Jackson, J. J., Bogg, T., Walton, K. E., Wood, D., Harms, P. D., Lodi-Smith, J., ... Roberts, B. W. (2009). Not all conscientiousness scales change alike: A multimethod, multisample study of age differences in the facets of conscientiousness. *Journal of Personality and Social Psychology*, 96(2), 446–459. doi:10.1037/a0014156

- Lemogne, C., Nabi, H., Zins, M., Cordier, S., Ducimetière, P., Goldberg, M., & Consoli, S. M. (2010). Hostility may explain the association between depressive mood and mortality: Evidence from the French GAZEL cohort study. *Psychotherapy and Psychosomatics*, 79(3), 164–171. doi:10.1159/000286961
- Maier, H., & Smith, J. (1999). Psychological predictors of mortality in old age. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 54(1), 44–54. doi:10.1093/geronb/54B.1.P44
- Manuck, S. B., Kaplan, J. R., Adams, M. R., & Clarkson, T. B. (1988). Studies of psychosocial influences on coronary artery atherogenesis in cynomolgus monkeys. *Health Psychology*, 7(2), 113–124. doi:10.1037/0278-6133.7.2.113
- Martin, L. R., Friedman, H. S., & Schwartz, J. E. (2007). Personality and mortality risk across the life span: The importance of conscientiousness as a biopsychosocial attribute. *Health Psychology*, 26(4), 428–436. doi:10.1037/0278-6133.26.4.428
- Munafo, M. R., Zetteler, J. I., & Clark, T. G. (2007). Personality and smoking status: A meta-analysis. *Nicotine & Tobacco Research*, 9(3), 405–413. doi:10.1080/14622200701188851
- Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, 2(4), 313–345. doi:10.1111/j.1745-6916.2007.00047.x
- Rutledge, T., Redwine, L. S., Linke, S. E., & Mills, P. J. (2013). A meta-analysis of mental health treatments and cardiac rehabilitation for improving clinical outcomes and depression among patients with coronary heart disease. *Psychosomatic Medicine*, 75(4), 335–349. doi:10.1097/PSY.0b013e318291d798
- Sbarra, D. A., Law, R. W., & Portley, R. M. (2011). Divorce and death: A meta-analysis and research agenda for clinical, social, and health psychology. *Perspectives on Psychological Science*, 6(5), 454–474. doi:10.1177/1745691611414724
- Taga, K. A., Friedman, H. S., & Martin, L. R. (2009). Early personality traits as predictors of mortality risk following conjugal bereavement. *Journal of Personality*, 77(3), 669–690. doi:10.1111/j.1467-6494.2009.00561.x
- Weston, S. J., Hill, P. L., & Jackson, J. J. (2014). Personality traits predict the onset of disease. *Social Psychological and Personality Science*, 1–9. doi:10.1177/1948550614553248
- Weston, S. J., & Jackson, J. J. (2015). Identification of the healthy neurotic: Personality traits predict smoking after disease onset. *Journal of Research in Personality*, 54, 61–69. doi:10.1016/j.jrp.2014.04.008

Suggested Reading

- Bogg, T., & Roberts, B. W. (2004). Conscientiousness and health-related behaviors: A meta-analysis of the leading behavioral contributors to mortality. *Psychological Bulletin*, 130(6), 887–919.
- Friedman, H. S. (1991). *The self-healing personality. Why some people achieve health and others succumb to illness*. New York, NY: Henry Holt.
- Friedman, H. S., & Martin, L. R. (2011). *The longevity project: Surprising discoveries for health and long life from the landmark eight decade study*. London: Hay House, Inc.

Personality Hardiness

Salvatore R. Maddi

Department of Psychological Science, University of California, Irvine, Irvine, CA, USA

Introduction

Some psychologists consider resilience to emphasize how people can remain the same despite the imposition on them of stressful circumstances. One conclusion from this position is that it is helpful to avoid stressors. In contrast, personality hardiness emphasizes resilience as the process of change, growth, and development that is provoked by dealing with rather than avoiding stressful circumstances (e.g., Maddi, 2002; Maddi & Khoshaba, 2005).

In this regard, an assumption behind personality hardiness is that life is by its nature changing and stressful, so involving oneself in this process helps one grow and develop toward greater fulfillment. This assumption derives from the existential position (Kierkegaard, 1954) that everything we do in life is the result of making decisions. As life is an ongoing process of change, each decision with which we are faced involves choosing the unknown future or the familiar past. Although choosing the future is developmentally best, it brings anxiety because the outcome cannot be predicted in advance. But if we decide to avoid anxiety by choosing the past (i.e., what we already know and are doing), we encounter the existential guilt of missed opportunity, boredom, and emptiness of living.

Based on this dilemma, the hardiness approach assumes that, in one's ongoing life, the best way to establish a pattern of choosing the future, rather than the past, is to develop the personality pattern of hardiness. Hardiness is considered a pattern of attitudes and strategies that help one do the hard work of choosing the future rather than the past (Maddi, 2002). The more than 40 years of research, consulting, and counseling that emerged from this conceptualization has solidified this position. Early on, Maddi's (1965) work involved showing that people who express more interest in change are also likely to be more creative in their work. But in specifying the personality characteristics involved more completely and the particular effects of stressors on functioning, it seemed important to carry out a relevant experiment, rather than just doing correlational research.

The Illinois Bell Telephone Project

In the 1970s, Maddi was a consultant for Illinois Bell Telephone (IBT). At that time, the US telephone industry was a federally regulated monopoly, whose member companies only had to provide cheap and reliable telephone service and did not have to worry about their bottom lines. But it became clear that this situation had to change, as the beginning of the Internet was opening up the field and encouraging international competition. The US government made clear that the telephone industry would need to be deregulated in order to motivate competition for greater possibilities and income in the newly developing circumstances.

With the help of IBT decision makers, Maddi and his research team in 1974 began yearly testing of 450 managers in a longitudinal research study to determine the effects of the deregulation on them. Each yearly testing included not only the set of relevant questionnaires but also interviews. Also available were yearly job performance evaluations done by the company. Six years into the study (in 1980), the federal deregulation occurred, and its effects are still regarded as a major disruption on the industry and its companies. Indeed, in the year following the deregulation, nearly half of the managers in our sample were terminated. As to the research study, we continued to test the managers, whether they continued or were terminated, with the questionnaires, interviews, and job evaluations each year for the following 6 years. The study is still regarded as a classical natural experiment.

The study results showed that, in the 6 years following the deregulation of the telephone industry, nearly two-thirds of the sample fell apart in various ways. They showed clear signs of major anxiety, depression, and anger, undermining performance at work. These signs included violence, data loss, inability to make decisions, and decision-making avoidance. And in home life, they also showed signs of being undermined, such as divorces, substance abuse, and even suicides.

In contrast, the other third of the sample not only survived but also thrived following the deregulation. At work, they either rose up in the ranks, or if they were among those terminated, they found significant jobs in other organizations or even started their own companies. In all this, they were able to give and get social support with their significant others (at work or at home) and were able to grow and develop by dealing effectively with the stress of deregulation.

Of particular importance in developing hardiness assessment was the comparison of managers who reacted resiliently to the deregulation with those who did not (Maddi & Kobasa, 1984). In the 6 years before this upheaval, the managers who reacted well to the deregulation were much stronger than the others in the attitudes of *commitment*, *control*, and *challenge*. No matter how bad things might get, they wanted to stay involved with people and contexts (*commitment*), continue to struggle to have an influence on their outcomes (*control*), and learn how to grow and develop by trying to deal effectively with stressors (*challenge*). In contrast, the managers who fell apart under the deregulation had even before it showed definite signs of playing it safe by avoiding stressors and, if unsuccessful in this avoidance, by sinking into alienation and powerlessness. These results led to conceptualizing hardiness as the attitudes (the three Cs of commitment, control, and challenge) and strategies (problem solving rather than avoidance coping, socially supportive rather conflict-laden interactions, and beneficial rather than overindulgent self-care) associated with resilience. It appeared that hardy attitudes provided the courage and motivation to be able to do the hard work of hardy strategies that turned stressors into growth advantages. All of this is quite consistent with the existential view, well summarized by Nietzsche (1968, *Nietzsche's Twilight of the Idols*, p. 254) as "whatever doesn't kill me makes me stronger."

Also, the IBT data on interviews and questionnaires completed prior to the deregulation permitted a study (Khoshaba & Maddi, 1999) on the early life experiences of managers and the effects of these experiences on performance after the deregulation. Content analyses of the data showed that the managers who thrived after the deregulation had reported remembering a more disruptive, stressful early family life and having been selected by their parents as the hope of the family. They indicated having accepted this role and working hard to justify it. These results are consistent with the assumption that hardiness can be learned rather than being inborn. It also indicates that parents can help their children grow in hardiness by teaching them that life is by its nature stressful, and involving themselves in this process, rather than denying it, is the way to grow and develop toward a better life. In contrast, parents who are overprotective and controlling may be jeopardizing their children's growth toward hardiness.

Further Evolution of the Hardiness Approach

In the years since the IBT study, there have been many additional research studies and consulting work on the hardiness approach. In this process, our various versions of the hardiness questionnaire have been translated into more than 15 Asian and European languages and are also used in English in numerous countries. There have also been reviews of hardiness research (see Suggested Reading). In the research studies covered in these reviews, it has been shown that the hardy attitudes (the three Cs of commitment, control, and challenge) are positively related to each other and to the hardy strategies of problem-solving (rather than avoidance) coping, socially supportive (rather than conflict-laden) interactions, and beneficial (rather than overindulgent) self-care. This hardiness pattern has been positively associated with enhanced performance, conduct, mood, and health in samples of college and high-school students and working adults in military, firefighting, sports, and business contexts (e.g., Maddi, 2002). Some specific topics and studies are considered below.

In the process of research and consulting, there has been systematic improvement of hardy attitudes measurement. The original questionnaire for the three Cs of commitment, control, and challenge was 50 items in length. Over the years, the measure has been shortened and improved. The current version (Maddi, 2001) is the 18-Likert-item Personal Views Survey, III-R (third edition, revised). Examples of items are, for commitment, "I often wake up eager to take on life wherever it left off"; for control, "When I make plans, I'm certain I can make them work"; and for challenge, "Changes in routine provoke me to learn." Cronbach's alpha coefficients for all three Cs range in the .70's and for total hardiness are even higher. The three Cs are positively correlated, and each of them shows a high correlation with the total hardiness score. Also, consistent with hardiness theorizing, Sinclair and Tetrick (2000) have found that factor analysis of the hardiness test items yields the three empirically related first-order factors of commitment, control, and challenge and that these factors are indeed positively related to the second-order factor of hardiness. This pattern has supported the conceptually emphasized use of a total hardiness score as necessary. In another study (Maddi & Harvey, 2005), no cross-cultural or demographic differences were found in hardiness.

By now, there are many especially significant studies on the role of hardiness in various performance, conduct, and health activities. For example, in samples of bus drivers (Bartone, 1989), lawyers (Kobasa, 1982), and nurses (Keane, Ducette, & Adler, 1985), hardiness was found to be positively correlated with measures of performance and satisfaction and negatively correlated with anxiety and depression. There are also similar findings in American employees experiencing the culture shock of work missions abroad (Atella, 1989) and in foreign

immigrants to the United States (Kuo & Tsai, 1986). In a study of sports performance (Maddi & Hess, 1992), hardiness levels of male, high-school varsity basketball players were measured in the summer, and then their performance was obtained in the ensuing season. Hardiness predicted six out of seven indices of performance in the expected direction, even though all the subjects were varsity players.

Also, there have been studies concerning hardiness and military personnel. For example, Bartone (1999) found that soldiers high in hardiness adjusted better to health and life stressors than did those who were low in hardiness. Further, among soldiers returning from operational deployments, there was a positive relationship between hardiness and readjustment to their former lives (Britt, Adler, & Bartone, 2001). Hardiness was also a positive predictor of performance success in soldiers undergoing special forces training among Norwegian Naval Academy cadets (Bartone, Johnsen, Eid, Brun, & Laberg, 2002). Similarly, hardiness was positively correlated with both retention and performance excellence in US Military Academy cadets (Maddi, Matthews, Kelly, Villarreal, & White, 2012).

In addition, studies support a negative relationship between hardiness and conduct problems, such as alcohol and gambling abuse. For example, Maddi, Wadhwa, and Haier (1996) found that among high-school graduates about to enter college, hardiness had a negative relationship with the reported frequency with which alcohol and drugs were used. In this study, the reported frequency of alcohol and drug use was objectively validated through urine screens. Also found in a sample of college students was a negative relationship between hardiness and gambling (Maddi et al., 2015).

Comparison of Hardiness and Other Possible Predictors

Understandably, questions have arisen as to whether hardiness is little more than yet another personality variable. This concern has led to several empirical studies comparing hardiness and other possible predictors of performance.

One other possible predictor that has been proposed is negative affectivity, or neuroticism (e.g., Hull, Van Treuren, & Virnelli, 1987). Maddi and Khoshaba (1994) conducted a relevant study in which hardiness and an accepted measure of negative affectivity were entered into a regression analysis as independent variables in an attempt to predict the clinical scales of the Minnesota Multiphasic Personality Inventory (MMPI). With the effects of negative affectivity controlled, hardiness was still a robust negative predictor of the MMPI clinical scales.

Another alternative predictor that might be confounded with hardiness is optimism. To test this possibility, Maddi and Hightower (1999) conducted three related studies that compared the relative influence of hardiness and optimism with transformational and regressive coping. Studying undergraduate students, the first two studies differed in the tests used to assess transformational and regressive coping styles. Nonetheless, the results showed consistently that, by comparison with optimism, hardiness was the better positive predictor of transformational coping and negative predictor of regressive coping. In the third study, the same approach was used in a sample of women who had breast lumps and were arriving at a specialty clinic for diagnosis of whether or not the lumps were cancerous. Under this life-threatening stressor, optimism showed similar effects on coping as did hardiness, but only hardiness was a negative predictor of regressive coping. Taken together, these three studies show that hardiness is a better predictor of effective coping and that optimism, by comparison, may be laced with naïve complacency.

Another explanatory alternative to hardiness is grit, which is the courage provided by having a definite goal that affects performance under stress and will never be given up (Duckworth & Quinn, 2009). By comparison, hardiness is the existential courage to continue to learn from and change under life's stressors (Maddi, 2002). To investigate the relative predicted power of hardiness and grit, a sample of cadets at the US Military Academy were tested for hardiness and grit several weeks before their training began, and then their performance evaluations were followed during the 4 years of this intentionally stressful training. The study results showed that hardiness was a better predictor than grit of cadet retention and performance (Maddi et al., 2012, 2015).

Hardiness Practice Applications

There is a growing need for hardiness assessment and training, due to the increased rate of social, technological, and cultural change being fueled by our transition from industrial to information societies (e.g., Peters, 1988). In an effort to adapt to the pressures of change, companies are continually restructuring (e.g., decentralizing, merging, downsizing, upsizing), which influences the lives of employees. In an attempt to help individuals deal well and grow with these changes, Maddi and Khoshaba have developed hardiness assessment and training procedures that can be used in consultation with individuals and organizations.

As to hardiness assessment, there is now the *HardiSurvey III-R*, a valid and reliable 65-item questionnaire that measures the vulnerability factors of stress, strain, and regressive coping, along with the resilience factors of hardiness attitudes, problem-solving coping, and supportive social interactions (Maddi & Khoshaba, 2001). The vulnerability and resilience factors of the test taker are compared with each other and with available norms, leading to a wellness ratio. This test can be taken on our website (www.HardinessInstitute.com) and provides a comprehensive report concerning your resilience or vulnerability under stress.

As to hardiness training, there is now a comprehensive workbook (Khoshaba & Maddi, 2008; Maddi & Khoshaba, 2005) that includes instructions, exercises, case studies, and evaluation procedures concerning how to engage in problem-solving (rather than avoidance) coping, socially supportive (rather than conflict-laden) interactions, and beneficial (rather than overindulgent) self-care. Also shown is how to use what is learned through these procedures to deepen your hardy attitudes of commitment, control, and challenge, so that once the training is over, you will have the courage and strategies to do the hard work involved in improving your functioning in everyday living. This workbook can be used by trainees on their own, or with the supervision of a certified hardiness trainer.

There are some studies supporting the effectiveness of the hardiness training procedure. In one study (Maddi, Kahn, & Maddi, 1998), IBT managers who went through hardiness training in the year following the stressful deregulation of the telephone industry improved in their performance and felt less anxious and overwhelmed, despite the huge disruption of the company. In this process, the IBT managers also increased in their hardy attitudes and simultaneously decreased in their self-reported strain. This beneficial pattern of results was still present at a 6-month follow-up test. Further, a recent study (Maddi, Harvey, Khoshaba, Fazel, & Resurreccoin, 2009) showed that undergraduate students who went through a hardiness training course based on the workbook, by comparison with a carefully developed control group, not only increased in hardiness but also in grade-point average at graduation.

Concluding Remarks

In terms of conceptualization and empirical support, it appears that the hardiness approach has growing validity. This encourages its use in assessment and training to increase the likelihood that individuals and organizations will be able to turn stressful circumstances from potential disasters into growth opportunities instead.

Author Biography

The son of Sicilian immigrants, Salvatore R. Maddi was born in New York in 1933. He received a PhD in clinical psychology with honors from Harvard University in 1960. Having taught at the University of Chicago (1960–1986) and the University of California, Irvine (1986–2015), he has developed the hardiness approach that shows how people can develop the courage, motivation, and capabilities to turn stressful circumstances into growth opportunities. He has won many awards, the latest of which is the 2012 American Psychological Foundation Gold Medal.

References

- Atella, M. (1989). Crossing boundaries: Effectiveness and health among western managers living in China (Unpublished doctoral dissertation). University of Chicago.
- Bartone, P. T. (1989). Predictors of stress related illness in city bus drivers. *Journal of Occupational Medicine*, 31, 657–663.
- Bartone, P. T. (1999). Hardiness protects against war-related stress in army reserve forces. *Consulting Psychology Journal*, 51, 72–82. doi:10.1037/1061-4087.51.2.72
- Bartone, P. T., Johnsen, B. H., Eid, J., Brun, W., & Laberg, J. C. (2002). Factors influencing small-unit cohesion in Norwegian Navy officer cadets. *Military Psychology*, 4, 1–22. doi:10.1207/S15327876MP1401_01
- Britt, T. W., Adler, A. B., & Bartone, P. T. (2001). Deriving benefits from stressful events: The role of engagement in meaningful work and hardiness. *Journal of Occupational Health Psychology*, 6, 53–63. doi:10.1037/1076-8998.6.1.53
- Duckworth, A. L., & Quinn, P. D. (2009). Development and validation of the short grit scale. *Journal of Personality Assessment*, 91, 166–174. doi:10.1080/00223890802634290
- Hull, J. G., Van Treuren, R. R., & Virnelli, S. (1987). Hardiness and health: A critique and alternative approach. *Journal of Personality and Social Psychology*, 53, 518–530. doi:10.1037/0022-3514.53.3.518
- Keane, A., Ducette, J., & Adler, D. (1985). Stress in ICU and non-ICU nurses. *Nursing Research*, 34, 231–236.
- Khoshaba, D. M., & Maddi, S. R. (1999). Early experiences in hardiness development. *Consulting Psychology Journal*, 51, 106–116. doi:10.1037/1061-4087.51.2.106
- Khoshaba, D. M., & Maddi, S. R. (2008). *HardiTraining: Managing stressful change* (4ed. ed.). Newport Beach, CA: Hardiness Institute.
- Kierkegaard, S. (1954). *The sickness unto death*. New York, NY: Doubleday.
- Kobasa, S. C. (1982). Commitment and coping in stress resistance among lawyers. *Journal of Personality and Social Psychology*, 42, 707–717. doi:10.1037/0022-3514.42.4.707
- Kuo, W. H., & Tsai, Y. (1986). Social networking, hardiness, and immigrant's mental health. *Journal of Health and Social Behavior*, 27, 133–149. Retrieved from <http://www.jstor.org/stable/2136312>
- Maddi, S. R. (1965). Motivational aspects of creativity. *Journal of Personality*, 33, 330–347. doi:10.1111/j.1467-6494.1965.tb01390.x

- Maddi, S. R. (2001). *HardiSurvey III-R: Test development and internet instruction manual*. Newport Beach, CA: Hardiness Institute.
- Maddi, S. R. (2002). The story of hardiness: Twenty years of theorizing, research, and practice. *Consulting Psychology Journal*, 54, 175–185. doi:10.1037/1061-4087.54.3.173
- Maddi, S. R., & Harvey, R. H. (2005). Hardiness considered across cultures. In P. T. Wong & L. C. J. Wong (Eds.), *Handbook of multicultural perspectives on stress and coping* (pp. 403–420). New York, NY: Springer.
- Maddi, S. R., Harvey, R. H., Khoshaba, D. M., Fazel, M., & Resurreccoin, N. (2009). Hardiness facilitates performance in college. *Journal of Positive Psychology*, 4, 566–577. doi:10.1080/17439760903157133
- Maddi, S. R., & Hess, M. (1992). Personality hardiness and success in basketball. *International Journal of Sports Psychology*, 23, 360–368.
- Maddi, S. R., & Hightower, M. (1999). Hardiness and optimism as expressed in coping patterns. *Consulting Psychology Journal*, 51, 95–105. doi:10.1037/1061-4087.51.2.95
- Maddi, S. R., Kahn, S., & Maddi, K. L. (1998). The effectiveness of hardiness training. *Consulting Psychology Journal*, 50, 78–86. doi:10.1037/1061-4087.50.2.78
- Maddi, S. R., & Khoshaba, D. M. (1994). Hardiness and mental health. *Journal of Personality Assessment*, 63, 265–274. doi:10.1207/s15327752jpa6302_6
- Maddi, S. R., & Khoshaba, D. M. (2001). *HardiSurvey III-R: Test development and internet instruction manual*. Newport Beach, CA: Hardiness Institute.
- Maddi, S. R., & Khoshaba, D. M. (2005). *Resilience at work: How to succeed no matter what life throws at you*. New York, NY: Amacom.
- Maddi, S. R., & Kobasa, S. C. (1984). *The hardy executive: Health under stress*. Homewood, IL: Dow Jones-Irwin.
- Maddi, S. R., Matthews, M. D., Kelly, D., Villarreal, B., & White, M. (2012). Hardiness and grit predict performance and retention of USMA cadets. *Military Psychology*, 24(1), 19–28.
- Maddi, S. R., Savino, S. C. M., Bach, S. C., Saifabad, N., Shirmohammadi, M., & Brown, S. D. (2015). Hardiness is negatively related to gambling. Manuscript submitted for publication.
- Maddi, S. R., Wadhwa, P., & Haier, R. J. (1996). Relationship of hardiness to alcohol and drug use in adolescents. *American Journal of Drug and Alcohol Abuse*, 22, 247–257. doi:10.3109/00952999609001657
- Peters, T. (1988). *Thriving on chaos*. New York, NY: Knopf.
- Sinclair, R. R., & Tetrick, L. E. (2000). Implications of item wording for hardiness structure, relations to neuroticism, and stress buffering. *Journal of Research in Personality*, 34, 1–5. doi:10.1006/jrpe.1999.2265

Suggested Reading

- Bartone, P. T. (1999). Hardiness protects against war-related stress in army reserve forces. *Consulting Psychology Journal*, 51, 72–82. doi:10.1037/1061-4087.51.2.72
- Funk, S. C. (1992). Hardiness: A review of theory and research. *Health Psychology*, 11, 335–345. doi:10.1037/0278-6133.11.5.335
- Maddi, S. R. (2002). The story of hardiness: Twenty years of theorizing, research, and practice. *Consulting Psychology Journal*, 54, 175–185. doi:10.1037/1061-4087.54.3.173
- Orr, E., & Westman, M. (1990). Hardiness as a stress moderator: A review. In M. Rosenbaum (Ed.), *Learned resourcefulness: On coping skills, self-control, and adaptive behavior* (pp. 64–94). New York, NY: Springer-Verlag.
- Ouellette, S. C. (1993). Inquiries into hardiness. In L. Goldberger & S. Bresnitz (Eds.), *Handbook of stress: Theoretical and clinical aspects* (2nd ed., pp. 77–100). New York, NY: Free Press.

Physical Activity Monitoring

Birte von Haaren-Mack¹, Johannes B.J. Bussmann², and
Ulrich W. Ebner-Priemer³

¹*German Sport University Cologne, Köln,, Nordrhein-Westfalen, Germany*

²*Erasmus MC University Medical Center Rotterdam, CA, Rotterdam,
South Holland, The Netherlands*

³*Karlsruhe Institute of Technology, Karlsruhe, Germany*

Although behavior is central to psychology in almost any definition of the field, research on actual behavior has reached a minimum in the field of psychology. This has been criticized several times, for example, by Baumeister and colleagues' seminal paper titled "Psychology as the Science of Self-Reports and Finger Movements: Whatever Happened to Actual Behavior?" In this chapter we will focus on the assessment of actual behavior. More precisely, we will focus on the assessment of physical behavior as an important subdomain of behavior—which encompasses overt postures, movements, and physical activities in everyday life—and its resultant physiological consequences (e.g., energy expenditure [EE]).

Measurement Principles and Technology

With the technological developments of the last 20 years, ambulatory activity monitoring—defined as a measurement strategy to assess physical activity, posture, and movement patterns continuously in everyday life—has become more and more feasible and is therefore increasingly applied to various research questions and clinical (feedback) applications. Activity monitors based on accelerometry have become most popular for assessing ambulatory activity; accelerometry can be used in almost every target group with relatively low participant burden and has contributed to advanced knowledge in epidemiological activity research over recent years.

Accelerosensors measure acceleration—that is the change in velocity over time. They are built into accelerometers, devices that are specially designed to measure acceleration, and into a variety of other products, like smartphones, cars, and cameras. Accelerosensors can

be used to describe the frequency, intensity, type, and time of physical activity. Although not the main focus of this chapter, it is important to know that accelerometer signals can also be used to quantify movement characteristics, such as walking speed, step length, and smoothness of movement. Furthermore, most current accelerometers also measure a component of gravitational acceleration; because the magnitude of this component depends on the angular position of the sensor, these accelerometers can also provide information on the angular position of the body segment the accelerometer is attached to. As a result, accelerometers can also be used to automatically detect body postures and movements (Chen, Janz, Zhu, & Brychta, 2012).

Most modern devices are based on a piezoelectric (sensing element causes displacement of seismic mass), capacitive (proof mass between two electrodes), or piezoresistive (piezoresistors in the cantilever beam and proof mass) microscopic technology (microelectromechanical systems [MEMS]). The main advantages of the new technology are increased sensitivity, reduced size and costs, expanded onboard memory, extended battery life, and wireless communication (Chen et al., 2012). Due to technological advancements, data can be collected for a longer period at higher time resolutions, and unfiltered raw data can be stored instead of integrating and filtering data across time intervals (epochs). This major improvement enhances the comparability of data assessed with different sensors and enables the reanalysis of data with improved algorithms because the analysis is conducted offline (Chen et al., 2012).

Although technological developments rendered counts as primary accelerometer output unnecessary, many accelerometers still have counts per unit time as their main output. Counts are arbitrary units without a physical meaning, but they express the volume of movement within a defined time interval (epoch). The term “volume” indicates that the number of counts per time interval depends on the amount (or duration) of physical activity within that time interval and its intensity. Although all of the sensors measure the same underlying construct, namely, acceleration, results are hardly comparable between studies and devices from different manufacturers and even between different generations of the same accelerometer (Chen et al., 2012). Differences in outcomes are due to differences regarding transducers, sampling frequencies, amplifiers, and filtering (Chen et al., 2012). Thus, when conducting an accelerometer study, it is highly recommended to use a sensor that records unfiltered raw such as acceleration (m/s^2) or the gravitational constant ($g = 9.81 \text{ m/s}^2$). When measuring raw signals, the sampling and storage rate have to be considered carefully. For example, accelerometers should provide a sampling rate that, according to the Nyquist principle, should be at least twice the highest frequency of assessed movements (Chen et al., 2012); thus, for example, for an arm movement of 25 Hz, the sampling rate has to be at least 50 Hz. Common accelerometers have sampling rates between 1 and 100 Hz (Chen et al., 2012).

In sum, when choosing a suitable accelerometer, the choice should strongly depend on the research question, the topic or outcome of interest, and the target group’s behavioral characteristics of interest (for a comparison in accelerometers, see Yang & Hsu, 2010). For example, children’s movement behavior is spontaneous with short intense intervals of high intensity, while older adults’ movement behavior is characterized by low intensity activity and long periods of sedentary behavior. Furthermore, factors such as reliability, validity, sensitivity, range, resolution, accuracy, and the precision of a device are important in the decision regarding which accelerometer to use. Finally, aspects such as battery length, capacity for data storage, and options of wear position should be considered (Chen et al., 2012; Matthews, Hagströmer, Poer, & Bowles, 2012).

Fields of Application

Accelerometry can be applied to various fields, for example, public health, elderly/falls research, rehabilitation, and other medical specialties. For the purpose of the present entry, we will focus on exemplary applications in the field of clinical and health psychology.

The important role of decreased or increased physical activity in many psychological disorders such as attention deficit hyperactivity disorder (ADHD), schizophrenia, major depression, or bipolar disorders has been underlined by Tryon in the early 2000s. For example, psychomotor retardation is a defining criterion in major depressive disorders. Volkers et al. (2003) reported a lower level of motor activity during waking hours and a higher level of motor activity during sleep. Furthermore, the authors observed longer periods of inactivity during waking hours and decreased immobility during sleep. Reichert et al. (2015) detected motor retardation of depressed patients both in gait and gestures. Furthermore, lower levels of motor activity amplitude and frequency indicated a reduced and decelerated activity denoting that patients walked both less and slower.

In addition, ambulatory activity monitoring has advanced knowledge regarding situation-specific characteristics of hyperactivity in children with ADHD, which could not have been identified with self-report measures. For example, in the early 1980s, Porrino and colleagues have already found increased activity during structured school tasks but not during free time in children with ADHD compared with healthy children. Teicher (1995) examined the distribution of activities during the day and showed that children with ADHD generally had few periods of low-level activity. They assumed that ADHD is characterized more by the relative absence of periods of rest than the presence of extreme activity. Using an interactive ambulatory activity feedback system, Tryon, Tryon, Kazlauskys, Gruen, and Swanson (2006) were able to decrease motor excess in children with ADHD during school periods.

Two systematic reviews of Reed and colleagues between 2006 and 2009 revealed that momentary and regular physical activity have been repeatedly associated with positive mood in studies using a between-subject perspective and retrospective self-report measures. By using accelerometry to assess unstructured physical activity and by using electronic diaries to repeatedly assess mood within subjects in everyday life, ambulatory assessment studies have recently contributed to advanced knowledge on the activity–affect association. For example, Schwerdtfeger’s studies between 2008 and 2012 revealed that mood was associated with the preceding physical activity level and momentary mood state was associated with subsequent activity level. Furthermore, the activity–affect association appeared to be more complex in inactive people (von Haaren et al., 2013).

Objective Assessment Versus Self-Report

Although many self-report (e.g., questionnaires) and objective measures (e.g., accelerometers) aim to measure the same construct, namely, the amount or volume of physical activity, they are low to moderately correlated (Prince et al., 2008). Furthermore, the discrepancies between subjective and objective physical activity data are well illustrated in the results of studies analyzing the achievement of physical activity guidelines suggesting tremendously higher achievement of health-related exercise targets with subjective measures compared with accelerometer-based studies (Gabriel, Morrow, & Woolsey, 2012). Gabriel et al. (2012) have identified several

aspects that point out why the use of self-report instruments may induce different results compared with objective measures of physical activity: social desirability, reporting bias, misclassification bias and recall bias, differences between self-reports (with respect to psychometric properties, mode of administration, recall time frame), and differences in physical activity definitions.

First, reporting bias and recall bias are relevant problems when using self-reports, as they involve problems with cognitive processes that are necessary to gather activity information stored in the memory (Gabriel et al., 2012). In general, participants tend to overestimate the amount of physical activity performed (Prince et al., 2008).

Second, misclassification bias describes the problems of methods to adequately classify participants regarding their physical activity level (e.g., an insufficiently active person is categorized as meeting the guidelines). Therefore, it can lead to the underestimation of activity–health associations (Gabriel et al., 2012).

Finally, even differences between self-report instruments may impact results. For example, an interviewer-administered questionnaire with a shorter recall time frame may be more useful and valid in older adults, although it may not be practical and valid in another target group (Gabriel et al., 2012). In addition, social desirability that characterizes the tendency to present oneself based on perceived cultural norms was associated with overreporting of physical activity in self-report measures (Prince et al., 2008).

Instead of questioning the use of self-report *per se*, it may be important to understand that questionnaires and objective measures derived from accelerometry sometimes focus on conceptually different aspects of physical activity (Gabriel et al., 2012). For example, “fishing” as a hobby might contribute to the amount of physical activity from the perspective of questionnaires, but will not significantly contribute to the amount of physical activity as captured by activity monitors.

Two Perspectives: Within Versus Between

In general, research in physical behavior can focus on a between-subject or within-subject perspective. This is important to mention, as there is often confusion about these two perspectives. For example, a between-subject investigation on affect and physical activity can show that persons with a higher activity level have higher mood ratings. However, it does not evidence that a within-subject relation is true also, which means that a person has higher mood ratings when performing exercise compared with non-exercise situations. Between-subject investigations often focus on the frequency, intensity, type, and time of components of physical behavior (e.g., walking or being upright) and its relationship with health targets. These studies generally consider person-specific (e.g., age, sex, cultural, and social background) and environmental (e.g., region, facilities) variables of interest. However, they completely neglect the time dependency of activity. Said more simply, if my interest is in understanding why people do physical exercise, I need to assess in which specific situations they start exercising. Examples for within-subject investigations are dose–response relations or the reciprocal associations between physical behavior and psychological variables such as stress, emotions, self-concept, motives, and barriers to being physically active. Furthermore, physical behavior is addressed and evaluated in intervention studies and in studies that examine physiological variables that depend on or are confounded by physical activity, such as heart rate or heart rate variability.

Measurement Issues

Number of Measurement Days

Although it is known that physical activity varies between days, it is unclear how many days are exactly needed to account for between-day variability. The results of studies that tried to identify how many assessment days are necessary vary from 3 to 5 days in adults and 4–9 days in children (Trost, Mciver, & Pate, 2005) or from three 24-hr periods to 10 days or 2 weeks. Because physical activity varies between weekend and weekdays, at least one weekend day is required (Trost et al., 2005). It must be noted, however, that many factors will affect between-day variability and—because of that—the number of measurement days. Examples of this are the capacity of a person to vary his/her physical behavior (e.g., due to type of work, a person's health/fitness) and the physical behavior outcome of interest; some outcomes (e.g., ratio of usage left/right arm) might be more stable between days than others (e.g., number of steps taken).

Data Processing

Current software for initializing accelerometers generally allow user-friendly setting of start and end time. Depending on the research question, some authors recommend programming the start of recording at midnight of the first day the sensor has to be worn (Matthews et al., 2012). Even though data processing of accelerometer data is much easier nowadays, it is important to realize that initializing accelerometers and analyzing data still need some time and, more importantly, requires skilled personnel (Matthews et al., 2012).

Data check

To detect problems during the course of a study and eventually be able as a researcher to repeat data collection, data should be controlled in terms of quantity and quality immediately after data recording (Heil, Brage, & Rothney, 2012). The quality of accelerometer data can be limited by implausible data (e.g., due to malfunction of the sensor, outliers exceeding specific thresholds, tampering by participants) and missing data over a predetermined time interval (Chen et al., 2012; Heil et al., 2012). Today, preset user settings of the software can support the identification of these data quality aspects.

Non-wear periods

To identify periods during which the sensor was not worn, different recommendations are reported in the literature: non-wear times can be detected based on thresholds of a predetermined number of counts (e.g., zero counts over a predetermined time period). Recommended time periods to consider for zero counts vary considerably between 10 and 90 min (Choi, Liu, Matthews, & Buchowski, 2011). The identification of non-wear periods is not trivial, as zero counts can have different reasons such as not only sleep or sitting quietly or malfunction of the sensor but also removal of the sensor without a reason, or due to specific activities that do not allow wearing the sensor, such as swimming, other water activities, or showering (sensors are not waterproof) or heavy contact sports (risk of breaking; Mâsse et al., 2005). Only removal of the sensor would be “true” non-wear time.

To be able to identify times during which the sensor was not worn (see *Data check*) and to facilitate the interpretation of the accelerometer data, we suggest that participants should record the following information depending on the research question: time at which the

sensor is removed in the evening, bed/sleep time, wake time in the morning, time of attachment of the sensor in the morning, and times and activities during which the sensor was removed during the day.

Identifying a valid day

Researchers should think about how many hours of wear time they consider reasonable. Studies often require a wear time of 10 hr/day as a precondition for a valid day (Mâsse et al., 2005). In children, the required wear time for a valid day can be reduced by trend due to shorter waking times (Mâsse et al., 2005). In addition, it might be reasonable to differentiate between weekend and weekdays in terms of required wear time for a valid day (Mâsse et al., 2005). Using data imputation to account for invalid days, the values of valid days and those of partly valid days are used to extrapolate values for the invalid days (Mâsse et al., 2005).

Outcome Variables

With the development of saving unfiltered raw acceleration data, accelerometry allows generating manifold outcome variables ranging from counts to movement-specific or gait-specific variables (e.g., walking speed), energy expenditure, body posture, and movement categories such as walking, jogging, standing, climbing stairs, etc. While ambulatory monitoring is an objective technique to assess physical activity, posture, and movement, it is not automatically guaranteed that the method generates reliable and valid outcome variables. Thus, a major challenge of ambulatory activity monitoring is to operationalize parameters that truly, uniquely express the aspect of physical behavior that is of interest. For example, in a literature review in 2006 on biomechanical exposure at work, Mathiassen pointed out that an overall measure of activity level may not reflect the negative effect of repetitive operations (behavior) at work on musculoskeletal disorders. Instead, the distribution (variation) of specific activities over the day is interesting (see Suggested reading). Several categorizations exist, but Heil et al. (2012) suggest the following categorization of outcome variables:

- Movement-based volume variables are variables that are not transformed into physiological units, for example, acceleration in standard physical units (g, m/s²), counts/min, counts/day.
- Time-based amount variables (minutes of moderate to vigorous physical activity [MVPA]/day, sedentary behavior/day).
- Energy expenditure-based variables (total EE/day, AEE/day, EE during MVPA).
- Activity-based variables (steps, walking time, jogging/week).

Influencing Factors

To account for between-day variability, we mentioned considering the minimum number of measurement days in the “number of measurement days” section above. However, there are multiple systematic factors influencing physical activity behavior in everyday life. Besides influencing factors such as age, gender, weight, sociocultural status, and many others that are assessed on the between-person level, there are also aspects that have to be considered on a within-subject level when conducting ambulatory activity monitoring. First, physical activity levels are higher or lower depending on the weather (and the temperature) and the

season. Usually people are more physically active during spring and summer compared with autumn and winter. Second, physical activity levels vary between weekend and weekdays (Trost et al., 2005) and can also vary between vacations and times during which individuals have to work or go to school (Heil et al., 2012). Furthermore, the placement of the monitor is not only relevant to gather the behavior of interest but also a factor that can influence the results (Trost et al., 2005). Placement at the waist is recommended for whole body ambulatory activities of the large skeletal muscles, such as walking during everyday life, because a placement close to the center of mass is supposed (Yang & Hsu, 2010). Other placement options such as thigh (sedentary behavior/posture, differentiation between sitting and standing), wrist (detect subtle activity during sleep), or ankle (step count and gait pattern detection) can be used depending on the research question (Matthews et al., 2012; Yang & Hsu, 2010).

Reliability, Validity, and Reactivity

Ambulatory activity assessment can improve both validity and generalizability, as it enables the assessment of real behavior in the natural environment instead of assessing behavior in an artificial laboratory setting. Looking at the literature reveals, unfortunately, considerable discrepancies regarding validity and reliability. For example, the results for reliability of accelerometers obtained in laboratory settings have been reported to be high with values between 97.5 and 99.4% in studies by Tryon. However, Esliger and Tremblay (2006) examined the reliability of three common accelerometers and found high discrepancies between devices in intra- and inter-instrument variability (range: CV = 0.5 to >40%).

Results of validation studies show that accelerometers under- or overestimate energy expenditure of specific activities depending on the investigated target group, the type of devices that were used, and the activities that had to be performed in these studies (Calabró, Lee, Saint-Maurice, Yoo, & Welk, 2014; Trost et al., 2005). Accelerometers still show considerable discrepancies with the criterion measures such as indirect calorimetry in low intensity activities, a domain that is of growing interest for researchers (Calabró et al., 2014). Furthermore, postures and movement patterns are misclassified when patterns are similar such as in walking and climbing stairs and in situations involving various patterns at the same time.

From a methodological perspective, the validity outcomes of accelerometers depend on the validation protocols (how extended the protocols are; real life vs. laboratory), the population specificity, the reference instruments and outcome variables (energy expenditure, posture, activity type classification), the accelerometer device, the psychometric topic of study, and the applied statistics. Definitely, more studies are necessary to obtain a more extensive picture on these issues. Common problems regarding validity in monitoring movement behavior in everyday life are (a) a lack of insight into the setting, (b) the control of confounding variables, (c) personal background and motivation for performing or refraining from activities, and (d) sensor placement.

For example, irregular walking patterns may either represent a lack of balance while walking on a flat surface or an appropriate adaptation to an uneven pavement. In addition, an upcoming exam can cause a low activity level as can laziness of a person. Furthermore, various movements cannot be detected adequately or require additional sensors, such as arm movements, cycling, or swimming. Moreover, the assessment of static activities, the carrying of additional load, and the differentiation between sedentary behavior and activities of low intensity are

problematic. For example, the differentiation between sitting and standing requires either various sensors, an integrated inclinometer or the placement of one sensor at the thigh that in turn induces less accurate data for whole body movements. Thus, the combination of accelerometry with electronic diaries to assess context information such as temperature, social contact, location, or light may help to identify situations and interpret the observed behavior (Ebner-Priemer, Koudela, Mutz, & Kanning, 2013).

The findings for reactivity to ambulatory activity monitoring are heterogeneous as well. For example, in one of Ebner-Priemer's studies in 2007, participants reported that they were minimally affected by 24-hr ambulatory activity monitoring. In addition, participants' ratings of distress and reactivity caused by the device were minimal. Several studies of Clemes and colleagues between 2008 and 2009 revealed that wearing an unsealed pedometer and displaying daily steps induced the highest reactivity compared with wearing an unsealed pedometer without displaying recorded steps or wearing a sealed pedometer. Dösegger et al. (2014) revealed an overall reactivity effect of approximately 5% in children and adolescents in response to accelerometers, while they reported no reactivity in other samples, for example, in a young active student sample. Additionally, van Sluijs, van Poppel, Twisk, and van Mechelen (2006) reported measurement effects in response to self-report but not in response to accelerometer measures in a randomized controlled trial. Furthermore, wearing a multichannel system did not affect the amount of wheelchair driving in patients with spinal cord injuries; however, patients reported some burden (Bussmann et al., 2010).

Due to the heterogeneous findings, it is not trivial to give general recommendations regarding reactivity. Allowing for at least 1 day of familiarization and a random assignment of start days, as recommended by Dösegger et al. (2014) when measuring habitual physical activity with accelerometry, it might not be feasible for a highly burdensome multiple device assessment procedure, which sometimes even lasts less than 24 hr. However, interviewing the participants after the monitoring period about burden and reactivity might be helpful.

The Future of Ambulatory Activity Monitoring in Psychology: Combining Accelerometry With Electronic Diaries

For psychology research, the association between psychological variables such as motivational processes, habits, and/or affective states is important to explain physical behavior in everyday life. Researchers often face the problem that the behavior of interest is rare: many people perform few active episodes because they are mainly sedentary. From a statistical point of view, a minimal number of active episodes are required to obtain sufficient variance or covariation. Recent studies addressing the association between affective states and physical activity in everyday life found a significant association between positive affect and physical activity but did not find an association between negative affect and physical activity. These results may be explained by different time courses of positive affect, negative affect, and physical activity, more specifically the effects on negative affective states that might not be parallel but shifted.

Activity-triggered e-diaries, sometimes also called interactive multimodal ambulatory monitoring, may be advantageous to understanding the relationship between actual physical behavior and affective states. By changing the affective state assessment strategy from a random or fixed mode to an interactive mode, interesting episodes are detected in real time and are used as triggers for the assessment of psychological variables and contextual information. Using this approach, variance can be enhanced, as affective states can be assessed during

episodes of high physical activity as well as during episodes of low physical activity (Ebner-Priemer et al., 2013).

Due to the fast technological developments in information technology and telemedicine, the assessment of physical behavior using accelerometers and connecting them with other sensors, smartphones, and technical applications is in its infancy. Multiparametric sensor fusion and the use of additional signals such as skin temperature can deliver useful information (e.g., whether a sensor is worn or not) and thus enhance the differentiation between inactivity and compliance. Additional contextual information that can be obtained via smartphones or Global Positioning System (GPS) signals delivers useful information about the situation, the social interaction, or the interaction with other devices. Since some activities take place at specific locations, activity classification can be improved. The various functions of smartphones such as video and audio options will offer unlimited opportunities for researchers but will also demand a reconsideration of how participants can be protected in terms of ethical fundamentals in future studies.

Author Biographies

Birte von Haaren-Mack is a postdoctoral research associate at the Department of Health and Social Psychology of the German Sport University Cologne. The focus of her research is examining psychophysiological effects of physical activity on stress and the relationship between physical activity and affect in everyday life; to assess these constructs in everyday life, she uses the methodology of ambulatory assessment, especially electronic diaries, accelerometry, and ambulatory ECG.

Johannes B.J. Bussmann is an associate professor at the Department of Rehabilitation Medicine, Erasmus MC, Rotterdam, Netherlands. The focus of his research is understanding the consequences of chronic conditions on physical behavior, the development and validation of objective devices and outcomes related to physical behavior, and the development and evaluation of treatment strategies. He is the president of the International Society for the Measurement of Physical Behavior (ISMPB) and board member of the Society of Ambulatory Assessment.

Ulrich W. Ebner-Priemer is a professor at the Institute for Sport and Sport Science at the Karlsruhe Institute of Technology (KIT), Germany. The focus of his research is characterized by its methodological focus on ambulatory assessment. Phenomena of interest are studied in everyday life (real life) in real time using psychophysiological methods (objective) and time-sensitive analysis (dynamics). He is currently the president of the SAA.

References

- Bussmann, J. B. J., Kikkert, M. A., Sluis, T. A. R., Bergen, M. P., Stam, H. J., & Vaccaro, A. R. (2010). Effect of wearing an activity monitor on the amount of daily manual wheelchair propulsion in persons with spinal cord injury. *Spinal Cord*. doi:<http://doi.org/10.1038/sc.2009.72>
- Calabró, M., Lee, J., Saint-Maurice, P. F., Yoo, H., & Welk, G. J. (2014). Validity of physical activity monitors for assessing lower intensity activity in adults. *International Journal of Behavioral Nutrition and Physical Activity*, 11(1), 119. <http://doi.org/10.1186/s12966-014-0119-7>
- Chen, K., Janz, K. F., Zhu, W., & Brychta, R. J. (2012). Redefining the roles of sensors in objective physical activity monitoring. *Medicine and Science in Sports and Exercise*, 44(Suppl 1), S13–S23. <http://doi.org/10.1249/MSS.0b013e3182399bc8>

- Choi, L., Liu, Z., Matthews, C., & Buchowski, M. (2011). NIH public access. *Medicine & Science in Sports & Exercise*, 43(2), 357–364. <http://doi.org/10.1249/MSS.0b013e3181ed61a3>
- Dösegger, A., Ruch, N., Jimmy, G., Braun-Fahrländer, C., Mäder, U., Hänggi, J., ... Bringolf-Isler, B. (2014). Reactivity to accelerometer measurement of children and adolescents. *Medicine & Science in Sports & Exercise*, 46(6), 1140–1146. <http://doi.org/10.1249/MSS.0000000000000215>
- Ebner-Priemer, U. W., Koudela, S., Mutz, G., & Kanning, M. (2013). Interactive multimodal ambulatory monitoring to investigate the association between physical activity and affect. *Frontiers in Psychology*, 3, 596. <http://doi.org/10.3389/fpsyg.2012.00596>
- Esliger, D. W., & Tremblay, M. S. (2006). Technical reliability assessment of three accelerometer models in a mechanical setup. *Medicine and Science in Sports and Exercise*, 38(12), 2173–2181. <http://doi.org/10.1249/01.mss.0000239394.55461.08>
- Gabriel, K. K. P., Morrow, J. R., & Woolsey, A. L. T. (2012). Framework for physical activity as a complex and multidimensional behavior. *Journal of Physical Activity & Health*, 9(Suppl 1), S11–S18.
- Heil, D. P., Brage, S., & Rothney, M. P. (2012). Modeling physical activity outcomes from wearable monitors. *Medicine and Science in Sports and Exercise*, 44, 50–60. <http://doi.org/10.1249/MSS.0b013e3182399dcc>
- Masse, L. C., Fuemmeler, B. F., Anderson, C. B., Matthews, C., Trost, S. G., Catellier, D. J., & Treuth, M. (2005). Accelerometer data reduction: A comparison of four reduction algorithms on select outcome variables. *Medicine and Science in Sports and Exercise*, 37, S544–S554. <http://doi.org/10.1249/01.mss.0000185674.09066.8a>
- Matthews, C., Hagströmer, M., Pober, D. M., & Bowles, H. R. (2012). Best practices for using physical activity monitors in population-based research. *Medicine and Science in Sports and Exercise*, 44(Suppl 1), S68–S76. <http://doi.org/10.1249/MSS.0b013e3182399e5b>
- Prince, S., Adamo, K., Hamel, M., Hardt, J., Gorber, S., & Tremblay, M. (2008). A comparison of direct versus self-report measures for assessing physical activity in adults: A systematic review. *International Journal of Behavioral Nutrition and Physical Activity*, 5, 56. <http://doi.org/10.1186/1479-5868-5-56>
- Reichert, M., Lutz, A., Deuschle, M., Gilles, M., Hill, H., Limberger, M. F., & Ebner-Priemer, U. W. (2015). Improving motor activity assessment in depression: Which sensor placement, analytic strategy and diurnal time frame are most powerful in distinguishing patients from controls and monitoring treatment effects. *PLoS One*, 10(4), e0124231. <http://doi.org/10.1371/journal.pone.0124231>
- Teicher, M. H. (1995). Actigraphy and motion analysis: New tools for psychiatry. *Harvard Review of Psychiatry*, 3(1), 18–35.
- Trost, S. G., Mciver, K. L., & Pate, R. R. (2005). Conducting accelerometer-based activity assessments in field-based research. *Medicine and Science in Sports and Exercise*, 37(Suppl. 11), 531–543. <http://doi.org/10.1249/01.mss.0000185657.86065.98>
- Tryon, W. W., Tryon, G. S., Kazlauskas, T., Gruen, W., & Swanson, J. M. (2006). Reducing hyperactivity with a feedback actigraph: Initial findings. *Clinical Child Psychology and Psychiatry*, 11(4), 607–617. <http://doi.org/10.1177/1359104506067881>
- van Sluijs, E. M. F., van Poppel, M. N. M., Twisk, J. W. R., & van Mechelen, W. (2006). Physical activity measurements affected participants' behavior in a randomized controlled trial. *Journal of Clinical Epidemiology*, 59(4), 404–411. <http://doi.org/10.1016/j.jclinepi.2005.08.016>
- Volkers, A. C., Tulen, J. H. M., Van Den Broek, W. W., Bruijn, J. A., Passchier, J., & Peplinkhuizen, L. (2003). Motor activity and autonomic cardiac functioning in major depressive disorder. *Journal of Affective Disorders*, 76(1–3), 23–30. [http://doi.org/10.1016/S0165-0327\(02\)00066-6](http://doi.org/10.1016/S0165-0327(02)00066-6)
- von Haaren, B., Loeffler, S. N., Haertel, S., Anastasopoulou, P., Stumpp, J., Hey, S., & Boes, K. (2013). Characteristics of the activity-affect association in inactive people: An ambulatory assessment study in daily life. *Frontiers in Psychology*, 4(April), 163. <http://doi.org/10.3389/fpsyg.2013.00163>
- Yang, C.-C., & Hsu, Y.-L. (2010). A review of accelerometry-based wearable motion detectors for physical activity monitoring. *Sensors*, 10(8), 7772–7788. <http://doi.org/10.3390/s100807772>

Suggested Reading

- Clemes, S. A., & Parker, R. A. A. (2009). Increasing our understanding of reactivity to pedometers in adults. *Medicine and Science in Sports and Exercise*, 41(21), 674–680. <http://doi.org/10.1249/MSS.0b013e31818cae32>
- Crouter, S. E., Churilla, J. R., & Bassett, D. R. (2006). Estimating energy expenditure using accelerometers. *European Journal of Applied Physiology*, 98, 601–612. <http://doi.org/10.1007/s00421-006-0307-5>
- Ebner-Priemer, U. W., & Trull, T. J. (2009). Ambulatory assessment: An innovative and promising approach for clinical psychology. *European Psychologist*, 14(2), 109–119. <http://doi.org/10.1027/1016-9040.14.2.109>
- Mathiassen, S. E. (2006). Diversity and variation in biomechanical exposure: What is it, and why would we like to know? *Applied Ergonomics*, 37(4), 419–427. <http://doi.org/10.1016/j.apergo.2006.04.006>
- Tryon, W. W. (2006). Activity measurement. In M. Hersen (Ed.), *Clinician's handbook of adult behavioral assessment* (pp. 85–120). New York, NY: Academic Press.

Physical and Social Pain: Twin Challenges to Well-Being in Adult Life

John A. Sturgeon¹, Alex J. Zautra², and Beth D. Darnall³

¹*Center for Pain Relief, University of Washington, Seattle, WA, USA*

²*Psychology Department, Arizona State University, Tempe, AZ, USA*

³*Department of Anesthesiology, Perioperative, and Pain Medicine, Stanford University School of Medicine, Stanford, CA, USA*

Your pain is the breaking of the shell that encloses your understanding.

—Kahlil Gibran

Shared Mechanisms Between Social and Physical Pain

Pain is a multidimensional construct, consisting of sensory, emotional, and cognitive aspects. Given its complex nature, the experience of pain is subject to a variety of physical and psychological factors. Additionally, pain has a profound and mutually influential relationship with the social world. Social factors may be considered a “double-edged sword” in the context of pain. For instance, social factors have the capacity to increase vulnerability to pain-related dysfunction or to contribute to healthier responses to pain.

Recent evidence supports the model of shared neural activation between nonphysical pain arising from social causes (e.g., exclusion, ostracism, and loss) and physical pain. In a seminal study, Eisenberger, Lieberman, and Williams (2003) reported that, after being exposed to social exclusion experimentally, participants demonstrated greater levels of activation of both the dorsal anterior cingulate cortex (ACC) and the right ventral prefrontal cortex, two brain regions previously associated with pain-related emotional distress. Subsequent research has also implicated the anterior insula, which appears to play a key role in the experience of physical and social pain (Eisenberger, 2012). Craig (2015) has found substantial evidence that pain is an emotion that is indicative of the brain’s self-regulatory response to disturbance in homeostasis. Like other feelings, pain has both sensory and motor components integrated within the

anterior insula and adjacent neural structures. These patterns of activation have been implicated in social rejection in experimental and real-life settings. For instance, individuals who had recently been rejected by a romantic partner evidenced neural activation similar to physical pain when shown pictures of the person who had rejected them (Kross, Berman, Mischel, Smith, & Wager, 2011). Additionally, similar patterns of regional activation have been noted in individuals viewing pictures of recently lost loved ones, suggesting that social pain is not specific to social rejection and can include other socially based sources of distress, such as loss. In essence, when we refer to hurt that arises from social interactions, we are not speaking metaphorically. Social and physical pain belong within the same family of neurophysiological responses that alert the mind to modify its current status.

Researchers have explored the potential evolutionary value of social pain, suggesting that social pain may have promoted physical safety: if an organism is separated from its group, it is exposed to greater predatory and other environmental threats (MacDonald & Leary, 2005). Thus, exclusion or rejection from one's group may have developed to signal imminent danger, similar to the experience of acute physical pain, which signals potential tissue damage and serves a biologically necessary purpose. Linguistically, socially painful experiences are described similarly to physical pain, suggesting that they are often experienced in a similar fashion. Importantly, these theoretical assertions have been supported by experimental data. For instance, social exclusion has been found to promote greater physical pain (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006). Furthermore, in this same study, baseline cutaneous heat pain sensitivity was directly related to the level of social distress after exclusion. This bidirectional relationship provides further evidence that, in the evolutionary sense, prosocial ties confer safety and that the loss of social relationships is indexed neurally by the human "alarm system"—pain—to signal a threat.

The immune system is implicated in social and physical pain, thereby revealing an additional shared mechanism. Indeed, pro-inflammatory cytokines—markers of the immune system—have been associated with physical pain and may underlie mood difficulties in chronic pain. The inflammatory response is a primary biological response to tissue damage and serves to protect a wound through tissue swelling, slowing use of damaged areas with pain registers, and slowing psychomotor energy expenditures through signaling fatigue. Responding as if the body were harmed, increased circulation of pro-inflammatory cytokines has been found following social stressors such as social exclusion (Slavich, Way, Eisenberger, & Taylor, 2010). Additionally, researchers found that the pro-inflammatory cytokine interleukin-6 (IL-6) mediated the relationship between social exclusion and later feelings of depression (Eisenberger, Inagaki, Rameson, Mashal, & Irwin, 2009). Interestingly, when inflammation was induced in participants via exposure to an endotoxin, they reported greater feelings of social disconnection and depression (Eisenberger, Inagaki, Mashal, & Irwin, 2010), again underscoring a bidirectional relationship between a physiological response to a threat or harm (inflammation) and social exclusion. In fact, these authors reported that the relationships between inflammatory markers and depressed mood were fully explained by perceived social disconnection.

Recent intervention studies showed that targeting these shared underlying systems may address both physical and social pain. DeWall and colleagues (Dewall et al. 2010) found that a 3-week course of acetaminophen, a common nonsteroidal anti-inflammatory analgesic thought to act on the central nervous system, also decreased feelings of social pain in response to social exclusion through reduced activity in the dorsal ACC and anterior insula. Individuals facing chronic social stress or isolation may be vulnerable to reliance on analgesic opioids to cope with this social pain. Indeed, polymorphisms of the μ -opioid receptor gene, traditionally implicated in physical pain processes, have been found to be predictive of greater social pain in response to

exclusion, as well as predicting greater ratings of physical pain unpleasantness (Way, Taylor, & Eisenberger, 2009). Accordingly, a greater emphasis on social therapeutic targets may be most effective for the dual purpose of treating pain and mitigating addiction risks.

Social Contributors to Vulnerability and Distress in Chronic Pain

Traditionally, pain studies have focused on intraindividual processes, such as cognition and emotion. However, several factors originating in the social environment may alter both the experience of pain and subsequent coping responses to pain. It has been suggested that one of the etiologic factors in chronic physical pain may be chronic social pain, caused by ostracism, rejection, conflict, or isolation. These factors may increase vulnerability to pain and heightened emotional or behavioral responses to pain. For example, feeling ostracized or rejected may contribute to greater pain intensity following noxious thermal cutaneous stimulation (Eisenberger et al., 2006). Feelings of loneliness also appear to predispose individuals to greater pain by increasing negative thoughts about pain. Similarly, individuals with chronic pain who face more frequent stressful or conflictual interactions with others tend to report greater pain intensity (Faucett & Levine, 1991). Further, specific interpersonal communications may exacerbate pain: receiving social feedback of a personal failure predicts greater pain intensity than feedback of success after an intelligence test (Hout, Vlaeyen, Peters, Engelhard, & Hout, 2000). Thus, the presence of social stress or conflict appears to have negative implications for pain perception.

The consequences of negative social factors may also have long-term consequences by altering individual trajectories of the development of pain. Perhaps the best-known example of this phenomenon is the effect of significant social stressors in early life; exposure to traumatic events, particularly episodes of abuse, predisposes individuals to risks for future chronic pain (Davis, Luecken, & Zautra, 2005) and to medically unexplained pain later in life. Although the effect sizes from meta-analytic studies are relatively modest, the effect is nevertheless robust across studies and populations (Davis et al., 2005). It has been hypothesized that early and severe social stressors may sensitize the central nervous system, thereby kindling a diathesis for poorer outcomes later in life (Rutten et al., 2013). Alternatively, early life trauma may increase exposure to future stressful social environments, thereby prolonging these negative effects (Davis et al., 2005). Though the exact mediators are unclear, exposure to early and severe social stress appears to have profound implications for pain, both immediately and in the long term.

In addition to the direct effects of social stress on pain perception, the social environment may indirectly predispose individuals to poorer pain outcomes through changes in behavior, cognition, and emotional states. Feelings of social exclusion undermine effective coping strategies and self-esteem and potentiate aggression against others. Exclusion also discourages prosocial behavior and efforts to reconnect with others after exclusion, thereby entrenching a pattern of social avoidance that compromises the potential benefits of an extant social support network (Maner, DeWall, Baumeister, & Schaller, 2007). Notably, these patterns have effects lasting beyond the initial social struggles; similar to “pain memories,” the recall of a socially painful experience may be sufficient to inspire social and emotional pain, increase feelings of aggression, and lead an individual to view a social relationship less favorably (Klages & Wirth, 2014). These findings are notable, as prior evidence suggests that a possible mechanism promoting long-term vulnerability to mood disturbance in chronic pain is a heightened and persistent fear of rejection (Ehnavall, Mitchell, Hadzi-Pavlovic, Malhi, & Parker, 2009).

Social threat has been a recent focus of empirical inquiry, particularly with regard to how it affects pain-related learning and coping. At present, the evidence in this area is mixed. Some experimental studies have found that a threatening social context, defined as a situation in which environmental cues signal either social threat (e.g., angry faces) or potential physical pain, facilitates the acquisition of pain-related fear (Karos, Meulders, & Vlaeyen, 2015). However, the fear elicited from these social contexts may have domain-specific effects: in an experimental setting, fear of physical pain, but not fear of social pain, was significantly predictive of greater pain intensity ratings in response to a cold water pain induction task, while only fear of social pain was found to predict social pain ratings after a social exclusion paradigm (Riva, Williams, & Gallucci, 2014). In both of these studies, the effects of a threatening social context had indirect effects on pain perception. More specifically, it was fear of pain, rather than general perception of a threat, that predicted increased pain intensity. These studies highlight the importance of proximal factors, such as current mood states, in the pain experience.

Social problems are known risk factors for mood dysregulation, higher levels of depression, anger, and negative affect (see Sturgeon, Zautra, & Arewasikporn, 2014). In turn, these negative emotional states may increase pain perception; this effect has been noted primarily for high-activation negative emotional states such as fear, anxiety, and anger. There is also evidence for a potentiating effect of depression on pain intensity, though this effect may be due to cognitive appraisals of pain, such as viewing pain in a catastrophic light (Berna et al., 2010). The mechanisms underlying the effects of negative mood on pain remain undefined but have been theorized to include increased muscle tension and overtaxing of the endogenous opioid system, which is responsible for pain perception and control in the body.

In addition to interpersonal conflict or threat, other social risks factors, such as limited or absent social resources or relationships, contribute to worsened pain and poorer long-term health for individuals with chronic pain. Chronic pain is associated with a susceptibility to social withdrawal (avoidance) when pain is high, which may decrease exposure to potential sources of positive social engagement, worsening the emotional consequences of pain (see Sturgeon et al., 2014). Isolation is also a risk factor for several pain-relevant difficulties, such as depression and decreased physical function. More generally, there is also evidence that loneliness may affect genome-wide transcriptional activity, leading to dysregulated glucocorticoid activity and potentially predisposing individuals facing chronic difficulties with loneliness to long-term inflammatory disease (Cole et al., 2007). Given that inflammatory dysregulation may contribute to difficulties with chronic pain in some instances, social isolation may be a significant risk factor for both the incidence of chronic pain and poorer adaptive responses once chronic pain has developed. In sum, social stressors, whether due to social conflict or challenge or to a lack of positive social interactions, constitute a significant source of vulnerability for individuals facing pain in the short and long term.

Social and Environmental Factors Promoting Effective Pain Adaptation

Although challenges in one's social environment can worsen the experience of pain, there is also evidence that the social world can promote positive adaptation to pain. Resilience to pain has been defined by an individual's capacity to effectively maintain meaningful function, recover from the deleterious effects of pain, and develop new capacities or knowledge after managing pain for a period of time (see Sturgeon & Zautra, 2010). A key contributor to resilience is social relationships, and earlier definitions highlighted the importance of individuals'

utilization of social resources to recover from stress. In this vein, the frequency, nature, and quality of social interactions can powerfully affect an individual's ability to cope with chronic pain. An important aspect of pain resilience is positive emotion, which is often fostered through positive and meaningful engagements with other people.

Positive affect has been shown to promote resilient coping with chronic pain, as it has demonstrated myriad benefits for individuals experiencing acute or chronic pain. Positive emotions facilitate recovery from stress, bolster immune function, and improve cognitive flexibility and problem solving under stress (Fredrickson, 2001). Positive emotions have also been found to attenuate both clinical and experimental pain and to protect against pain catastrophizing (Hood, Pulvers, Carrillo, Merchant, & Thomas, 2012). Importantly, positive affect confers enduring protection, as it may inhibit the generalization of pain-related fear, a known risk factor in the prolongation of pain from acute to chronic states. Thus it stands to reason that individuals who effectively glean positive emotions from their relationships with others may be more resistant to the deleterious effects of pain. Indeed, positive or enjoyable interpersonal events have been shown to increase positive affect and protect against later states of fatigue (see Sturgeon et al., 2014). Furthermore, extraversion, the personality trait most closely related to positive social engagement, is predictive of more effective pain coping in arthritis populations and has been shown to contribute to resilience in arthritis populations.

The positive effects of social factors may also directly influence pain experience and coping. In general, individuals who are more willing and able to seek out social support tend to exhibit greater individual resilience and report greater life satisfaction and lower levels of depression. Individuals with chronic pain also report reduced pain and show less activation of the central nervous system during a pain induction procedure when they are in the presence of their significant other (Montoya, Larbig, Braun, Preissl, & Birbaumer, 2004). These effects are observed even in the absence of a significant other: viewing a photo of a loved one has been shown to dampen ratings for experimentally induced pain (Eisenberger et al., 2011; Younger, Aron, Parke, Chatterjee, & Mackey, 2010). The potent effect of love on pain may be attributable to the attenuation of the common neural networks underlying physical and social pain, the engagement of neural networks associated with reward (Younger et al., 2010), or the interpretation of a loved one as a sign of physical safety (Eisenberger et al., 2011). Social support may also engage prefrontal cortex activity associated with prosocial behavior, which may serve as a protective factor against pain by buffering pain-relevant negative emotional states (Onoda et al., 2009). In the long term, social support also appears to have a protective effect, as individuals with rheumatoid arthritis receiving greater levels of social support report less pain and functional disability 5 years later (for a review, see Evers, Zautra, & Thieme, 2011).

The effects of social support are decidedly more nuanced than they might initially appear, however. Beyond the mere availability of friends and family who may provide support during periods of stress, an individual's perception of the quality of their social relationships may influence or determine whether such support fosters resilient coping. While both the quality and quantity of social support predict successful adaptation to pain in fibromyalgia, the quality of social support appears to play a greater role in determining physical and psychological functionality (Franks, Cronan, & Oliver, 2004). A social network that is too large or focused on aiding an individual in coping with their pain may actually reduce self-sufficiency and long-term pain coping (Franks et al., 2004). Indeed, certain individuals may value extra emotional or instrumental support over the reduction of pain or emotional distress. Thus, individuals who value social validation above continued meaningful function may successfully achieve these goals and inadvertently lead themselves to less adaptive methods of coping with pain.

Indeed, women with fibromyalgia whose primary goals relate to social validation (rather than self-sufficiency) report lower levels of perceived social support, higher levels of interpersonal conflict, and more negative life events (Hamilton, Karoly, & Zautra, 2005). Such findings point to the need to assess each individual's social values, needs, and priorities within the context of pain.

Similarly, the quality of interactions with one's social network plays a meaningful role in the promotion of effective pain adaptation. When solicitous responses to pain are more frequent (i.e., asking a spouse for help in managing pain), individuals with chronic pain commonly demonstrate greater vulnerability to disability, greater pain intensity, and poorer overall adjustment to pain. Conversely, more mindful and empathic spousal responses are associated with greater perceptions of spousal support. Though this area of research is relatively nascent, the value of emotional support may be enhanced by mutual prosocial behavior between an individual coping with pain and a loved one. This type of interaction is predictive of greater well-being, lower distress, and greater perceptions of relationship quality for both members of a romantic relationship.

Social Interventions for People with Chronic Pain

Can the troubled social relations of those with chronic pain conditions be reversed and the potential for positive social relations realized through carefully designed interventions? In some quarters the prevalent thought is that capacity for social connections is fixed in childhood, or at the latest, in early adulthood. Others have argued that social development can be advanced even into adulthood (e.g., Division of Behavioral and Social Research, National Institute on Aging, 2013; <https://www.nia.nih.gov/research/dbsr>). A research team led by one of the authors of this chapter (www.socialintelligenceinstitute.org) has developed an online social intelligence (SI) intervention that focuses on enhancing capacities of adults to develop the skills to sustain positive social relationships and the awareness of the value of sustainable social connections. The SI training program has been guided by the objective of the humanization of relationships, with lesson plans informed by latest evidence from social neuroscience and related fields with attention to cognitions that facilitate healthy social connections. However, the approach extends beyond cognitive models and behavioral principles to include attention to evidence of barriers to social-emotional development from adverse experiences in childhood and adult life and ways to move beyond those stressful experiences. Recent empirical evidence shows this type of program offers a promising resource for chronic pain patients and those who care for them (Zautra, Zautra, Gallardo, & Velasco, 2015).

Summary

Though models of pain have evolved over the years to incorporate a greater number of non-biological processes, including cognition, emotion, and behavior, relatively less attention has been paid to social factors that may influence perception and adaptation to pain. The social environment is a powerful influence on the experience of pain and may either promote or inhibit effective coping, depending on the circumstances. As the understanding of the neural, genetic, biological, and psychological factors at play in pain becomes more sophisticated, future models of pain and its relationships to all aspects of human functioning can be further enhanced by acknowledging the social context in which pain is experienced.

Author Biographies

John A. Sturgeon is an acting assistant professor and clinical psychologist at the Center for Pain Relief at the University of Washington Medical School. His research examines the physiological, psychological, and social factors that contribute to resilience and vulnerability in chronic pain and stress, as well as psychological interventions designed to promote effective adaptation to chronic pain.

Alex J. Zautra was a professor of clinical psychology at Arizona State University Foundation. He dedicated his career to the study of resilience and ways of enhancing adaptation to stress. His latest work included the development of interventions that further the humanization of social relations. His most recent book, with coauthors Reich and Hall, is *Handbook of Adult Resilience* by Guilford Press. He also authored *Emotions, Stress, and Health* published by Oxford University Press.

Beth D. Darnall is a clinical associate professor in the Department of Anesthesiology, Perioperative, and Pain Medicine in the Stanford University School of Medicine and faculty in the Stanford Systems Neuroscience and Pain Laboratory. Her research broadly focuses on psychosocial dimensions of pain, characterizing the psychophysical impacts of pain-related stressors and developing targeted psychological interventions. She is the author of *Less Pain, Fewer Pills* (Bull Publishing).

References

- Berna, C., Leknes, S., Holmes, E. A., Edwards, R. R., Goodwin, G. M., & Tracey, I. (2010). Induction of depressed mood disrupts emotion regulation neurocircuitry and enhances pain unpleasantness. *Biological Psychiatry*, 67, 1083–1090. doi:10.1016/j.biopsych.2010.01.014
- Cole, S. W., Hawkey, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in human leukocytes. *Genome Biology*, 8, R189. doi:10.1186/gb-2007-8-9-r189
- Craig, A. (2015). *How do you feel?: An interoceptive moment with your neurobiological self*. Princeton, NJ: Princeton University Press.
- Davis, D. A., Luecken, L. J., & Zautra, A. J. (2005). Are reports of childhood abuse related to the experience of chronic pain in adulthood? A meta-analytic review of the literature. *Clinical Journal of Pain*, 21, 398–405.
- Dewall, C. N., Macdonald, G., Webster, G. D., Masten, C. L., Baumeister, R. F., Powell, C., ... Eisenberger, N. I. (2010). Acetaminophen reduces social pain: Behavioral and neural evidence. *Psychological Science*, 21, 931–937. doi:10.1177/0956797610374741
- Ehnvall, A., Mitchell, P. B., Hadzi-Pavlovic, D., Malhi, G. S., & Parker, G. (2009). Pain during depression and relationship to rejection sensitivity. *Acta Psychiatrica Scandinavica*, 119, 375–382. doi:10.1111/j.1600-0447.2008.01316.x
- Eisenberger, N. I. (2012). The pain of social disconnection: Examining the shared neural underpinnings of physical and social pain. *Nature Reviews Neuroscience*, 13, 421–434. doi:10.1038/nrn3231
- Eisenberger, N. I., Inagaki, T. K., Mashal, N. M., & Irwin, M. R. (2010). Inflammation and social experience: An inflammatory challenge induces feelings of social disconnection in addition to depressed mood. *Brain, Behavior, and Immunity*, 24, 558–563. doi:10.1016/j.bbi.2009.12.009
- Eisenberger, N. I., Inagaki, T. K., Rameson, L. T., Mashal, N. M., & Irwin, M. R. (2009). An fMRI study of cytokine-induced depressed mood and social pain: The role of sex differences. *Neuroimage*, 47, 881–890. doi:10.1016/j.neuroimage.2009.04.040
- Eisenberger, N. I., Jarcho, J. M., Lieberman, M. D., & Naliboff, B. D. (2006). An experimental study of shared sensitivity to physical pain and social rejection. *Pain*, 126, 132–138. doi:10.1016/j.pain.2006.06.024

- Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, 302, 290–292. doi:10.1126/science.1089134
- Eisenberger, N. I., Master, S. L., Inagaki, T. K., Taylor, S. E., Shirinyan, D., Lieberman, M. D., & Naliboff, B. D. (2011). Attachment figures activate a safety signal-related neural region and reduce pain experience. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 11721–11726. doi:10.1073/pnas.1108239108
- Evers, A., Zautra, A., & Thieme, K. (2011). Stress and resilience in rheumatic diseases: A review of 25 years of research. *Nature Reviews Rheumatology*, 7, 409–415. doi:10.1038/nrrheum.2011.80
- Faucett, J. A., & Levine, J. D. (1991). The contributions of interpersonal conflict to chronic pain in the presence or absence of organic pathology. *Pain*, 44, 35–43. doi:10.1016/0304-3959(91)90144-m
- Franks, H. M., Cronan, T. A., & Oliver, K. (2004). Social support in women with fibromyalgia: Is quality more important than quantity? *Journal of Community Psychology*, 32, 425–438. doi:10.1002/jcop.20011
- Fredrickson, B. L. (2001). The role of positive emotions in positive psychology. The broaden-and-build theory of positive emotions. *American Psychologist*, 56, 218–226. doi:10.1037//0003-066x.56.3.218
- Hamilton, N. A., Karoly, P., & Zautra, A. J. (2005). Health goal cognition and adjustment in women with fibromyalgia. *Journal of Behavioral Medicine*, 28, 455–466. doi:10.1007/s10865-005-9013-8
- Hood, A., Pulvers, K., Carrillo, J., Merchant, G., & Thomas, M. (2012). Positive traits linked to less pain through lower pain catastrophizing. *Personality & Individual Differences*, 52, 401–405. doi:10.1016/j.paid.2011.10.040
- Hout, J. H., Vlaeyen, J. W., Peters, M. L., Engelhard, I. M., & Hout, M. A. (2000). Does failure hurt? The effects of failure feedback on pain report, pain tolerance and pain avoidance. *European Journal of Pain*, 4, 335–346. doi:10.1053/eujp.2000.0195
- Karos, K., Meulders, A., & Vlaeyen, J. W. (2015). Threatening social context facilitates pain-related fear learning. *Journal of Pain*, 16, 214–225. doi:10.1016/j.jpain.2014.11.014
- Klages, S. V., & Wirth, J. H. (2014). Excluded by laughter: Laughing until it hurts someone else. *Journal of Social Psychology*, 154, 8–13. doi:10.1080/00224545.2013.843502
- Kross, E., Berman, M. G., Mischel, W., Smith, E. E., & Wager, T. D. (2011). Social rejection shares somatosensory representations with physical pain. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 6270–6275. doi:10.1073/pnas.1102693108
- MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin*, 131, 202–223. doi:10.1037/0033-2909.131.2.202
- Maner, J. K., DeWall, C. N., Baumeister, R. F., & Schaller, M. (2007). Does social exclusion motivate interpersonal reconnection? Resolving the “porcupine problem”. *Journal of Personality and Social Psychology*, 92, 42. doi:10.1037/0022-3514.92.1.42
- Montoya, P., Larbig, W., Braun, C., Preissl, H., & Birbaumer, N. (2004). Influence of social support and emotional context on pain processing and magnetic brain responses in fibromyalgia. *Arthritis and Rheumatism*, 50, 4035–4044. doi:10.1002/art.20660
- Onoda, K., Okamoto, Y., Nakashima, K., Nittono, H., Ura, M., & Yamawaki, S. (2009). Decreased ventral anterior cingulate cortex activity is associated with reduced social pain during emotional support. *Social Neuroscience*, 4, 443–454. doi:10.1080/17470910902955884
- Riva, P., Williams, K. D., & Gallucci, M. (2014). The relationship between fear of social and physical threat and its effect on social distress and physical pain perception. *Pain*, 155, 485–493. doi:10.1016/j.pain.2013.11.006
- Rutten, B. P., Hammels, C., Geschwind, N., Menne-Lothmann, C., Pishva, E., Schruers, K., ... Wichers, M. (2013). Resilience in mental health: Linking psychological and neurobiological perspectives. *Acta Psychiatrica Scandinavica*, 128, 3–20. doi:10.1111/acps.12095
- Slavich, G. M., Way, B. M., Eisenberger, N. I., & Taylor, S. E. (2010). Neural sensitivity to social rejection is associated with inflammatory responses to social stress. *Proceedings of the National Academy of Sciences of the United States of America*, 107, 14817–14822. doi:10.1073/pnas.1009164107

- Sturgeon, J., & Zautra, A. J. (2010). Resilience: A new paradigm for adaptation to chronic pain. *Current Pain & Headache Reports*, 2010(14), 105–112. doi:10.1007/s11916-010-0095-9
- Sturgeon, J., Zautra, A. J., & Arewasikporn, A. (2014). A multilevel structural equation modeling analysis of vulnerabilities and resilience resources influencing affective adaptation to chronic pain. *Pain*, 155, 292–298. doi:10.1016/j.pain.2013.10.007
- Way, B. M., Taylor, S. E., & Eisenberger, N. I. (2009). Variation in the μ -opioid receptor gene (OPRM1) is associated with dispositional and neural sensitivity to social rejection. *Proceedings of the National Academy of Sciences of the United States of America*, 106, 15079–15084.
- Younger, J., Aron, A., Parke, S., Chatterjee, N., & Mackey, S. (2010). Viewing pictures of a romantic partner reduces experimental pain: Involvement of neural reward systems. *PLoS One*, 5, e13309. doi:10.1371/journal.pone.0013309
- Zautra, E. K., Zautra, A. J., Gallardo, E. C., & Velasco, L. (2015). Can we learn to treat one another better? A test of a social intelligence curriculum. *PLoS One*, 1–17. doi:10.1371/journal.pone.0128638

Suggested Reading

- Evers, A., Zautra, A., & Thieme, K. (2011). Stress and resilience in rheumatic diseases: A review of 25 years of research. *Nature Reviews Rheumatology*, 7, 409–415. doi:10.1038/nrrheum.2011.80
- Infurna, F., Rivers, C., Reich, J. W., & Zautra, A. J. (2015). Childhood trauma and personal mastery: Their influence on emotional reactivity to everyday events in a community sample of middle-aged adults. *PLoS One*, 10(4), 1–21. doi:10.1371/journal.pone.0121840
- Sturgeon, J., & Zautra, A. J. (2010). Resilience: A new paradigm for adaptation to chronic pain. *Current Pain & Headache Reports*, 2010(14), 105–112. doi:10.1007/s11916-010-0095-9
- Sturgeon, J., Zautra, A. J., & Arewasikporn, A. (2014). A multilevel structural equation modeling analysis of vulnerabilities and resilience resources influencing affective adaptation to chronic pain. *Pain*, 155, 292–298. doi:10.1016/j.pain.2013.10.007
- Zautra, A., Infurna, F. J., Zautra, E., Gallardo, C., & Velasco, L. (2016). The humanization of social relations: Nourishment for resilience in mid-life. In A. D. Ong & C. E. Löckenhoff (Eds.), *Bronfenbrenner series on the ecology of human development. Emotion, aging, and health* (pp. 207–227). Washington, DC: American Psychological Association. <https://doi.org/10.1037/14857-011>
- Zautra, E. K., Zautra, A. J., Gallardo, E. C., & Velasco, L. (2015). Can we learn to treat one another better? A test of a social intelligence curriculum. *PLoS One*, 1–17. doi:10.1371/journal.pone.0128638

Physician–Patient Communication

Tricia A. Miller and M. Robin DiMatteo

Department of Psychology, University of California Riverside, Riverside, CA, USA

Introduction

While impressive technological advances abound in modern medicine, effective physician–patient communication remains one (if not *the*) central component in the process and delivery of quality healthcare. Effective communication plays a critical role in the physician–patient relationship; communication allows for the exchange of meaningful information and ideas in order to achieve patients’ therapeutic goals (Beck, Daughtridge, & Sloane, 2002; DiMatteo, 1994). To provide the best care, physicians must communicate trust and empathy (both verbally and nonverbally) in order for patients to feel comfortable disclosing medical information and sharing their experiences of living with illness (DiMatteo, 2004; DiMatteo, Hay, & Prince, 1986). In addition, research suggests that patients prefer and are more satisfied with a patient-centered approach to care (Cousin, Mast, Roter, & Hall, 2012). Patient-centered care encompasses a communication style in which physicians seek to understand patients’ perspectives, attend to patients’ psychosocial needs, and involve and collaborate with patients, especially in any decision making (Cousin et al., 2012).

Numerous studies, spanning over 40 years examining physician–patient communication, suggest positive and significant associations with various patient outcomes, including adherence to treatment recommendations, satisfaction, and physical and psychological health (Mallinger, Griggs, & Shields, 2005). Despite this empirical evidence, physician–patient communication is frequently judged to be inadequate (Beck et al., 2002). Some physicians overestimate their ability to effectively communicate (Tongue, Epps, & Forese, 2005), perhaps because of the complexity of the time-limited medical interaction itself or other variables (e.g., patients’ type of illness or disease) that may influence the physician–patient relationship.

This entry will highlight the published literature on medical communication, including both the benefits and challenges associated with effective physician–patient communication. In addition, the effects of communication training and strategies for improving physicians’ communication skills will be discussed.

Problems in Physician–Patient Communication

The communication problems in the physician–patient relationship include problems with diagnosis, a lack of patient involvement, or the inadequate provision of information to the patient (Stewart, 1995; Williams, Davis, Parker, & Weiss, 2002). Many of these problems can arise during history taking or even during the discussion of how the patient’s illness or disease should be appropriately managed (Stewart, 1995). Problems may also arise from a lack of communication skills on the part of either the physician or the patient. For patients, low levels of health literacy are particularly challenging, leading to misunderstanding of medical directives, nonadherence, and poor health outcomes (Zhang, Terry, & McHorney, 2014). Studies suggest that due to ineffective communication during the medical visit, physicians fail to recognize 50% of psychosocial and psychiatric problems that exist among their patients (Davenport, Goldberg, & Millar, 1987).

More than half of patients’ problems and concerns are neither elicited by the physician nor disclosed by the patient, and about half of the time, physicians and their patients do not agree on the issue or medical problem presented during the medical visit (Starfield et al., 1981; Stewart, McWhinney, & Buck, 1979). Some studies also demonstrate that physicians interrupt their patients within 18 s of the patient beginning to speak in an effort to describe their medical issue. As a result, patients often leave the medical interaction dissatisfied with the communication and with the information they received from their physicians (Frankel & Beckman, 1989). These studies suggest that the problems in physician–patient communication are very common; research should continue to focus attention on communication skills training at all levels of medical education.

Physician–Patient Communication and Improved Outcomes

The benefits of effective communication are many. Studies examining the physician–patient relationship have found that effective communication has the potential to help regulate patients’ emotions, facilitate the complete comprehension of medical information, and allow physicians to efficiently identify and understand their patients’ specific needs, perceptions (including health beliefs), and treatment expectations (Ha, Anat, & Longnecker, 2010). Early research on effective physician–patient communication has identified positive and significant associations with patients’ sense of control, autonomy, and ability to tolerate and adjust to pain (Roter, 1983). Some more recent studies have observed decreases in the length of hospital stays and fewer referrals and decreases in the cost of individual medical visits and malpractice litigation as a result of effective communication (Eastaugh, 2004; Little et al., 2001).

Physicians with adept communication skills can improve patient adherence. Patients typically make their health decisions based on their own beliefs, which may or may not be accurate, in the value and expected efficacy of the recommended treatment (DiMatteo, 2004). When effective communication allows physicians to be part of this evaluative process, it can be carried out in a way that is evidence based and avoids irrational or anecdotal conclusions. Additionally, interpersonal aspects of communication can affect patients’ responses to medical recommendations. Rapport and the communication of emotional support through nonverbal communication skills (e.g., facial expressions, body posture and movements, touch, eye contact, physical distance and orientation, vocal cues) can also help to facilitate and ensure trust within the therapeutic relationship (DiMatteo, 1994; Mast & Cousin, 2013). Research suggests that if the verbal information being communicated by physicians does not align with the

nonverbal behaviors that patients expect, the result can be a decrease in the level of support and empathy felt by patients (Ong, Visser, Lammes, & de Haes, 2000).

Effective communication is also related to physician satisfaction. Studies suggest that patients who are more satisfied (as a result of effective communication) tend to have physicians with greater job satisfaction and less work-related stress and who feel less burnout (Maguire & Pitceathly, 2002). Some researchers even suggest a possible reciprocal relationship between communication and physician–patient satisfaction. It is possible that patients who are more satisfied with the communication during the medical visit behave in ways that reflect this satisfaction and thus positively influence their physicians’ overall satisfaction and vice versa.

Collaborative Partnership and Improving Communication

One of the best-known ways of improving communication in the physician–patient relationship is to ensure an exchange in which both physicians and patients collaborate and engage in shared decision making (Ha et al., 2010). This type of collaborative partnership requires physicians to offer treatment choices and allows patients to share in both the responsibility and control over any decisions made. Through the process of negotiation, care options are evaluated and tailored to the context of patients’ situations and needs, rather than a standardized treatment protocol (Ha et al., 2010). Research suggests, however, that patients differ in the degree and type of participation they prefer. For example, older and/or more seriously ill emergency patients desire to be less involved in their care compared with patients who are younger and/or less seriously ill (Jahng, Martin, Golin, & DiMatteo, 2005). This research not only suggests the importance of collaborative partnership through effective communication but also highlights the need for prescreening of patients’ involvement preferences in order to maximize the effectiveness of medical care delivery.

Many techniques exist to help physicians improve their communication skills. Eastaugh (2004) detailed three effective ways to improve communication. First, physicians must focus on their patients’ concerns by exhibiting empathy through actively listening and making eye contact with their patients. Physicians must also engage their patients by using open-ended questions and avoiding using medical jargon or interrupting their patients; this sets the groundwork for a true collaborative partnership. Second, physicians must take the time to properly educate their patients (e.g., clearly defining and describing unfamiliar terms, using the “teach-back” method, and providing patients with written medical information in the form of pamphlets and brochures) to ensure complete comprehension of the prescribed treatment plan. This technique is especially important given some empirical evidence that suggests that physicians often overestimate the amount of information they provide. For example, physicians in one study believed that they provided at least 6 min of patient education, but after careful observation and analysis, it was found that the physicians provided only 40 s of education (Eastaugh, 2004). Lastly, physicians must seek to enlist their patients as collaborative partners in the decision-making process. Doing so can empower patients to question and voice their prior misconceptions and ultimately learn new disease management techniques (Eastaugh, 2004).

Communication Skills Training

Health psychologists and medical educators have sought to determine how best to teach communication skills to physicians and medical students in order to improve patient care (Haskard

et al., 2009). Many training programs have been developed and evaluated (e.g., Communication Skills Mentors Program introduced by the Bayer Institute for Healthcare Communication), and the assessment of physicians' communication skills has recently been added to medical certification programs (Haskard et al., 2009). Effective communication may not be easy, but it is a trainable skill; intervention studies show that training in communication skills can make a significant difference in patient outcomes. Communication skills such as information giving and active listening, for example, can be enhanced through training (Fallowfield, Jenkins, Farewell, & Solis-Trapala, 2003).

In a quantitative meta-analysis, researchers found that communication skills training for physicians resulted in substantial and significant improvements in patient adherence; the odds of patient adherence were 1.62 times higher when physicians received communication training compared with when physicians were not trained (Zolnierrek & DiMatteo, 2009). Additionally, training both physicians and patients to be effective communicators was related to increases in physician and patient satisfaction, willingness to recommend the physician, and the amount of health behavior counseling provided by physicians (Haskard et al., 2009). If only physicians *or* patients were trained in communication skills, however, physicians' level of stress increased, and their satisfaction decreased (Haskard et al., 2009). Currently, there is much debate over whether or not these improved behaviors may lapse over time, given that medical students' skills in talking with patients tend to decline as their medical education progresses (Ha et al., 2010). Thus, in order to deal with the challenges of shifting patient expectations, the increases in complex treatments, and the many constraints imposed by accountable/managed care, it is imperative that physicians practice optimal communication skills and seek ongoing performance feedback (Tongue et al., 2005).

Conclusion

Effective physician–patient communication fosters more positive health outcomes and a higher quality of healthcare for patients. Most complaints and malpractice suits against physicians are related to issues of communication and not necessarily clinical competency. Patients want physicians who can skillfully diagnose, detect problems early, prevent medical crises and expensive interventions, and provide better support; effective communication can help to facilitate and ensure these outcomes (Levinson, Lesser, & Epstein, 2010). Continued efforts must be made to identify the types of communication skills (or communication training interventions) that are most effective in improving communication and the degree to which communication training can reduce healthcare disparities and improve care for all patients. Policy makers and health systems leaders must recognize that in order for physicians to provide patient-centered care, they must be trained in the skills that enable them to communicate most effectively (Levinson et al., 2010). Finally, communication skills must be taught at all levels of medical education, and physicians must actively seek out, practice, and receive feedback on effective communication techniques toward the goal of better patient care.

Author Biographies

Tricia A. Miller received her PhD in psychology from the University of California at Riverside. Her research focuses on the social psychological process of health and medical care delivery including patients' health literacy. She also studies the effect of provider–patient communication

on the promotion and maintenance of treatment adherence and patients' overall health outcomes.

M. Robin DiMatteo, PhD, is Distinguished Professor of Psychology Emerita at the University of California, Riverside. She received her PhD degree from Harvard University and has served as a resident consultant in health policy at the RAND Corporation. Dr. DiMatteo has been elected Fellow of the American Association for the Advancement of Science and received grant funding from numerous agencies and foundations including the Investigator Award in Health Policy Research from the Robert Wood Johnson Foundation.

References

- Beck, R. S., Daughtridge, R., & Sloane, P. D. (2002). Physician-patient communication in the primary care office: A systematic review. *Journal of the American Board of Family Practice*, 15(1), 25–38.
- Cousin, G., Mast, M. S., Roter, D. L., & Hall, J. A. (2012). Concordance between physician communication style and patient attitudes predicts patient satisfaction. *Patient Education and Counseling*, 87(2), 193–197. doi:10.1016/j.pec.2011.08.004
- Davenport, S., Goldberg, D., & Millar, T. (1987). How psychiatric disorders are missed during medical consultations. *Lancet*, 2, 439–440.
- DiMatteo, M. R. (1994). The physician-patient relationship: Effects on the quality of healthcare. *Clinical Obstetrics and Gynecology*, 37(1), 149–161.
- DiMatteo, M. R. (2004). Variations in patients' adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care*, 43, 200–209. doi:10.1097/01.mlr.0000114908.90348.f9
- DiMatteo, M. R., Hays, R. D., & Prince, L. M. (1986). Relationship of physicians' nonverbal communication skill to patient satisfaction, appointment noncompliance, and physician workload. *Health Psychology*, 5(6), 581–594.
- Eastaugh, S. R. (2004). Reducing litigation costs through better patient communication. *Physician Executive*, 30(3), 36–39.
- Fallowfield, L., Jenkins, V., Farewell, V., & Solis-Trapala, I. (2003). Enduring impact of communication skills training: results of a 12-month follow-up. *British Journal of Cancer*, 89, 1445–1449. doi:10.1038/sj.bjc.6601309
- Frankel, R., & Beckman, H. (1989). Evaluating the patient's primary problem(s). In M. Stewart & D. Roter (Eds.), *Communicating with medical patients* (pp. 86–98). Newbury Park, CA: Sage Publications.
- Ha, J. F., Anat, D. S., & Longnecker, N. (2010). Doctor-patient communication: A review. *The Ochsner Journal*, 10, 38–43. doi:10.1043/TOJ-09-0040.1
- Haskard, K. B., Williams, S. L., DiMatteo, M. R., Rosenthal, R., White, M. K., & Goldstein, M. G. (2009). Physician and patient communication training in primary care: Effects on participation and satisfaction. *Health Psychology*, 27(5), 513–522. doi:10.1037/0278-6133.27.5.513
- Jahng, K. H., Martin, L. R., Golin, C. E., & DiMatteo, M. R. (2005). Preferences for medical collaboration: Patient-physician congruence and patient outcomes. *Patient Education and Counseling*, 57(3), 308–314. doi:10.1016/j.pec.2004.08.006
- Levinson, W., Lesser, C. S., & Epstein, R. M. (2010). Developing physician communication skills for patient-centered care. *Health Affairs*, 29(7), 1310–1318. doi:10.1377/hlthaff.2009.0450
- Little, P., Everitt, H., Williamsen, I., Moore, M., Could, C., ... Payne, S. (2001). Observational study of effect of patient centeredness and positive approach on outcome of general practice consultations. *BMJ*, 323(7318), 908–911. doi:10.1136/bmj.323.7318.908
- Maguire, P., & Pitceathly, C. (2002). Key communication skills and how to acquire them. *BMJ*, 325(7366), 697–700.
- Mallinger, J. B., Griggs, J. J., & Shields, C. G. (2005). Patient-centered care and breast cancer survivors' satisfaction with information. *Patient Education and Counseling*, 57(3), 342–349. doi:10.1016/j.pec.2004.09.009

- Mast, M. S., & Cousin, G. (2013). The role of nonverbal communication in medical interactions: Empirical results, theoretical bases, and methodological issues. In L. R. Martin & M. R. DiMatteo (Eds.), *The oxford handbook of health communication, behavior change and treatment adherence* (pp. 38–53). Oxford, UK: Oxford University Press.
- Ong, L. M., Visser, M. R., Lammes, F. B., & de Haes, J. C. (2000). Doctor-patient communication and cancer patients' quality of life and satisfaction. *Patient Education and Counseling*, 41(2), 145–156. doi:10.1016/S0738-3991(99)00108-1
- Roter, D. L. (1983). Physician/patient communication: Transmission of information and patient effects. *Medicine*, 32, 260–265.
- Starfield, B., Wray, C., Hess, K., Gross, R., Birk, P. S., & D'Lugoff, B. C. (1981). The influence of patient-practitioner agreement on outcome of care. *American Journal of Public Health*, 71, 127–131.
- Stewart, M. A. (1995). Effective physician-patient communication and health outcomes: A review. *Canadian Medical Association Journal*, 152(9), 1423–1433.
- Stewart, M. A., McWhinney, I. R., & Buck, C. W. (1979). The doctor/patient relationship and its effect upon outcome. *The Journal of the Royal College of General Practitioners*, 29, 77–82.
- Tongue, J. R., Epps, H. R., & Forese, L. L. (2005). Communication skills for patient-centered care. *The Journal of Bone and Joint Surgery*, 87(3), 652–658.
- Williams, M. V., Davis, T., Parker, R. M., & Weiss, B. D. (2002). The role of health literacy in patient-physician communication. *Family Medicine*, 34(5), 383–389.
- Zhang, N. J., Terry, A., & McHorney, C. A. (2014). Impact of health literacy on medication adherence: A systematic review and meta-analysis. *Annals of Pharmacotherapy*, 48, 741–751. doi:10.1177/1060028014526562
- Zolnierck, K. B., & DiMatteo, M. R. (2009). Physician communication and patient adherence to treatment: A meta-analysis. *Medical Care*, 47, 826–834. doi:10.1097/MLR.0b013e31819a5acc

Suggested Reading

- Martin, L. R., & DiMatteo, M. R. (2013). *The Oxford handbook of health communication, behavior change, and treatment adherence*. New York, NY: Oxford University Press.
- Martin, L. R., Haskard-Zolnierck, K. B., & DiMatteo, M. R. (2010). *Health behavior change and treatment adherence*. New York, NY: Oxford University Press.
- Miller, T. A., & DiMatteo, M. R. (2015). Communication in the aging community. In B. A. Benson (Ed.), *Psychology and geriatrics: Integrated care for an aging nation* (pp. 67–89). New York, NY: Elsevier.
- Rotar, D., & Hall, J. A. (2006). *Doctors talking with patients/patients talking with doctors: Improving communication in medical visits*. Connecticut, CT: Praeger Publishers.

Placebo and Nocebo Effects

Andrew L. Geers and Fawn C. Caplandies

Department of Psychology, University of Toledo, Toledo, OH, USA

Historical Context

Prior to the scientific era of medicine, the remedies used for medical care were largely ineffective or, worse, harmful to recipients. In the few cases in which therapeutic ingredients had value, they were inappropriately administered, given in the wrong dose, or combined with so many diluting and contrary substances that the remedies were rendered useless (Shapiro & Shapiro, 1997). Examples of early ingredients include the eating of live frogs, cast-off snake skin, spider webs, wood lice, human bones, and goat intestines and drinking the blood of an enemy. Medical interventions involved activities such as dehydration, bloodletting, magnetism, exorcism, and attaching leeches to the body. Although such treatments appear peculiar now, there was considerable belief in their utility and frequent reports of treatment efficacy. Many medical scholars have concluded that the benefits experienced in early medicine were, to a large extent, the product of what has been called placebo effects.

The word placebo entered the English language in the thirteenth century when it was translated from Latin as meaning “to please.” Between the eighteenth and nineteenth century, the word placebo developed into a term for fake treatments that are administered to placate patients. While the administration of placebos was openly viewed as the unsavory practice of disreputable practitioners, placebo treatments remained a common medical tool. Perspectives on placebos shifted in the middle of the twentieth century when concern about bias in medical research led to the scientific practice of administering inert treatments to control participants in randomized controlled trials (RCTs). The use of placebo control groups in RCTs was spurred on by an influential paper, “The Powerful Placebo,” in which Henry Beecher (1955) concluded that approximately 35% of patients in research trials benefit by simply being given a placebo. Since this time, the use of placebo control groups has become the gold standard in clinical research, and more placebos have been dispensed to participants in RCTs than any other drug or medical treatment.

Even though placebo treatments have been administered throughout recorded human history and lay theories of placebo effects abound, empirical exploration into the topic is rela-

tively recent. Initial experimentation on placebo effects began in the 1960s and has flourished during the past two decades. This increased attention can be observed in publication records, with fewer than 25 empirical publications in PubMed focused on placebo effects in the year 1992 to more than 200 in the year 2011. The interest in placebo effects likely came about for a number of reasons, including pioneering neurobiological studies linking placebo effects to detectable changes in the brain and a broader recognition of the importance of psychological mechanisms in health outcomes. With this influx of research has emerged the perspective that placebo effects, like pharmacologically active treatments, are fundamental to successful health-care (Colloca & Benedetti, 2005). Rather than discounting placebo effects, there is now a push to find strategies to enhance them. From a broader scientific perspective, placebo effects provide a rich opportunity to study the interplay of complex psychological and neurological processes. Further, work on placebo effects is transdisciplinary and integrates diverse scholarly fields including psychology, anthropology, neuroscience, ethics, chemistry, and countless branches of medicine.

Reconceptualizing Placebo Effects

Placebo effects have traditionally been defined as the result of inert treatments. As data mounted, however, such definitions proved problematic. For example, if placebo effects are defined as the consequences of inert agents, how does this explain the genuine psychobiological changes that are observed in numerous placebo studies? Recent research supports the position that the phenomenon actually centers on expectations and the meaning ascribed to the medical situation rather than to the inertness of a treatment. Contemporary definitions of placebo effects now describe them as psychological or physiological changes that are directly attributed to receiving a substance or undergoing a procedure, but that are not the direct consequence of the inherent power of that substance or procedure (Stewart-Williams & Podd, 2004). When defined in this way, placebo effects are not only a result of the administration of inert agents. Instead, they are a component of all medical treatments—the component that can be attributed to an individual's treatment expectations and interpretations (Benedetti, 2009).

In discussing placebo effects, it is important to address three common points of confusion. First, improvements observed in placebo control conditions in RCTs are not a direct indicator of placebo effects because placebo-controlled RCTs are designed to assess treatment effects by comparing a treatment group to a placebo group. To isolate placebo effects, researchers need an additional group, termed a no-placebo control group, in which participants are not given either an active or inert treatment. The difference between a placebo group and a no-placebo control group corresponds to the placebo effect. Without this third condition, it is unclear if any improvement observed in the placebo group was the sole result of placebo effects or the result of confounding factors such as spontaneous remission, history effects, and regression to the mean.

Second, placebo effects are sometimes classified as a non-specific effect. However, research has uncovered much about the causes of placebo effects, as well as the precise psychological and biochemical mechanisms underlying placebo effects. Thus, placebo effects have demonstrated specificity and do not fit the non-specific category. Third, and relatedly, as with medical treatment effects, there is a wide array of pathways by which placebo effects emerge. As such, rather than only one placebo effect, there appear to be many types of placebo effects. This final point is notable as it speaks to the long-standing question of the extent of placebo effects'

power in medical care. Attempts to estimate the magnitude of placebo effects began with Beecher's (1955) influential paper on the power of placebo effects and have been extended in more recent meta-analytic reviews. As there are various and distinct processes responsible for placebo effects, however, there is likely no single answer to this magnitude question.

Nocebo Effects

Nocebo effects refer to negatively valenced placebo effects and result from the anticipation of unpleasant treatment outcomes. As with placebo effects, contemporary research finds nocebo effects across symptom domains and in both clinical and experimental studies (Colloca, Arve Flaten, & Meissner, 2013). Because of the ethical issues involved in providing negative symptom expectations to patients, nocebo effects have been investigated less frequently than placebo effects. Although nocebo and placebo effects may involve some distinct processes, nocebo effects are often deemed a variant of the broader placebo effect phenomenon. Keeping with this perspective, much of what is discussed herein pertains to both placebo and nocebo effects.

One notable way nocebo effects diverge from placebo effects is the occurrence of unpleasant treatment side effects in patients. Researchers have found that side effect warnings provided by practitioners, direct-to-consumer medical advertising, and informed consent protocols can all produce nocebo responses. For example, Myers, Cairns, and Singer (1987) found that listing gastrointestinal side effects on an informed consent document significantly increased the frequency of gastrointestinal symptoms reported by unstable angina patients. Notably, nocebo side effects do not always manifest, and they can be moderated by variables including worry and, in pain studies, beliefs about how one reacts to pain.

Placebo Effect Research

The basic experimental procedure to test for placebo effects is to provide a treatment efficacy message to one group of participants (e.g., this pill will reduce your pain) and then compare the responses of this group with a second group who was not given this efficacy message. To isolate the impact of this manipulation on treatment outcomes, inert treatments are typically administered to all participants. Studies using this design find that the verbal information provided about a treatment is central in determining the illness experience and the symptomatic expressions of disease. Experiments have uncovered placebo effects across cultures and ages and with samples ranging from healthy volunteers to patients undergoing surgery. Although placebo effects occur in various domains, they are particularly likely to arise in studies of pain, anxiety, and depression.

Placebo effects have been found with a wide variety of dependent measures, including subjective assessments of moods, itch, sleep quality, pain, and drug cravings. Although important, findings on subjective measures are not incontrovertible evidence for placebo effects. This is because subjective measures are prone to biases including demand characteristics, experimenter expectancy effects, and response shifts. Importantly, placebo effects have been exhibited on other dependent variables including cognitive processing measures such as reaction time, word generation, Stroop interference, and implicit learning; behavioral measures such as sleep latency, alcohol consumption, talking time by socially anxious individuals, and motor performance in Parkinson's disease patients; and physiological measures including salivary cortisol, startle eyeblink reflex, bronchoconstriction in asthmatics, and beta-band frequency

during sleep. The reach of placebo effects, however, is not unlimited. Placebo effects do not alter disease parameters such as the size of cancerous tumors.

In addition to treatment efficacy information altering the effects of *inactive* treatments, it also changes the efficacy of *active* treatments. For example, many researchers have studied placebo effects by using the balanced placebo design in which treatment efficacy information (e.g., stating that a pill contains caffeine or not) and treatment agency (e.g., administering caffeine pills or not) are independently manipulated. Although results from balanced placebo designs vary, many find that stating that a treatment is efficacious amplifies the effectiveness of active treatments. In a conceptually similar vein, studies employing the open–hidden paradigm (Colloca & Benedetti, 2005) compare the impact of pharmacologically active treatments when patients are aware and unaware of the timing of a treatment administration (e.g., automatic intravenous drip hidden from view). This work has revealed that when the dose of an active treatment is covertly given, its specific efficacy can be substantially reduced—that is, without the awareness of their administration, active drugs are less effective.

Despite the remarkable progress in research on placebo effects, many uncertainties remain. Perhaps most notably, the magnitude and direction of placebo effects can be unpredictable. Some of these inconsistencies may be attributable to the diversity in treatment domains, samples, verbal efficacy manipulations, and contexts used in placebo studies.

Psychological Mechanisms

Theoretical dialogue regarding psychological processes underlying placebo effects have largely focused on two factors: expectations and classical conditioning. Classical conditioning explanations posit that placebo effects can be conditioned responses, with active medications as the unconditioned stimuli and the methods or techniques used to administer or accompany treatments as the conditioned stimuli. A substantial body of human and animal laboratory work demonstrates that conditioning can produce placebo effects. Also, increasing the number of conditioning trials results in stronger placebo effects, prior experience with an effective treatment strengthens placebo effects, and placebo effects can emerge from social observational learning (Colloca et al., 2013). Even though placebo research has given considerable attention to conditioning, expectations are generally viewed as the primary psychological mechanism underlying placebo effects. In fact, theoretical and experimental work now points to the view that conditioning is an associative process by which expectancies develop.

Expectancy explanations hold that the placebo effects are driven by the belief that receiving a treatment results in a particular response or outcome. One influential approach is response expectancy theory (Kirsch, 1999), which posits that placebo effects are caused by anticipated automatic reactions. That is, expectations are said to serve a preparatory function and directly stimulate expectancy-based responses. An extensive array of work supports the position that expectancies cause placebo effects. For example, the same inert treatment can lead to arousal or sedation depending upon the expectancy message provided. Further, modifying the characteristics of an expectancy message can change the magnitude of placebo effects. For example, double-blind expectations (e.g., you may or may not be given an active drug) often produce weaker placebo effects than deceptive expectations (e.g., you have been given an active drug). Investigations also find that self-reported expectations can mediate the association between treatment efficacy messages and placebo effects (Stewart-Williams & Podd, 2004).

What are the pathways by which expectations bring about placebo effects? It appears there are at least four overlapping pathways (see Benedetti, 2009; Colloca et al., 2013; Geers &

Miller, 2014). First, expectations can produce beneficial outcomes by reducing anxiety and decreasing the activation of threat-related centers in the brain. Thus, sometimes expectations lead to placebo effects because expecting an effective treatment lessens anxiety. In the case of nocebo effects, negative expectations increase rather than decrease threat and anxiety. Second, other work suggests that anticipating improvements from a treatment increases positive feelings through opioid and dopaminergic reward mechanisms in the brain. That is, expectations engender placebo responding by activating endogenous reward processes. Third, expectations can act as an interpretive guide, directing attention, detection, weighting, attributions, behavior, and recall of somatic experiences. Finally, conditioning and nonconscious priming studies indicate that placebo effects can arise from cues activating simple associative responses.

Neurobiological Mechanisms

One of the fastest growing areas of placebo research concerns the neurobiological pathways involved in the effect. This line of work clearly overlaps with the aforementioned work on psychological mechanisms. Research in this area began with a landmark study by Levine, Gordon, and Fields (1978) in which the opioid antagonist naloxone was found to block placebo responses in a pain paradigm. These results were the first to implicate the endogenous opioid system in placebo pain relief (termed placebo analgesia). Since this time, opioid, cannabinoid, and cholecystokinin endogenous mechanisms have been connected to placebo analgesia.

Brain imaging studies have been critical in clarifying the neurobiological mechanisms underlying placebo effects (for reviews, see Benedetti, 2009; Colloca et al., 2013). For example, experimental work indicates that placebo analgesics work by decreasing activity in the same pain-processing regions of the brain (e.g., the anterior cingulate cortex and the prefrontal cortex) as pharmacological pain treatments. Thus, placebos appear to produce change through some of the same pathways as the drugs they are often tested against. Several studies have revealed that in addition to changing activity in brain networks, placebo treatments can involve the descending inhibition of spinal nociceptive processes. Other work reveals that, in contrast to placebo expectations, nocebo expectations are associated with greater activity in the anterior cingulate cortex and the prefrontal cortex and stronger pain-related activity in the spinal cord. Outside of the arena of pain, researchers have found that in patients suffering from Parkinson's disease, placebo effects in motor performance are associated with dopamine release in the striatum.

Moderators of Placebo Effects

One of the most vexing puzzles in the placebo literature has been why some people display a placebo response in a given context and others do not. In early placebo research this issue was often framed in terms of locating the "type" of individuals who responds to a treatment expectation—labeled placebo responders. The possibility that there is a placebo-prone personality has been frequently studied. Despite these efforts, few if any individual difference variables reliably predict placebo responding across contexts (Geers, Kosbab, Helfer, Weiland, & Wellman, 2007). For example, relationships between placebo effects and personality variables such as introversion, optimism, empathy, social desirability, and self-esteem occur in some contexts, but not others. Relatedly, individuals responding to one placebo manipulation do

not reliably respond to a different placebo manipulation. That said, much of this research has focused on personality differences, whereas a new wave of research is assessing whether genetic and biological factors predict placebo responding (Hall, Loscalzo, & Kaptchuk, 2015).

Other lines of research have focused on situational factors that alter the success of placebo treatments. Many contextual factors alter the magnitude of placebo effects. These factors include aspects of the healthcare practitioner (e.g., white lab coat, a practitioner's belief in treatment efficacy), properties of the treatment (e.g., number, size, color, brand, cost, and taste of pills), and properties of the administration procedure (e.g., injection, oral). Another factor that appears to moderate placebo effects is whether an active or passive placebo is employed. Active placebos produce side effects that are detectable by individuals, whereas passive placebos do not. Active placebos provide cues to the recipient that a treatment is effective and may lead to a stronger belief in treatment effectiveness than passive placebos.

Recently, research has begun to consider the interactive effects of individual differences and situational factors in producing placebo effects. This work suggests that placebo effects are determined by the match between the context and the person's current motives, prior experiences, and chronic tendencies. For example, Colloca and Benedetti (2009) induced placebo effects in three groups, one through a verbal message, one via conditioning, and one via observational learning. In this study, empathy motivation predicted placebo responses, but only when the placebo effect was induced through observational learning.

Placebo Effects in Modern Times

Placebo effects may be commonplace in modern treatments for physical and mental health. For example, in controlled clinical trials, some standard treatments, including cough suppressants and dietary supplements, often do not differ from placebo controls—suggesting that they possess a strong placebo component (Moerman, 2002). There is also considerable data indicating that the benefits of many contemporary and alternative medicines, which amount to approximately 34 billion dollars annually, benefit substantially from placebo effects (Moerman, 2002). Evidence consistent with this notion comes from a series of clinical trials with chronic pain patients that compared real acupuncture with sham acupuncture. Although no differences were found between the two groups, patient beliefs about the efficacy of acupuncture were a reliable predictor of pain relief (Bausell, Lao, Bergman, Lee, & Berman, 2005). In terms of mental health, Kirsch found that approximately 70% of antidepressant effects can be attributed to placebo effects (Kirsch, 1999). Psychotherapy is another arena in which placebo effects are likely to be strong. Difficulties in developing precise placebo control groups for psychotherapy, however, leave the exact contribution of placebo effects in this domain unknown. Finally, recent surveys across a variety of countries indicate that many physicians continue to use placebo treatments in routine practice. For example, a survey of 679 practicing internists and rheumatologists in the United States found that 58% reported prescribing (mainly active) placebo treatments on a regular basis (Tilburt, Emanuel, Kaptchuk, Curlin, & Miller, 2008).

Clinical Translation

A difficulty in the placebo effect literature has been determining how to harness the placebo phenomenon to improve patient care. This problem arises because research generally employs

deceptive expectation manipulations to generate placebo effects, whereas the overt use of deception is not permitted by medical codes of conduct. The fact that placebo effects can be conditioned provides some opportunities for translation. For example, researchers have paired placebos and active drugs given to participants and then removed portions of the active drug over time (Doering & Rief, 2012). These studies suggest that placebos can be used to maintain drug responses after the initial associative pairing. Another intriguing development in translation is the creation of the “open label” paradigm in which participants are explicitly informed that they have been administered a placebo and are additionally told that placebos yield beneficial effects. In open label experiments, participants have improved from placebo treatments even when they were aware that their treatment was inert (Kam-Hansen et al., 2014).

Another approach to enhancing placebo effects in medical care arises from the notion that all treatments have a placebo (e.g., expectation) component. Thus, rather than directly administering inert treatments, benefits can arise from altering contextual factors that strengthen the placeboogenic portion of a standard treatment. For example, research indicates that when individuals have an opportunity to choose between placebo treatments, they may exhibit larger placebo effects compared with participants assigned a placebo treatment (Geers et al., 2013). This work suggests that in some cases physicians could increase placebo responding by encouraging greater patient involvement. The patient–provider interaction provides another opportunity for enhancing placebo effects in medical settings (Benedetti, 2009). Evidence for the importance of the therapeutic encounter in placebo responding comes from a study in which patients with irritable bowel syndrome were randomly assigned to a wait list group, a placebo acupuncture group, or a group that received placebo acupuncture with a supportive practitioner (Kaptchuk et al., 2008). The supportive practitioners were instructed to listen to participants and to be warm, confident, and thoughtful. The results revealed that participants with the supportive practitioner reported greater improvement than the other two groups 3 weeks later. These data suggest that practitioners can play a key role in strengthening the placebo component of treatments. Although aspects of the patient–provider relationship seem to alter placebo effects, more research is needed to clarify the specific factors that best enhance placebo responding in such interactions.

Conclusion

Over the last several decades, the concept of placebo effects has evolved into a topic of scientific inquiry in its own right. Research reveals that placebo effects influence meaningful physical and mental health outcomes and this phenomenon is not static but has many moderators. Research is now uncovering the psychobiological processes responsible for placebo effects. This work bridges disciplines and points to avenues by which to improve patient care, as well as our understanding of the interplay of psychological and neurological processes.

Author Biographies

Andrew L. Geers is a professor of psychology at the University of Toledo. He is interested in the advancement and application of personality and social psychology theory within health and medical contexts. His primary research interests are in the areas of placebo effects, optimism, health behavior, and assimilation and contrast models. He received his BA from the University of Cincinnati and his PhD in experimental psychology from Ohio University.

Fawn C. Caplandies is a graduate student in social psychology at the University of Toledo. She received her BA from Hartwick College in 2013. Her primary research interests are in placebo effects, expectations, and the psychophysiology of stress and coping.

References

- Bausell, R. B., Lao, L., Bergman, S., Lee, W. L., & Berman, B. M. (2005). Is acupuncture analgesia an expectancy effect? Preliminary evidence based on participants' perceived assignments in two placebo-controlled trials. *Evaluation & the Health Professions*, 28, 9–26. doi:10.1177/0163278704273081
- Beecher, H. K. (1955). The powerful placebo. *Journal of the American Medical Association*, 159, 1602–1606. doi:10.1001/jama.1955.02960340022006
- Benedetti, F. (2009). *Placebo effects: Understanding the mechanisms in health and disease*. New York, NY: Oxford University Press.
- Colloca, L., Arve Flaten, M., & Meissner, K. (Eds.). (2013). *Placebo and pain: From bench to bedside*. Boston, MA: Academic Press.
- Colloca, L., & Benedetti, F. (2005). Placebos and painkillers: Is mind as real as matter? *Nature Reviews Neuroscience*, 6, 545–552. doi:10.1038/nrn1705
- Colloca, L., & Benedetti, F. (2009). Placebo analgesia induced by social observational learning. *Pain*, 144, 28–34. doi:10.1016/j.pain.2009.01.033
- Doering, B. K., & Rief, W. (2012). Utilizing placebo mechanisms for dose reduction in pharmacotherapy. *Trends in Pharmacological Sciences*, 33, 165–172. doi:10.1016/j.tips.2011.12.001
- Geers, A. L., Kosbab, K., Helfer, S. G., Weiland, P. E., & Wellman, J. A. (2007). Further evidence for individual differences in placebo responding: An interactionist perspective. *Journal of Psychosomatic Research*, 62, 563–570. doi:10.1016/j.jpsychores.2006.12.005
- Geers, A. L., & Miller, F. G. (2014). Understanding and translating the knowledge about placebo effects: The contribution of psychology. *Current Opinion in Psychiatry*, 27, 326–331. doi:10.1097/YCO.0000000000000082
- Geers, A. L., Rose, J. P., Fowler, S. L., Rasinski, H. M., Brown, J. A., & Helfer, S. G. (2013). Why does choice enhance treatment effectiveness? Using placebo treatments to demonstrate the role of personal control. *Journal of Personality and Social Psychology*, 105, 549–566. doi:10.1037/a0034005
- Hall, K. T., Loscalzo, J., & Kaptchuk, T. J. (2015). Genetics and the placebo effect: The placeboome. *Trends in Molecular Medicine*, 21, 285–294. doi:10.1016/j.molmed.2015.02.009
- Kam-Hansen, S., Jakubowski, M., Kelley, J. M., Hoaglin, D. C., Kaptchuk, T. J., & Burstein, R. (2014). Altered placebo and drug labeling changes the outcome of episodic migraine attacks. *Science Translational Medicine*, 6, 218–225. doi:10.1126/scitranslmed.3006175
- Kaptchuk, T. J., Kelley, J. M., Conboy, L. A., Davis, R. B., Kerr, C. E., Jacobson, E. E., ... Lembo, A. J. (2008). Components of placebo effect: Randomised controlled trial in patients with irritable bowel syndrome. *British Medical Journal*, 336, 999–1003. doi:10.1136/bmj.39524.439618.25
- Kirsch, I. (1999). *How expectancies shape experience*. Washington, DC: American Psychological Association.
- Levine, J. D., Gordon, N. C., & Fields, H. L. (1978). The mechanisms of placebo analgesia. *Lancet*, 2, 654–657. doi:10.1016/S0140-6736(78)92762-9
- Moerman, D. (2002). *Meaning, medicine, and the 'placebo effect'*. Cambridge, UK: Cambridge University press. doi:10.1017/CBO9780511810855
- Myers, M. G., Cairns, J. A., & Singer, J. (1987). The consent form as a possible cause of side effects. *Clinical Pharmacology & Therapeutics*, 42, 250–253. doi:10.1038/clpt.1987.142
- Shapiro, A. K., & Shapiro, E. (1997). *The powerful placebo effect*. Baltimore, MA: John Hopkins University Press.
- Stewart-Williams, S., & Podd, J. (2004). The placebo effect: Dissolving the expectancy versus conditioning debate. *Psychological Bulletin*, 130, 324–340. doi:10.1037/0033-2909.130.2.324

- Tilburt, J. C., Emanuel, E. J., Kaptchuk, T. J., Curlin, F. A., & Miller, F. G. (2008). Prescribing “placebo treatments”: Results of national survey of U.S. internists and rheumatologists. *British Medical Journal*, 337, 7678–7683. doi:10.1136/bmj.a1938

Suggested Reading

- Benedetti, F. (2014). *Placebo effects: Understanding the mechanisms in health and disease* (2nd ed.). New York, NY: Oxford University Press.
- Colloca, L., Arve Flaten, M., & Meissner, K. (Eds.) (2013). *Placebo and pain: From bench to bedside*. Oxford, UK: Elsevier.
- Finniss, D. G., Kaptchuk, T. J., Miller, F., & Benedetti, F. (2010). Biological, clinical, and ethical advances of placebo effects. *Lancet*, 375(9715), 686–695. doi:10.1016/S0140-6736(09)61706-2
- Geers, A. L., & Miller, F. G. (2014). Understanding and translating the knowledge about placebo effects: The contribution of psychology. *Current Opinion in Psychiatry*, 27, 326–331. doi:10.1097/YCO.0000000000000082
- Stewart-Williams, S., & Podd, J. (2004). The placebo effect: Dissolving the expectancy versus conditioning debate. *Psychological Bulletin*, 130, 324–340. doi:10.1037/0033-2909.130.2.324

Positive Affect

Brooke N. Jenkins, Amanda M. Acevedo,
and Sarah D. Pressman

Department of Psychological Science, University of California Irvine, Irvine, CA, USA

Positive affect (PA), such as feelings of happiness or joy, has been tied to many health and physiological benefits. For example, a meta-analysis of the benefits of PA on mortality found that individuals high in PA were 18% less likely to die within the follow-up periods of over 20 years (Chida & Steptoe, 2008). This effect size is similar in magnitude to the effects of smoking cessation programs for smokers on mortality indicating that it may be critical to uncover the dynamics of the PA–health relation. This entry will start by outlining the methodological issues when studying PA, briefly review the literature, and then discuss potential mechanisms connecting PA to health. Finally, we end with a discussion of positive psychology interventions (PPIs) in a health context and future directions for this field.

Methodological Themes and Issues in the Study of PA and Health

Definitions of PA vary with an array of adjective and question types utilized to assess this characteristic. Thus, it is critical for researchers to think carefully about which types of PA are assessed by their measurement tools. The Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) and the Profile of Mood States (POMS) questionnaire (Curran, Andrykowski, & Studts, 1995) are popular self-report methods used to assess PA in health studies. These measures assess either state or trait affect by asking participants to report on how they have felt in a recent period of time or how they generally feel, respectively. In the case of large medical studies, researchers often rely on alternative methodologies to assess PA. Because of time constraints, for example, some researchers use single-item measures of happiness or similar emotions. While these measures are not preferred, since they do not capture the full spectrum of the PA experience, they have been tied to future health outcomes. An alternative self-report measurement approach, especially in archival data approaches, is to use the positive items from depression scales as a source of PA (e.g., Center for Epidemiological

Studies Depression Scale [CESD]). While this approach has proved useful in predicting future health such as risk of AIDS mortality and stroke (e.g., Moskowitz, 2003), it is difficult to interpret what forms of positivity are most beneficial in these studies. The CESD PA items assess other positive constructs beyond PA (e.g., optimism, self-esteem), and since these have also been tied to wellness, we are left with a question as to what the active ingredients for good health are in these studies.

Related to this issue of “active ingredients” is the question of which types of items should be assessed and/or included in measures of PA and whether these components should be considered independently. The PANAS and POMS discussed above measure high arousal feelings of positivity to a large extent (e.g., vigor, excitement, interest). While these specific subcomponents have been tied to better health (e.g., longer lifespan, decreased cold infection; Cohen, Alper, Doyle, Treanor, & Turner, 2006; Pressman & Cohen, 2012), in excess, they can be harmful. In contrast, low arousal feelings of calm and peacefulness may be helpful for populations experiencing pain or high stress. Recent research has also examined specific positive emotions like love, desire, amusement, pride, and awe and shown that these do have distinct physiological profiles as well as distinctive behavioral correlates and signals. Although there is little research on the types of PA most helpful to different health outcomes at this point, there are many reasons to think that PA type matters and that, in some cases, examining broad measures of PA might obscure interesting subtype differences in predictions (Pressman, Jenkins, Kraft, Rasmussen, & Scheier, 2017).

Another consideration when picking a PA measurement tool in the context of health is *duration*. Instructions for affect assessment are often not considered carefully when scales are chosen, but how long PA lasts may have important implications for the health or physiological outcome at hand. For example, strong changes in state PA (i.e., transient emotions and moods) may influence physiology, for example, when assessed in a laboratory setting with acute challenges or manipulations of affect. Trait (i.e., stable, dispositional) PA, on the other hand, is likely the stronger predictor of future longevity and disease because it indicates how an individual *typically* feels. With that said, there is a high correlation between state and trait PA, which is likely responsible for studies showing state PA ties to survival. Of possible future interest, although not assessed in the literature broadly at this point, is the possibility that variability in state PA (i.e., fluctuation as opposed to average levels) may also be important.

Also of interest in measurement is the issue of whether there are limitations to the reliance on self-reports in this field, given that there are known response biases and social desirability concerns, among other possible problems. To address this, some researchers use alternative methods like obtaining writing and audio samples and coding positive words and laughter as markers of emotional style (Pressman & Cohen, 2012). Others have relied on observable expressions of positivity by coding facial expressions in photos or in person and have found them to be tied to mortality and disease outcomes. While there are limitations to these methods as well (e.g., language and culture norm differences), they do provide convergent validity and some indication of the generalizability of PA–health findings beyond self-report.

One final important issue in the measurement of PA is the role of negative affect (NA). Negative traits have been extensively related to health and, depending on the measurement, can be strongly correlated with PA (Watson et al., 1988). Thus, it is critical for researchers to answer the question of whether these effects are distinct phenomena. While rare in the NA–health field, PA researchers broach this problem by controlling for NA in their PA studies and/or contrasting the relative effects. This can be difficult, as many trait measures of affect have highly correlated PA/NA scales, creating multicollinearity issues in analyses. Thus, many

researchers choose the PANAS to resolve this, which was specifically designed to have low overlap between valence dimensions, or rely on alternative negative measures (e.g., depressive symptoms), which overlap less with PA.

There are also methodological concerns specific to the health component of the PA–health question. One important concern is the conceptual and measurement overlap that exists when assessing health by self-report. Many health studies rely on participants to self-assess their health using questionnaires like the 36-item Short-Form Health Survey (SF-36). While known to be a good and valid predictor of objective health, the SF-36 actually includes items about happiness within it creating some clear overlap and interpretation problems. Researchers should therefore be cautious when picking self-report health scales. This is especially important when comparing self-report health scales with PA scales that assess energy (e.g., vigor, active) since these types of PA scales may tap elements of physical fitness and health. This can be partly addressed by controlling for these types of outcomes in analyses. Finally, it is important to remember that physiological and behavioral outcomes are not equivalent to health. While assessments of cortisol, blood pressure, and immune function as well as health behaviors (e.g., sleep, exercise) are key hypothesized mediators in the broader PA–health relation, when interpreting these outcomes, it is important to not reach too far since the level of change in parameters may not reach clinical significance.

PA–Health Review

The following sections consider the impacts of PA on the health outcomes most studied in conjunction with PA. When examining the influence of PA on future health, the gold standard is to survey participants on both PA and health (e.g., medical records, physicals, medications, fitness) at baseline and then follow up with them after a period of time has passed. While this prospective longitudinal design does not allow us to infer causality, it does give us more confidence that PA *may* be leading to future health if we control for health and its connection with baseline PA as best we can.

Longevity/Mortality

It is well established that PA is related to lower rates of mortality (e.g., Chida & Steptoe, 2008; Diener & Chan, 2011; Pressman & Cohen, 2005). A typical study design can be found by Kawamoto and Doi (2002) who evaluated older adults on baseline self-rated happiness and then assessed mortality rates 3 years later. Similar to other studies, individuals reporting higher happiness and cheerfulness at baseline were more likely to still be alive at follow-up. Beyond the typical self-report strategy, studies coding positive words in writing samples from autobiographies as an indication of PA have also found years of extended life benefit especially among those using more activated words. Similarly, coding of positive facial expressions in baseball player cards found a 7-year lifespan benefit for those with the most intense smiles even after controlling for relevant demographic variables. Although these non-self-report studies could not assess baseline health, attempts to do so by coding health words did not cancel out this effect, and we can assume that baseball players in their prime were in similarly good health given their professional athlete status. Thus, across numerous studies and PA measurement approaches, we see PA-related lifespan benefits.

While the consistency is impressive, there are some studies that have not shown these benefits. However, these studies are typically different from the above in some way. For example,

studies that do not find such links assess child cheerfulness, middle-aged women, and older adults in assisted living facilities. Thus, PA does not hold equal benefit at all life stages, and perhaps other factors are more important predictors of longevity in these groups (e.g., current health status and/or health behaviors). However, given the paucity of research on these groups, more research is needed before making any firm conclusions.

Survival

PA–health researchers have also investigated the effects of PA on survival in populations with life-threatening disease. For example, one study assessed women with HIV for positive expectations, finding meaning, and PA using words adapted from the POMS and found PA was related to longer survival. However, this study did not separate the effects of PA from expectations and meaning, which made it difficult to know what drove these benefits. Related, Moskowitz (2003) found that for every standard deviation increase in PA (based on the CESD subscale), HIV+ men were 28% less likely to die during follow-up. These results were independent of CESD, NA, and other health-related factors relevant to this population. These results are consistent with other positive findings in samples undergoing heart surgery or with congestive heart failure.

Despite these benefits, the effects of PA in unhealthy populations are mixed. For example, the PA subscale of the NEO-PI (Neuroticism, Extraversion, Openness Personality Inventory) predicted 11-year survival in people with coronary artery disease after accounting for relevant demographic and health factors. However, the effect of PA became nonsignificant when the NEO-PI depressive subscale was added into the model. Similarly, PA assessed using the Mood Adjectives Checklist did not predict 1-year survival in recently diagnosed cancer patients with varying levels of cancer type, stage, and severity while depression (assessed with the CESD) did. It has been previously hypothesized that these mixed results are due to the limits of PA (Pressman & Cohen, 2005). That is, early-stage diseases that can be benefited by healthy behavior, support, and lower stress may be helped by high PA, whereas diseases that are far along (e.g., failing kidneys, late-stage cancer) may not benefit since at that point the pathways connecting PA to better health may be less potent. There is also the possibility that too much PA may be indicative of a failure to realistically cope with the negative aspects of a life-threatening disease, in which case affect balance may be a useful pursuit to examine in future studies.

Morbidity

Prospective studies assessing PA at baseline and then following up on rates of morbidity find that individuals higher in PA less frequently develop illness. For example, high PA is associated with a decreased likelihood of future coronary heart disease, injury, and stroke. While most studies are observational, experimental studies that exposed participants to cold viruses found that individuals with greater PA (averaged over 2 weeks) were less likely to develop a cold or flu (Cohen et al., 2006). These results were independent of NA. This study's objective illness measures (e.g., mucus weight, viral cultures) and experimental design are instrumental in clarifying that even when high PA individuals get exposed to a virus, they are less likely to get sick (as opposed to the alternative explanation that high PA individuals are less likely to be exposed to a virus). Together with the naturalistic findings, these results support that PA may confer resiliency to illness and disease. The strength of this literature is the impressive consistency (i.e., studies support an array of morbidity benefits). The weakness is that few morbidity

outcomes have been replicated, calling for future studies to generate more data and increase our confidence regarding these positive associations.

Symptom Report, Severity, and Pain

PA is consistently associated with an array of self-reported health outcomes such as decreased symptom report, reduced reports of symptom severity, and less pain (e.g., DuBois et al., 2015; Finan & Garland, 2015; Sturgeon & Zautra, 2013). While most studies are cross-sectional, studies manipulating PA through, for example, loving-kindness meditation training (Fredrickson, Cohn, Coffey, Pek, & Finkel, 2008) or smiling (Pressman, Acevedo, Aucott, & Kraft, under review) in healthy samples have shown reductions in illness symptoms and perceived pain, respectively. Similar results have been found in studies of patient populations, such as those suffering from chronic pain (e.g., Finan & Garland, 2015; Sturgeon & Zautra, 2013) and cardiovascular disease (e.g., DuBois et al., 2015). These findings are more consistent across populations than the findings reviewed in previous sections. However, there may be reason to think PA can bias self-reports in a way that might not match underlying objective symptoms. For example, individuals high in trait PA who got sick following experimental exposure to an influenza virus reported fewer subjective symptoms than was expected given the objective markers of disease (e.g., mucus weight) suggesting a symptom reporting bias (Cohen et al., 2006). This study highlights the importance of measuring and reporting both subjective and objective markers of symptoms and/or pain when possible to clarify whether the relationship between PA and health is due to biased subjective reports or actual, biological resilience.

How Does PA “Get Under the Skin” and Benefit Health and Well-Being?

Main Effect Model of PA

Most researchers studying PA and health presume that PA benefits health through behavioral (e.g., better sleep, diet, exercise, medication adherence; Boehm & Kubzansky, 2012; Pressman & Cohen, 2005), physiological (e.g., cardiovascular, immune), and social (e.g., more social support and integration) pathways. These pathways are multidirectional (e.g., more social support may lead to better health behaviors, better health behaviors lead to healthier physiology). Newer studies in this area of research explore important pathways like inflammation, diet, and vagal tone (e.g., Kok et al., 2013) revealing consistent evidence that PA has direct connections to many of these pathways that provide a compelling explanation for *how* PA could influence health.

Stress-Buffering Model of PA

In contrast to the main effect model, PA may improve health by improving an individual's ability to handle stress, either by altering stress perceptions, reactions, or recovery (Pressman & Cohen, 2005). Under this model, PA is beneficial to health because it counters the negative health sequelae and physiological arousal tied to stressful experiences. For example, high PA individuals may have resources to help them cope better with stressors and recover faster. Although evidence supports that PA is beneficial during stress, it is unlikely that it is *only*

beneficial during these times and, as explained with the main effect model, that PA even during non-stressful times has health-enhancing benefits.

Broaden-and-Build Theory

The broaden-and-build theory suggests that the evolutionary advantage of PA is to allow us to expand our resources by facilitating approach behaviors (Fredrickson, 2001) in contrast to NA, which narrows focus to dispel danger. For example, emotions such as joy, interest, and excitement may lead to actions like exploration, creativity, or play. These behaviors can build resources such as skills, knowledge, or social ties that will endure after the positive emotion has passed (Fredrickson, 2001) and benefit individuals during future situations. In the context of health, the broadened behaviors and cognitions experienced as a result of PA may provide individuals with physical resources, knowledge, or social connections that enable them to perform better health behaviors or reduce stress.

Taken together, these models show that happiness is not a black box that pushes out health, but that there are many reasons why PA should be good for an individual. Importantly, many of these paths do not make sense when considering the argument that PA is simply the lack of NA. For example, *not being negative* (but also not being positive) would not confer protection or recovery against stress. Similarly, the absence of NA would not encourage individuals to explore and build resources, while the presence of PA would. Certainly, further work is necessary with well-designed experimental and longitudinal studies to help us unpack these pathways; however, there are already several detailed conceptual models to build off of that will allow us to better understand the PA–health link.

Does Increasing PA Improve Health?

Now that we understand the multitude of interconnections between PA and health, we can ask the question: Can increasing PA improve health? This may be difficult given that many researchers consider PA (especially trait PA) to be relatively stable due to genetic factors and/or unchangeable circumstances (Lyubomirsky, Sheldon, & Schkade, 2005). Nevertheless, evidence also indicates that a reasonably large portion of PA may be unfixed (Lyubomirsky et al., 2005), and a meta-analysis of over 50 PPIs revealed that, overall, PPIs have a small-to-medium effect on increasing PA (Sin & Lyubomirsky, 2009). So, if we can successfully increase PA, will this initiate improvement in health? While there are only a few studies on this topic to date, they are promising. For example, researchers found a simple positive writing intervention led to fewer health center visits and better self-reported health and health behavior, even when the writing intervention was only 2 min each day for 2 days. A randomized controlled trial provided an educational intervention or an education plus PA induction intervention to a hypertensive African American sample. Individuals in the latter group had significantly higher medication adherence. While not typically assessing PA as an outcome, studies on mindfulness (which likely increases low arousal PA) have shown immunological, cardiovascular, and neurological changes (Kok et al., 2013). Similarly, increased PA after a loving-kindness meditation intervention led to increases in perceived social connectedness, which, in turn, increased vagal tone (Kok et al., 2013). While studies have not yet tested major differences in mortality, morbidity, or survival from PPIs, research has shown that these methods are acceptable for diseased populations. Although more research is needed to establish the benefits of PPIs, current results are promising. One important unknown, however, is what the active ingredients are

that are responsible for health improvements. PA is a multifaceted construct, and it is likely that different kinds of PA are more or less helpful for future health and for those facing disease. Thus, researchers should take a nuanced and cautious approach as they enter this new territory.

Conclusion

The PA–health relationship is multifaceted and requires refined knowledge of how to measure and conceptualize both PA and health. Understanding these complex issues allows us to investigate how PA influences health outcomes like longevity/mortality, morbidity, and survival, as well as symptom report, severity, and pain. Several pathways, theories, and models can explain this PA–health link, and researchers can use this knowledge to fine-tune the effectiveness of interventions to target specific health outcomes. Future research in the area of PA–health will make great strides in our understanding of the role affect can play in health in particular by examining how different arousal components of PA influence health differentially, expanding research on the types of illnesses PA may impact, refining our understanding of the pathways of the PA–health link, and testing the bounds of the health benefits of PPIs.

Author Biographies

Brooke N. Jenkins is an assistant professor at Chapman University. Her work examines the effects of emotions on health. She is interested in methods for assessing emotions over time and how emotion variability influences physiological and behavioral health outcomes. She investigates new methods for assessing this variability, and in her research she works with a number of different populations including healthy children undergoing surgery and children with cancer, as well as healthy adults.

Amanda M. Acevedo is a graduate student at the University of California, Irvine. Her research program lies at the intersection of culture, emotion, and health. She is interested in how positive emotions and cultural factors might confer resilience during stress in general and in the context of pain in particular. She uses cardiovascular stress reactivity/recovery methods to examine how emotions and cultural factors might buffer stress in the lab.

Sarah D. Pressman is an associate professor of psychology and social behavior at the University of California, Irvine. Dr. Pressman's work examines how positive emotions are beneficial for physical health and the pathways by which positive psychosocial factors “get under the skin” to influence biology. She is especially interested in the role of positive psychosocial factors in protecting individuals from the harmful effects of stress and whether specific emotions confer resilience in the face of adversity.

References

- Boehm, J. K., & Kubzansky, L. D. (2012). The heart's content: The association between positive psychological well-being and cardiovascular health. *Psychological Bulletin*, 138, 655–691. doi:10.1037/a0027448
- Chida, Y., & Steptoe, A. (2008). Positive psychological well-being and mortality: A quantitative review of prospective observational studies. *Psychosomatic Medicine*, 70, 741–756. doi:10.1097/PSY.0b013e31818105ba

- Cohen, S., Alper, C. M., Doyle, W. J., Treanor, J. J., & Turner, R. B. (2006). Positive emotional style predicts resistance to illness after experimental exposure to rhinovirus or influenza A virus. *Psychosomatic Medicine*, 68, 809–815. doi:10.1097/01.psy.0000245867.92364.3c
- Curran, S. L., Andrykowski, M. A., & Studts, J. L. (1995). Short Form of the Profile of Mood States (POMS-SF): Psychometric information. *Psychological Assessment*, 7, 80–83. doi:10.1037/1040-3590.7.1.80
- Diener, E., & Chan, M. Y. (2011). Happy people live longer: Subjective well-being contributes to health and longevity. *Applied Psychology: Health and Well-Being*, 3, 1–43. doi:10.1111/j.1758-0854.2010.01045.x
- DuBois, C. M., Lopez, O. V., Beale, E. E., Healy, B. C., Boehm, J. K., & Huffman, J. C. (2015). Relationships between positive psychological constructs and health outcomes in patients with cardiovascular disease: A systematic review. *International Journal of Cardiology*, 195, 265–280. doi:10.1016/j.ijcard.2015.05.121
- Finan, P. H., & Garland, E. L. (2015). The role of positive affect in pain and its treatment. *The Clinical Journal of Pain*, 31, 177–187. doi:10.1097/AJP.0000000000000092
- Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist*, 56, 218–226. doi:10.1037/0003-066X.56.3.218
- Fredrickson, B. L., Cohn, M. A., Coffey, K. A., Pek, J., & Finkel, S. M. (2008). Open hearts build lives: Positive emotions, induced through loving-kindness meditation, build consequential personal resources. *Journal of Personality and Social Psychology*, 95, 1045–1062. doi:10.1037/a0013262
- Kawamoto, R., & Doi, T. (2002). Self-reported functional ability predicts three-year mobility and mortality in community-dwelling older persons. *Geriatrics and Gerontology International*, 2, 68–74. doi:10.1046/j.1444-1586.2002.00024.x
- Kok, B. E., Coffey, K. A., Cohn, M. A., Catalino, L. I., Vacharkulksemsuk, T., Algoe, S. B., ... Fredrickson, B. L. (2013). How positive emotions build physical health: Perceived positive social connections account for the upward spiral between positive emotions and vagal tone. *Psychological Science*, 24, 1123–1132. doi:10.1177/0956797612470827
- Lyubomirsky, S., Sheldon, K. M., & Schkade, D. (2005). Pursuing happiness: The architecture of sustainable change. *Review of General Psychology*, 9, 111–131. doi:10.1037/1089-2680.9.2.111
- Moskowitz, J. T. (2003). Positive affect predicts lower risk of AIDS mortality. *Psychosomatic Medicine*, 65, 620–626. doi:10.1097/01.PSY.0000073873.74829.23
- Pressman, S. D., Acevedo, A. M., Aucott, K., & Kraft, T. L. (under review). Smiling through the pain: The clinical benefits of facial expression during needle injection.
- Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin*, 131, 925–971. doi:10.1037/0033-2909.131.6.925
- Pressman, S. D., & Cohen, S. (2012). Positive emotion word use and longevity in famous deceased psychologists. *Health Psychology*, 31, 297–305. doi:10.1037/a0026071
- Pressman, S. D., Jenkins, B. N., Kraft, T. L., Rasmussen, H., & Scheier, M. (2017). The whole is not the sum of its parts: Specific types of positive affect influence sleep differentially. *Emotion*, 17(5), 778–793.
- Sin, N. L., & Lyubomirsky, S. (2009). Enhancing well-being and alleviating depressive symptoms with positive psychology interventions: A practice-friendly meta-analysis. *Journal of Clinical Psychology*, 65, 467–487. doi:10.1002/jclp
- Sturgeon, J. A., & Zautra, A. J. (2013). Psychological resilience, pain catastrophizing, and positive emotions: Perspectives on comprehensive modeling of individual pain adaptation topical collection on psychiatric management of pain. *Current Pain and Headache Reports*, 17. doi:10.1007/s11916-012-0317-4
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54, 1063–1070. doi:10.1037/0022-3514.54.6.1063

Suggested Reading

- Boehm, J. K., Peterson, C., Kivimaki, M., & Kubzansky, L. (2011). A prospective study of positive psychological well-being and coronary heart disease. *Health Psychology, 30*, 259–267. doi:10.1037/a0023124
- Chida, Y., & Steptoe, A. (2008). Positive psychological well-being and mortality: A quantitative review of prospective observational studies. *Psychosomatic Medicine, 70*, 741–756. <http://doi.org/10.1097/PSY.0b013e31818105ba>
- Cohen, S., Alper, C. M., Doyle, W. J., Treanor, J. J., & Turner, R. B. (2006). Positive emotional style predicts resistance to illness after experimental exposure to rhinovirus or influenza a virus. *Psychosomatic Medicine, 68*, 809–815. <http://doi.org/10.1097/01.psy.0000245867.92364.3c>
- Moskowitz, J. T. (2003). Positive affect predicts lower risk of AIDS mortality. *Psychosomatic Medicine, 65*, 620–626. <http://doi.org/10.1097/01.PSY.0000073873.74829.23>
- Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971. <http://doi.org/10.1037/0033-2909.131.6.925>

The Precaution Adoption Process Model

Neil D. Weinstein¹, Peter M. Sandman¹,
and Susan J. Blalock²

¹*Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA*

²*Eshelman School of Pharmacy, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA*

To understand why many young adults put themselves at risk for AIDS, it seems logical to assess their beliefs about HIV and AIDS. A questionnaire based on some popular theories of health behavior might ask them about the likelihood that they will have sexual contact with someone who is HIV positive, their chance of becoming infected by this person, the effectiveness of various precautions, the social consequences of taking precautions, and other related topics.

This research strategy makes sense today. But in 1987, when the public was first hearing about AIDS, young adults would be completely unable to answer most of the questionnaire. The riskiness of their behavior would vary: some would have had many sexual partners; others would have had few or none. Some would use condoms, and others would not. Yet, these behaviors could not be explained or predicted by their beliefs about AIDS. *They had not yet formed such beliefs.*

As this example shows, theories that try to explain health behavior by focusing on beliefs about the costs and benefits of particular actions are relevant only to people who have been engaged sufficiently by the health threat to have formed such beliefs. The precaution adoption process model (PAPM) seeks to identify the stages that people need to pass through in order to commence health protective behaviors—including stages prior to being engaged by the issue—and to determine the factors that cause people to move from one stage to the next.

How Stage Theories Approach the Issue of Explaining and Changing Behavior

Many theories of individual health behavior, such as those focusing on the perceived pros and cons of action, specify and use a single equation to predict behavior. These theories acknowledge

quantitative differences among people in their positions on different variables and, consequently, in their likelihood of action. The goal of interventions is to maximize the variables that increase the value of the prediction equation. Any action-promoting variable that has not already reached its maximum value is an appropriate target for intervention.

Advocates of stage theories, like PAPM, claim that there are *qualitative* differences among people and question whether changes in health behaviors can be described by a single prediction equation. They suggest, in effect, that we must develop a series of explanatory equations, one for each stage transition. This is a much more complicated goal than finding a single prediction rule, but it offers the possibility of improving prediction, accuracy, and intervention effectiveness and efficiency.

Stage theories have four principal elements and assumptions (Weinstein, Rothman, & Sutton, 1998):

- 1 A category system to define the stages

Stages are theoretical constructs. An ideal or “prototype” must be defined for each stage even if few people perfectly match this ideal. Furthermore, stages can be useful constructs even if the actual boundaries between stages are not as clear-cut as the theories suggest.

- 2 An ordering of the stages

Stage theories assume that before people act, they usually pass through all the stages in order. Forward progression, however, is neither inevitable nor irreversible. There is no minimum length of time people must spend in a particular stage. In fact, people may sometimes progress so rapidly that, for practical purposes, they can be said to skip stages (e.g., when a doctor recommends a new vaccine and the patient gets the shot without any further deliberation). Some stages may lie outside the route to action. An example is a stage representing people who have decided not to act.

- 3 Common barriers to change facing people in the same stage

Knowing the stage of an individual or group is helpful in designing an intervention program only if people at that stage must address similar types of issues before they can progress to the next stage. Thus, interventions can be tailored on the basis of stage.

- 4 Different barriers to change facing people in different stages

If factors producing movement toward action were the same regardless of a person’s stage, the same intervention could be used for everyone, and the concept of stages would be superfluous.

A completely specified stage theory would include both the criteria that define stages and factors that govern movement between stages. Although stage definitions are meant to apply across behaviors, the specific barriers to progress may be behavior or hazard specific. Factors that enter into decisions to lose weight, for example, may be quite different from those that affect decisions to use condoms. A model that proposes a particular sequence of stages in the change process could be correct about these stages even if it has not identified all the determinants of each transition from one stage to the next. At present, the PAPM does not provide detailed information about barriers at each stage. It is a conceptual framework or skeleton that needs to be fleshed out with information about why each stage transition occurs.

The Precaution Adoption Process Model

Description of the Model

The PAM attempts to explain how a person comes to a decision to take action and how he or she translates that decision into action. Adoption of a new precaution or cessation of a risky behavior requires deliberate steps unlikely to occur outside of conscious awareness. The PAM applies to these types of actions, not to the gradual development of habitual behaviors, such as exercise and diet, in which health considerations may play little role (though it would apply to the initiation of a new exercise program or a new diet). Nor does the PAM explain the commencement of risky behaviors—such as a teenager accepting her first cigarette—which seem to be better explained in terms of a “willingness” to act than in terms of any decision to act (Gibbons, Gerrard, Blanton, & Russell, 1998).

All PAM stages prior to action are defined in terms of mental states, rather than in terms of factors external to the person, such as current or past behaviors. Neither are PAM stages defined by criteria that are salient only to health professionals. For example, having less than 20% of calories in one’s diet come from fat might be a behavioral goal of importance to a dietitian, but laypeople are unlikely to be aware of or be able to track their progress toward such a goal. Their goals, and the appropriate definitions of stages for them, are more likely to center on particular foods, such as increasing the consumption of fish to three meals a week, and PAM stages are defined in terms of goals that reflect lay objectives.

The present formulation of the PAM (Weinstein & Sandman, 1992) identifies seven stages along the path from lack of awareness to action (see Figure 1). At some initial point in time, people are unaware of the health issue (stage 1). When they first learn something about the issue, they are no longer unaware, but they are not yet engaged by it either (stage 2). People who reach the decision making stage (stage 3) have become engaged by the issue and are considering their response. This decision making process can result in one of three outcomes: They may suspend judgment, remaining in stage 3 for the moment. They may decide to take no action, moving to stage 4 and halting the precaution adoption process, at least for the time being. Or they may decide to adopt the precaution, moving to stage 5. For those who decide to adopt the precaution, the next step is to initiate the behavior (stage 6). A seventh stage, if relevant, indicates that the behavior has been maintained over time (stage 7).

The stages have been labeled with numbers, but these numbers have no more than ordinal values. They would not even have ordinal value if stage 4 were included, since it is not a stage on the path to action. The numbers should never be used to calculate correlation coefficients, the mean stage for a sample, or conduct regression analyses with stage treated as a continuous variable. Such calculations assume that the stages represent equal-spaced intervals along a single underlying dimension, which violates a fundamental assumption of stage theory. Although

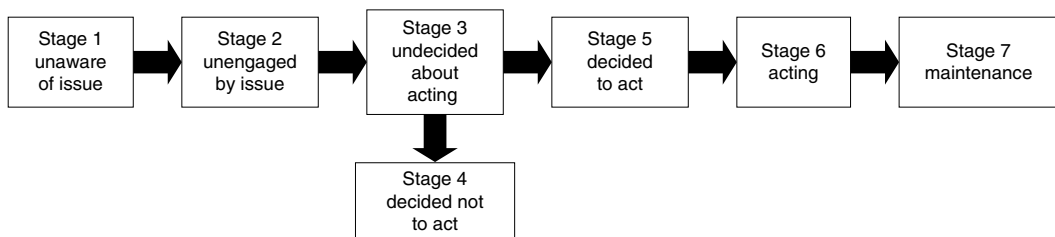


Figure 1 Stages of the precaution adoption process model.

not shown in Figure 1, movement backward toward an earlier stage can also occur, without necessarily going back through all the intermediate stages, although it is not possible to go from later stages to stage 1.

On the surface, the PAPM resembles another stage theory, the transtheoretical model developed by Prochaska and DiClemente (1983). However, even those stages with similar names in the two theories are defined according to quite different criteria. For example, the PAPM refers primarily to mental states, whereas the TTM emphasizes days or months until intended action. We are not aware of any research directly comparing the two theories.

Justification for the PAPM Stages

There should be good reasons to propose the separate stages that make up a stage theory. What is the justification for the stages in the PAPM?

Stage 1 (unaware)

Much health research deals with well-known hazards, like smoking, AIDS, and high-fat diets. In such cases, asking someone about his or her beliefs and plans is quite reasonable; most people have considered the relevance of these threats to their own lives. But if people have never heard of a hazard or a potential precaution, they cannot have formed opinions about it. The reluctance of respondents to answer survey questions about less familiar issues suggests that investigators ought to allow people to say that they “do not know” or have “no opinion” rather than forcing them to state a position. Participants in many health behavior investigations are not given this opportunity. Even when participants are permitted to say that they “do not know,” these responses are often coded as missing or are collapsed into another category. To say “I do not know” indicates something important and is real data that should not be discarded.

Media often have a major influence in getting people from stage 1 of the PAPM to stage 2 and from stage 2 to stage 3 and much less influence thereafter. This and other factors that may be important in producing different transitions are given in Table 1 and in Weinstein (1988). These are suggestions for consideration, not core assumptions of the PAPM.

Table 1 Factors likely to determine progress between stages.

<i>Stage transition</i>	<i>Factor</i>
Stage 1 to stage 2	Media messages about the hazard and precaution
Stage 2 to stage 3	Media messages about the hazard and precaution Communications from significant others Personal experience with hazard
Stage 3 to stage 4 or stage 5	Beliefs about hazard likelihood and severity Beliefs about personal susceptibility Beliefs about precaution effectiveness and difficulty Behaviors and recommendations of others Perceived social norms Fear and worry
Stage 5 to stage 6	Time, effort, and resources needed to act Detailed “how-to” information Reminders and other cues to action Assistance in carrying out action

Stage 2 (unengaged) versus stage 3 (undecided)

Once people have heard about a health precaution and have begun to form opinions about it, they move out of stage 1. However, so many issues compete for their limited time and attention that people can know a moderate amount about a hazard or a precaution without ever considering whether they need to do anything about it.

We believe that this condition of awareness without personal engagement is quite common. In a 1986 survey of radon testing (Weinstein, Sandman, & Klotz, 1987), for example, 50% of respondents in a high-risk region said they had never thought about testing their own homes even though all indicated that they knew what radon was, and most correctly answered more than half of the questions on a knowledge test.

The PAPM suggests further that it is important to distinguish between people who have never thought about an action and those who have given the action some consideration but are undecided. There are several reasons for making this distinction. People who have thought about acting are likely to be more knowledgeable. Also, getting people to think about an issue may require different sorts of communications (and entail different sorts of obstacles) than getting them to adopt a particular conclusion. Thus, whether a person has or has not thought about taking action appears to be an important distinction.

Stage 3 (undecided) versus stage 4 (decided not to act) and stage 5 (decided to act)

Research reveals important differences between people who have not yet formed opinions and those who have made decisions. People who have come to a definite position on an issue, even if they have not yet acted on their opinions, have different responses to information and are more resistant to persuasion than people who have not formed opinions (Cialdini, 1988; Ditto & Lopez, 1992; Nisbett & Ross, 1980, chapter 8). This widely recognized tendency to adhere to one's own position has been termed "confirmation bias." It manifests itself in a variety of ways. According to Klayman (1995), these include overconfidence in one's beliefs, searches for new evidence that are biased to favor one's beliefs, biased interpretations of new data, and insufficient adjustment of one's beliefs in light of new evidence. For these reasons, the PAPM holds that it is significant when people say that they have decided to act or have decided not to act and that the implications of someone saying that they have decided not to act are not the same as saying it is "unlikely" they will act.

We believe that cost-benefit theories of health behavior, such as the health belief model, the theory of reasoned action, protection motivation theory, and subjective expected utility theory, are dealing mainly with factors that govern how people who get to stage 3 decide what to do. Factors these theories focus on are certainly important, but they relate mostly to this one phase of the precaution adoption process. These theories also overlook another possibility that people faced with a difficult decision might get stuck and quit trying to make up their minds, moving back to stage 2. Determinants of this regression to an earlier stage might be different from the factors that lead people toward stages 4 or 5.

Perceived susceptibility is one factor that can influence what people decide and is included in most theories of health behavior (Connor & Norman, 1995). People are reluctant to acknowledge personal susceptibility to harm even when they acknowledge risks faced by others. Consequently, overcoming this reluctance is a major barrier to getting people to decide to act.

Stage 5 (decided to act) versus stage 6 (acting)

The distinction between decision and action is common to most stage theories. For example, Schwarzer's health action process approach (Schwarzer, 1992) distinguishes between an initial motivation phase during which people develop an intention to act, based on beliefs about risk,

outcomes, and self-efficacy, and the volition phase in which they plan the details of action, initiate action, and deal with the difficulties of carrying out that action. Even Ajzen's (Ajzen, 1985) theory of planned behavior, which is not a stage theory, separates intentions from actions. Protection motivation theory (Rogers & Prentice-Dunn, 1997) is also not a stage theory, but its developers implicitly recognize the need for sequencing interventions. According to these authors "[Protection Motivation Theory] experiments always present information in the same order: threatening information followed by coping information" (p. 116). These researchers also speak of first developing motivation and then developing coping skills.

A growing body of research suggests that there are important gaps between intending to act and carrying out this intention and that helping people develop specific implementation plans can reduce these barriers. The PAPM suggests that detailed implementation information would be uninteresting to people in early stages. Yet, for people who have decided to act, such information is often essential to produce the transition from decision to action. This claim is echoed by temporal construal theory (Trope & Liberman, 2003), which asserts that decisions about action are based initially on abstract construals of the options but become more focused on concrete details as the actual choice approached.

Stage 6 (acting) versus stage 7 (maintenance)

For any health behavior that is more than a one-time action, adopting the behavior for the first time is different from repeating the behavior at intervals, or developing a habitual pattern of response. Once a woman gets her first mammogram, for example, she will have acquired both more information in general and personal experience (perhaps both positive and negative). These will influence the decision to be rescreened. Similarly, a man who stops smoking or loses weight must deal with the acute withdrawal experience and/or the glow of success in the early stage of taking action but must address different challenges in the maintenance stage. The distinction between action and maintenance is widely recognized (e.g., Marlatt & Gordon, 1985; Meichenbaum & Turk, 1987).

Stages of inaction

One value of the PAPM is its recognition of differences among the people who are neither acting nor intending to act. People in stage 1 (unaware), stage 2 (unengaged), stage 3 (undecided), and stage 4 (decided not to act) all fit in this broad category. Those in stage 1 need basic information about the hazard and recommended precautions. People in stage 2 need something that makes the threat and action personally relevant. Individualized messages and contact with friends and neighbors who have considered action should help these individuals move to the next stage. Another powerful influence on the transition from stage 2 to stage 3 is the awareness that others are making up their minds and that one is obliged to have some opinion on this current issue of the day.

As stated earlier, people who have thought about and rejected action, stage 4, are a particularly difficult group. Evidence shows that they can be quite well informed (Blalock et al., 1996; Weinstein & Sandman, 1992) and, as noted earlier, they will tend to dispute or ignore information that challenges their decision not to act.

Criteria for Applying Stage-Based Interventions

A variety of issues should be considered to determine the practical utility of the PAPM or any other stage theory.

Superiority Over Unstaged Messages

The value of a stage theory depends on the extent to which it leads to interventions that are more effective than generic messages.

One might ask, “Why not just employ combination treatments that contain elements for people at each stage?” There are several problems with this suggestion. First, a combination treatment will be much longer than a stage-matched treatment. Media time is expensive, speakers usually have a limited length of time for their presentations, and audiences have a limited attention span. Thus, attempting to replace a stage-focused intervention with a multi-faceted combination treatment would involve substantial costs.

Second, people are most likely to be engaged by a treatment that matches their stage, and a mismatched treatment may lose their attention. Thus, members of the general public who have already decided to act may be put off by basic risk information and may fail to attend to the subsequent, detailed procedural information describing what they do need. Nevertheless, if only a single message can be given to a mixed-stage audience, a combination intervention would probably be the most appropriate.

Stage Assessment

A second criterion is the ability to identify stages accurately and efficiently. The PAPM requires only a simple process to assess a person’s stage, so it can be used easily in individual and small-group settings. Clinicians could integrate this assessment without disrupting their practices. Similarly, a filtering question on a website can assess stage and send visitors to the page most relevant to their stage. Even in a large audience, a show of hands might be used to determine the distribution of stages present.

Delivery of Stage-Targeted Messages

The feasibility of delivering stage-targeted messages in different situations varies greatly. If communication is one on one, delivering the message appropriate for an individual is relatively easy. In group settings, messages can be chosen to fit the overall audience, though not individual members. In mass communications, a stage approach is more often practicable with print than with broadcast media. Within print channels, pamphlets and magazines offer more opportunities for stage targeting than newspapers; within broadcasting, cable offers more opportunities for stage targeting than networks. The Internet makes it possible for individual users to choose different information pathways depending on their self-perceived information needs. This should provide unprecedented opportunities for low-cost message targeting, though evidence on the cost of such tailored messaging is scanty. There is evidence, though, that people in different PAPM stages perceive themselves as needing different types of information (Weinstein, Lyon, Sandman, & Cuite, 1998).

The ability to deliver targeted messages to members of a group also depends on the range of stages present in that group. The greater the range of stages, the more difficult it is to choose a single message. For a mass audience, the most efficient way to encourage a new health protective action may be with a comprehensive broadcast message that ignores stage or assumes everyone to be at a very early stage. As the issue matures, however, distinctive audiences, separable by stage, merit distinctive messages, and print or “narrowcasting” becomes the medium of choice for mass communications.

The Difficulty of Behavior Change

A final important criterion concerns the difficulty of the action being advocated and the expected resistance of the audience to the behavior change recommendation. When a behavior is easy and resistance is low, stage may matter little. In such situations, interventions and messages needed to help people progress from stage to stage can be brief, and several may be combined into a single comprehensive treatment. In contrast, when change is difficult and resistance is high, there is a greater need to have separate messages for each stage. Note that although some precautions—such as changing to a fluoride-containing toothpaste—are easy to carry out, the ease of others may vary greatly from one person to the next. Health professionals should never assume that a behavior is easy without considering carefully the obstacles that may exist.

Using the PAM to Develop and Evaluate Behavior Change Interventions

Blalock and colleagues used the PAM in three studies conducted from 1994 to 2000 that focused on osteoporosis prevention (Blalock et al., 2002). Osteoporosis is a metabolic bone disorder that results in decreased bone density and increased susceptibility to fracture (Riggs & Melton, 1986). Precautions recommended to reduce the risk of developing osteoporosis include adequate calcium intake and weight-bearing exercise (Kling, Clarke, & Sandhu, 2014). Their research was designed to better understand the factors that (a) discriminate among women in different stages with respect to calcium intake and exercise and (b) predict different types of stage transitions (Blalock et al., 1996). This information was used to develop stage-based educational interventions. These studies provide examples of the necessary steps in using the PAM to develop and evaluate behavior change interventions.

The first step involves identifying and clearly defining the behavior of interest. Although the PAM focuses on the adoption of specific health behaviors (e.g., “daily walking for at least 30 min”), it may also be used to intervene at a broader behavioral category level (e.g., “increasing exercise”). In either case, care should be taken to define the target behavior(s) in terms that are meaningful to laypeople. Although Blalock and colleagues defined the target behavior in terms of a specific daily calcium intake, a value that has little meaning to most laypeople, they overcame this problem by providing study participants with feedback that informed them of their current calcium intake. This step would not have been needed if the behavior criterion had been simpler, such as “using a calcium supplement.”

Second, a system must be developed to classify individuals according to their current stage. The stage classification system allows health professionals to assess the distribution of stages within a target population at a particular point in time, guiding the design of both individual and community-level interventions. As described in the AIDS example at the beginning of this chapter, if awareness and knowledge of a health threat change over time, the effectiveness of different types of interventions is likely to change as well. Thus, monitoring temporal changes in the distribution of people across stages makes it possible to design dynamic interventions that accommodate the dynamic nature of the behavior change process.

Third, it is necessary to have at least a preliminary understanding of the factors that influence different types of stage transitions. This understanding is needed to tailor interventions to people, or groups of people, who are in different stages of change. Early work by Blalock et al. (1996) suggested that to move people, or groups of people, from stages 1 and 2 (unaware,

unengaged) to stage 3 (undecided), interventions should focus on increasing awareness of the health problem of interest, behavioral recommendations to minimize risk, and potential benefits associated with adopting the behavioral recommendations—including the effectiveness of the recommended behaviors in terms of risk reduction (i.e., precaution effectiveness).

The cross-sectional and prospective research carried out by Blalock and colleagues examined between-stage differences. Significant differences between stages on particular variables suggest that these variables are worthy of additional attention—and of inclusion in interventions at early stages of research—but they are not proof of causation. Furthermore, if interventions succeed in altering these variables but fail to move people to stages closer to action, this does not prove that the stages themselves are invalid. The variables that actually cause each stage transition must be identified empirically.

Fourth, intervention strategies are needed to address variables associated with different stage transitions. For example, media campaigns and informational materials may be able to increase awareness of a health problem, behavioral recommendations, and the benefits associated with action. However, more intensive interventions are often needed to help individuals acquire the skills and resources needed to support behavior change efforts. The intensity of the intervention required will depend on the behavior of interest and what barriers need to be overcome. For example, Blalock and colleagues used a combination of written materials and telephone counseling focused on helping women identify potential barriers to action and develop strategies to overcome the barriers identified. This approach led to a significant increase in calcium intake among women who were thinking about or trying to increase their calcium intake at baseline. However, a similar intervention focused on exercise had no effect on exercise level. Fifth, health educators must specify how the effectiveness of the intervention will be determined. Will it be considered effective if it results in stage progression, even if the proportion of people in the action and maintenance stages remains the same? Or, is success contingent upon behavior change in the target group? If a behavior is difficult to change and people are in early stages, the PAPM suggests that a single, one-shot intervention—especially an intervention that focuses on movement to the next stage—should not be judged solely by whether it changes behavior.

Finally, educators must determine the timeframe for follow-up assessments. The PAPM and other stage theories suggest that the behavior change process is dynamic. Intervention-induced changes in beliefs and behavior may be transient, so intervention effects may be missed if only long-term follow-up assessments are used. Although long-term behavior change is generally desired, a stage model perspective raises the possibility that even transient changes may be steps in the right direction, helping us to understand the barriers at different stages and increasing the success of subsequent behavior change attempts.

Research Using the PAPM

The PAPM has been applied to many types of health behaviors, including osteoporosis prevention (e.g., Blalock et al., 1996), cancer screening (e.g., Glanz, Steffen, & Taglialatela, 2007), hepatitis B vaccination (Hammer, 1998), home radon testing (e.g., Weinstein, Lyon, et al., 1998), smoking cessation (Borrelli et al., 2002), and red meat consumption (Sniehotta, Luszczynska, Scholz, & Lippke, 2005). As discussed earlier in this chapter, stage theories—with their numerous assumptions about stages and about the changing barriers between stages—are complex. Given the limited, though growing, number of studies relating to the PAPM and the variety of behaviors examined, it is not yet possible to reach firm conclusions

about the model's validity or its helpfulness for designing interventions. For further information on the ways in which the PAPM is being used, the problems researchers encounter in these investigations, and a detailed analysis of stage-matched field study to encourage home radon testing, see Weinstein, Sandman, and Blalock (2008).

Conclusion and Future Directions

Most other (non-stage) theories of individual health behavior regard adoption of new precautions as involving only one step, from inaction to action (or, perhaps, inaction to intention), and the variables typically claimed to produce this step clearly characterize it as a judgment about the relative costs and benefits of action. The PAPM does not reject the variables identified by these theories. Rather, it sees the theories as describing just one part of the precaution adoption process, the stage when people are actively weighing options and deciding what to do. The PAPM, however, shows that other issues important to behavior change arise before people ever think seriously about action and still different issues arise after people have decided to act.

Because the PAPM is not composed of a short list of variables, it does not offer a simple process for designing interventions. Rather, it is a framework that can be used to guide interventions by identifying barriers that inhibit movement from one stage to the next. As additional research is conducted, we will learn more about barriers at each stage and will see how consistent these barriers are from one health behavior to the next.

Acknowledgments

This chapter is adapted from Weinstein et al. (2008).

Author Biographies

Neil D. Weinstein is a professor emeritus of Rutgers, The State University of New Jersey, where he was a member of the Departments of Human Ecology and Psychology. His major areas of scholarship include risk perception, unrealistic optimism about personal risk, communication of risk information, stage theories, the precaution adoption process model, smokers' understanding of the risks of smoking, the relationship between perceived personal risk and health behavior, and theory testing.

Peter M. Sandman was a Rutgers University professor from 1977 to 1995 and is one of the preeminent risk communication and reputation management speakers and consultants in the world today. Dr. Sandman works in three risk communication venues: helping arouse concern when people are insufficiently upset about serious risks, helping diminish concern when people are excessively upset about small risks, and helping guide concern when people are appropriately upset about serious risks.

Susan J. Blalock is a professor in the Eshelman School of Pharmacy at the University of North Carolina at Chapel Hill. A hallmark of her research involves the application of behavioral science theories to understand the mechanisms through which intervention effects are obtained. Her current research focuses on how patients process information concerning medication risks and benefits and the effect this type of information has on patient judgments and decisions regarding medication use.

References

- Ajzen, I. (1985). From intentions to actions: A theory of planned behavior. In J. Kuhl & J. Beckmann (Eds.), *Action control: From cognition to behavior* (pp. 11–39). Heidelberg: Springer Verlag. http://dx.doi.org/10.1007/978-3-642-69746-3_2
- Blalock, S. J., DeVellis, B. M., Patterson, C. C., Campbell, M. K., Orenstein, D. R., & Dooley, M. A. (2002). Effects of an osteoporosis prevention program incorporating tailored educational materials. *American Journal of Health Promotion*, 16, 146–156. <http://dx.doi.org/10.4278/0890-1171-16.3.146>
- Blalock, S. J., DeVellis, R. F., Giorgino, K. B., DeVellis, B. M., Gold, D. T., Dooley, M. A., ... Smith, S. L. (1996). Osteoporosis prevention in premenopausal women: Using a stage model approach to examine the predictors of behavior. *Health Psychology*, 15, 84–93. <http://dx.doi.org/10.1037/0278-6133.15.2.84>
- Borrelli, B., McQuaid, E. L., Becker, B., Hammond, K., Papandanos, G., Fritz, G., & Abrams, D. (2002). Motivating parents of kids with asthma to quit smoking: The PAQS project. *Health Education Research*, 17, 659–669. <http://dx.doi.org/10.1093/her/17.5.659>
- Cialdini, R. B. (1988). *Influence: Theory and practice*. Glenview, IL: Scott Foresman.
- Connor, M., & Norman, P. (1995). *Predicting health behavior*. Philadelphia, PA: Open University Press.
- Ditto, P. H., & Lopez, D. F. (1992). Motivated skepticism: Use of differential decision criteria for preferred and nonpreferred conclusions. *Journal of Personality and Social Psychology*, 63, 568–584. <http://dx.doi.org/10.1037/0022-3614.63.4.568>
- Gibbons, F. X., Gerrard, M., Blanton, H., & Russell, D. W. (1998). Reasoned action and social reaction: Willingness and intention as independent predictors of health risk. *Journal of Personality and Social Psychology*, 74, 1164–1181. <http://dx.doi.org/10.1037/0022-3514.74.5.1164>
- Glanz, K., Steffen, A. D., & Taglialatela, L. A. (2007). Effects of colon cancer risk counseling for first-degree relatives. *Cancer, Epidemiology, Biomarkers and Prevention*, 16, 1485–1491. <http://dx.doi.org/10.1158/1055-9965.EPI-06-0914>
- Hammer, G. P. (1998). Factors associated with hepatitis B vaccine acceptance among nursing home workers. Unpublished doctoral dissertation, Department of Health Policy and Management, Johns Hopkins University.
- Klayman, J. (1995). Varieties of confirmation bias. In J. Busemeyer & R. Hastie (Eds.), *The psychology of learning and motivation. Vol. 32: Decision making from a cognitive perspective*. New York, NY: Academic Press.
- Kling, J. M., Clarke, B. L., & Sandhu, N. (2014). Osteoporosis prevention, screening, and treatment: A review. *Journal of Women's Health*, 23, 563–572. <http://dx.doi.org/10.1089/jwh.2013.4611>
- Marlatt, G. A., & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York, NY: Guilford.
- Meichenbaum, D., & Turk, D. C. (1987). *Facilitating treatment adherence: A practitioner's handbook*. New York, NY: Plenum. <http://dx.doi.org/10.1007/978-1-4684-5359-1>
- Nisbett, R., & Ross, L. (1980). *Human inference: Strategies and shortcomings of social judgment*. Englewood Cliffs, NJ: Prentice-Hall. <http://dx.doi.org/10.2307/2184495>
- Prochaska, J. O., & DiClemente, C. C. (1983). Stages and processes of self-change in smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology*, 51, 390–395. <http://dx.doi.org/10.1037//0022-006X.51.3.390>
- Riggs, B. L., & Melton, L. J. I. (1986). Involutional osteoporosis. *New England Journal of Medicine*, 314, 1676–1686. <http://dx.doi.org/10.1056/NEJM198606263142605>
- Rogers, R. W., & Prentice-Dunn, S. (1997). Protection motivation theory. In D. Gochman (Ed.), *Handbook of health behavior research. Vol. 1. Determinants of health behavior: Personal and social* (pp. 113–132). New York, NY: Plenum.
- Schwarzer, R. (1992). Self-efficacy in the adoption and maintenance of health behaviors: Theoretical approaches and a new model. In R. Schwarzer (Ed.), *Self-efficacy: Thought control of action* (pp. 217–242). Washington, DC: Hemisphere.

- Sniehotta, F. F., Luszczynska, A., Scholz, U., & Lippke, S. (2005). Discontinuity patterns in stages of the precaution adoption process model: Meat consumption during a livestock epidemic. *British Journal of Health Psychology*, *10*, 221–235. <http://dx.doi.org/10.1348/135910705X26137>
- Trope, Y., & Liberman, N. (2003). Temporal construal theory. *Psychological Review*, *100*, 403–421. <http://dx.doi.org/10.1037/0033-295X.110.3.403>
- Weinstein, N. D. (1988). The precaution adoption process. *Health Psychology*, *7*, 355–386.
- Weinstein, N. D., Lyon, J. E., Sandman, P. M., & Cuite, C. L. (1998). Experimental evidence for stages of precaution adoption. *Health Psychology*, *17*, 445–453. <http://dx.doi.org/10.1037/0278-6133.17.5.445>
- Weinstein, N. D., Rothman, A., & Sutton, S. (1998). Stage theories of health behavior. *Health Psychology*, *17*, 290–299. <http://dx.doi.org/10.1037/0278-6133.17.3.290>
- Weinstein, N. D., & Sandman, P. M. (1992). A model of the precaution adoption process: Evidence from home radon testing. *Health Psychology*, *11*, 170–180. <http://dx.doi.org/10.1037/0278-6133.11.3.170>
- Weinstein, N. D., Sandman, P. M., & Klotz, M. L. (1987). *Public response to the risk from radon, 1986*. New Brunswick, NJ: Environmental Communications Research Program, Rutgers University.
- Weinstein, N. D., Sandman, P. S., & Blalock, S. J. (2008). In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *The precaution adoption process model* (pp. 123–147). San Francisco, CA: Jossey-Bass.

Prejudice and Stereotyping in Healthcare

Elizabeth S. Focella

Department of Psychology, University of Wisconsin Oshkosh, Oshkosh, WI, USA

In the social psychological literature, stereotypes are defined as beliefs, usually overgeneralizations, associating particular traits and attributes with members of a particular group (e.g., “members of this group are lazy”). Prejudice, in contrast, is an attitude (usually unfavorable, e.g., dislike) toward a person based solely on that person’s presumed group membership. Both stereotyping and prejudice may be considered a form of bias. Literature in social psychology has long been interested in the concepts of stereotyping and prejudice as a way to understand intergroup relations. More recently, stereotyping and prejudice have been studied in the context of healthcare to help explain the apparent health disparities between groups, particularly between Whites (a term that will be used hereafter to refer to a diverse group of people who identify as White, Caucasian, or European American and people of European descent who may or may not be American) and Blacks (a term that will be used hereafter to refer to a diverse group of people who identify as Black or African American and people of African descent who may or may not be American).

Researchers in a variety of disciplines, including psychology, medicine, and public health, find that even when controlling for factors such as access to healthcare, low socioeconomic status (SES), and disease severity, members of low-power groups (Blacks in particular) still have worse health outcomes, including increased risk of mortality. Among the potential sources for this disparity, some research documents the presence of differential treatment in numerous areas of healthcare, including medicine, surgery, primary care, and mental health, and for a variety of conditions and procedures, including coronary artery disease, renal disease and kidney transplantation, stroke, cancer, HIV/AIDS, medication for pain management, and advice for smoking cessation (e.g., see Geiger, 2003). Among a number of explanations for these disparities in treatment and outcomes, stereotyping and prejudice in healthcare has received compelling empirical support (for a review, see Chapman, Kaatz, & Carnes, 2013; Dovidio & Fiske, 2012).

A substantial portion of literature concerning stereotyping and prejudice in healthcare examines racial/ethnic bias, particularly against Black patients (van Ryn & Burke, 2000). This is likely due to the fact that Blacks have poorer health outcomes, including risk of mortality, than other racial and ethnic groups. Hence, the literature on bias against Blacks represents a large portion of our empirical knowledge on the topic of bias in healthcare. The literature examining stereotyping and prejudice in healthcare does, however, document prejudice and stereotyping for other racial and ethnic groups, including Hispanic and Native American patients, and exists beyond examination of racial and ethnic bias, including bias toward other low-power groups including people who identify as lesbian, gay, bisexual, or transgender (LGBT), women, the elderly, and people who are overweight, are diagnosed with mental illness, and abuse drugs and alcohol.

At times, people are able to consciously access and report their feelings (prejudice) and beliefs (stereotypes) about particular groups of people. In such cases, prejudices and stereotypes are conscious, or *explicit*. In the past few decades, however, research has shown that in some cases, people hold stereotypes and prejudices that they are not consciously aware of (i.e., nonconscious), or are *implicit*. The literature on prejudice in healthcare examines both *explicit* and *implicit* stereotyping and prejudice among clinicians.

Explicit Stereotyping and Prejudice

In the context of healthcare, the literature documenting clinicians' explicit bias is comparatively less robust than literature documenting implicit bias. This may be the case because most physicians and other healthcare providers intend to treat their patients fairly and may not wish to endorse stereotyping or prejudice directed at these groups, thus making the prevalence of explicit stereotyping and prejudice less pronounced among healthcare providers. When explicit stereotypes are documented, they are typically directly related to concerns about compliance and adherence to treatment. Explicit negative attitudes (i.e., dislike) are typically less reported than stereotypes and are often expressed toward groups for which bias is more socially accepted (e.g., people who are overweight, people who are drug users).

The literature finds that racial and ethnic groups including Blacks, Hispanics, and Native Americans, as well as members of other groups, including persons who are overweight and former injecting drug users, are explicitly stereotyped by healthcare providers as noncompliant. For example, one study found that medical and nursing students reported awareness of stereotypes associating Hispanics and Native Americans with noncompliance (Bean et al., 2014). Similarly, other studies show that healthcare providers stereotype Blacks, people who are overweight, and former injecting drug users as less likely to adhere to treatment. Further, the aforementioned study of medical and nursing students found that medical and nursing students reported awareness of stereotypes associating Hispanics and Native Americans with risky health behavior and difficulty understanding and/or communicating health-related information.

While the stereotype of noncompliance is associated with several groups, other stereotypes are more group specific. In particular, overweight patients face more unique stereotypes in addition to noncompliance, including perceptions that they lack motivation and are lazy, self-indulgent, and physically exhausting to care for. Other more general negative stereotypes toward overweight patients include being lower in social attractiveness, as well as physically unattractive.

Other well-documented explicit stereotypes in healthcare relate to perceptions of controllability, blame, and fault for the patient's health state. These beliefs are often associated with patients who abuse drugs and alcohol, are overweight, are diagnosed with mental illness, and

are current or former injecting drug users. Interestingly, these negative attitudes toward those who abuse substances may be influenced by beliefs about controllability and social mores. Literature on healthcare providers' beliefs about overweight patients, as well as those coping with mental illness (e.g., those with schizophrenia) and substance abuse, shows that a portion of healthcare providers express the belief that those patients are responsible for their current state. For example, as documented in some literature, nurses have expressed the belief that their overweight patients' failure to lose weight can be attributed to noncompliance with treatment recommendations and lack of motivation. Further, the literature suggests a link between these negative stereotypes and the clinician's preexisting beliefs about personal responsibility. One study shows that clinicians who identify as conservative, as opposed to liberal, may have more negative attitudes toward patients who are injecting drug users. Mediation analyses indicated that conservative clinicians' negative attitudes toward injecting drug users are due to the belief that injecting drug use is in the patient's control (Brener, Hippel, Kippax, & Preacher, 2010). Taken together, the literature suggests that negative attitudes toward these groups of patients can be characterized by negative personal attributions for the patients' state.

In relation to stereotyping, literature documents the possibility that healthcare providers may, at times, overly attribute their patients' physical ailments to the patients' more salient condition. For example, studies show that nurses in particular express the belief that their overweight patients' ailments are likely the result of their patients' weight and that nurses caring for patients who are injecting drug users express the belief that their patients' ailments are caused by their injecting drug user status. Importantly, this occurs regardless of whether the ailment is in fact due to the patients' obesity or drug use. The tendency to overly rely on a patient's group membership when making a determination about their treatment and diagnoses is a form of stereotyping and presents a potential for diagnostic overshadowing, in which a patients' symptoms and ailments are blamed on another, more obvious condition. In diagnostic overshadowing, a disease or otherwise harmful or life-threatening condition is not detected because it is "overshadowed" by a more obvious and more salient condition—a circumstance that delays treatment for the true cause of the patient's ailments and possibly an increased likelihood of death. Diagnostic overshadowing occurs for people with a variety of conditions including people who are overweight, have intellectual disabilities, have physical disabilities, and are diagnosed with mental illness.

In addition to explicit stereotyping of patients, the literature also documents the existence of prejudice. In a study about prejudice toward schizophrenic patients, researchers found that healthcare providers showed greater desire for social distance from a schizophrenic, compared with a non-schizophrenic patient. Other research documents some evidence of explicit prejudice against Black patients, including physicians' lower feelings of affiliation with Black, compared with White patients. Further, prejudice toward those who are overweight among healthcare providers is widely documented, including some healthcare providers' feelings of disgust, repulsion, and pity for their obese patients. Physicians also express embarrassment at the prospect of discussing sexuality and sexually related healthcare with LGBT patients. The literature also documents more general negative affective responses of healthcare providers toward those who abuse drugs and alcohol, as well as those with mental disorders.

Implicit Stereotyping and Prejudice

Implicit bias can be assessed in a number of ways including psychophysical reactions, neuroimaging, and computerized reaction time tasks that measure participants' associations of particular

groups of people with certain attributes as well as general evaluative concepts like “good” or “bad.” Implicit bias is most often assessed through a particular reaction time task known as the Implicit Association Test (IAT). The IAT is administered on a computer, in which participants are asked to categorize a set of words or images. The IAT measures the strength of associations between particular concepts (typically groups of people such as Blacks and Whites) and evaluations (such as good or bad) or stereotypes (such as noncompliant). The notion behind the IAT is that when a person holds an implicit association between a group of people and a stereotype or evaluation (e.g., bad), they should be able to categorize that group of people with words related to the stereotype or evaluation more quickly and with greater accuracy compared with when they are asked to categorize that stereotype or evaluation with other groups of people. For example, if a clinician holds an association between Blacks and noncompliance, they should be able to more easily categorize Blacks with words related to noncompliance compared with when they are asked to categorize Whites with noncompliance.

The literature documenting implicit stereotypes and prejudice among healthcare providers documents a number of empirical findings. First, numerous studies on implicit bias in healthcare show that both physicians and other clinicians such as nurses hold an implicit, and often strong, preference for Whites relative to Blacks. The literature also documents a similar bias against those who are overweight. On a “fat–thin” IAT, healthcare providers show an implicit preference for thin, compared with fat, people. The literature also finds a similar preference among physicians and nurses toward people who are higher, compared with lower, in SES, and there is some discussion in the literature about physicians’ implicit as well as explicit bias against LGBT older adults (e.g., see Foglia & Fredrikson-Goldsen, 2014). Interestingly, the implicit bias that physicians and nurses exhibit toward Blacks and those who are overweight is similar to that of the larger population, suggesting that current medical education does little to reduce implicit bias.

The literature on implicit bias toward patients also shows evidence of stereotyping. Often, these implicit stereotypes pertain to noncompliance. Numerous studies show that physicians, nurses, and medical students implicitly associate Blacks, for example, as being noncompliant, medically uncooperative, and uncooperative generally. Literature shows that Hispanics are also implicitly stereotyped as noncompliant, as well as implicitly associated with health risk (Bean, Stone, Moskowitz, Badger, & Focella, 2013). Notably, the literature also shows that Blacks are not only implicitly associated with diseases that are epidemiologically relevant for Blacks (e.g., sickle cell anemia) but that they are also implicitly associated with conditions and social behaviors with no inherently biological or genetic relationship with Blacks including obesity and drug use (Moskowitz, Stone, & Childs, 2012).

The Influence of Stereotyping and Prejudice on Clinical Decision Making and Interactions with Patients

Some recent empirical studies not only document the existence of prejudice and stereotyping among clinicians but also show that these biases influence their clinical decision making, including treatment, as well as their interactions with patients. In a landmark study by Green et al. (2007), physicians read a clinical vignette of either a Black or White patient presenting to the emergency department with an acute coronary syndrome. These physicians were later asked to make recommendations of whether to treat the patient with thrombolysis. Green et al. (2007) found that while physicians reported no explicit preference for Whites relative to Blacks or differences between Whites and Blacks on perceived cooperativeness, they did exhibit

an implicit preference favoring Whites over Blacks as well as implicit stereotypes of Blacks as less cooperative with medical procedures and less cooperative more generally. Importantly, they identified a link between physicians' implicit bias and their treatment recommendations—as physicians' implicit pro-White bias increased, the lower their likelihood of treating Black patients with thrombolysis.

Other studies show similar findings, providing evidence that bias influences clinical decision making. For example, literature shows that implicit bias against the mentally ill can predict overdiagnosis, while explicit bias predicts more negative prognoses (Peris, Teachman, & Nosek, 2008). Additionally, research finds that perceptions of cooperativeness are predictive of treatment recommendations. This may disadvantage members of minority groups including Blacks, Hispanics, people who are overweight, and people who are mentally ill, who are stereotyped (implicitly as well as explicitly) as being less medically cooperative relative to Whites, the thin, and those not labeled as mentally ill.

Further, it is widely observed that White patients are more likely to receive pain treatment than non-White patients. This disparity in treatment, research indicates, can be predicted by physicians' implicit pro-White bias. For example, as pediatricians' implicit pro-White bias increases, the lower their likelihood of prescribing narcotic medication to Black patients (Sabin & Greenwald, 2012). To explain why pro-White bias influences pain treatment, some research has explored the possibility of an empathy gap—namely, that physicians with a stronger implicit pro-White bias are less able to empathize with their non-White patients. One study in particular (Drwecki, Moore, Ward, & Prkachin, 2011) showed that the more participants (including undergraduate students, physicians, and nurses) empathized with Whites compared with Blacks, the less pain treatment participants gave to Blacks relative to Whites. This work suggests that the widely observed disparity in pain treatment between Whites and Blacks is due, in part, to clinicians' lowered ability to empathize with, and thus assess, their Black patients' pain.

Stereotyping and prejudice not only influence treatment recommendations, but they also impact the way that clinicians interact with their patients. For example, some research shows that nurses who have implicit prejudice against those who are overweight are less likely to make eye contact with an overweight patient (Persky & Eccleston, 2011). Other studies show that healthcare providers' (including physicians' and genetic counselors') pro-White bias predicts using less emotionally responsive communication when counseling non-White patients and, when interacting with Black patients, more clinical verbal dominance, lower patient positive affect, and poorer ratings of interpersonal care. When interacting with White patients, in contrast, physicians and other healthcare providers exhibit lower levels of verbal dominance, receive more positive ratings of nonverbal effectiveness, and have higher ratings of interpersonal care.

With regard to patient stereotyping, race and compliance stereotyping is associated with longer visits, slower speech, less patient-centeredness, and poorer ratings of interpersonal care with Black patients. When interacting with White patients, in contrast, physicians exhibit less verbal dominance and typically have shorter visits, faster speech, and higher clinician positive affect. Importantly, several studies show that medical visits with minority patients compared with White patients are less patient-centered, which is predicted by implicit pro-White bias. The more pro-White bias on the part of the clinician, the less patient-centered their care of their minority patients (e.g., see Cooper et al., 2012).

Other sophisticated research on the topic of stereotyping and prejudice in healthcare finds interesting nuances and implications of the physician-patient interaction on patient health. For example, research exploring patients' perceptions of discrimination on the part of their

clinicians finds that the more patients' perceive discrimination and bias from their healthcare providers, the lower their adherence to their clinicians' treatment recommendations, which may have negative impacts on their health (e.g., see Bird, Bogart, & Delahanty, 2004).

In addition, research in social psychology finds that while people might be low in explicit prejudice, they could nonetheless harbor nonconscious, or implicit, bias. In the context of healthcare, clinicians who possess egalitarian beliefs (i.e., who have low explicit prejudice), but who have implicit prejudice (termed aversive racists), may have particularly poor interactions with their patients. Notably, research shows that many healthcare providers, including physicians, tend to have low explicit bias, but nonetheless hold implicit biases. In interactions with a minority group member, people who are aversive racists tend to send mixed messages—while their low explicit prejudice tends to express itself in overt behavior (e.g., positive verbal content), their high implicit prejudice tends to “leak out” in their nonverbal behavior (e.g., less eye contact, distancing nonverbal behaviors). Since the minority interaction partner attends to the nonverbal cues in making judgments about the quality of the interaction, the minority person tends to have more negative ratings of the interaction and of their interaction partner. In fact, a study by Penner et al. (2010) found that Black patients even had more positive interactions with physicians who were high in both explicit and implicit bias than physicians who had low explicit but high implicit bias (i.e., aversive racists). Taken together, the work on explicit and implicit bias suggests that the conscious (explicit) and the unconscious (implicit) bias held by clinicians interact to influence clinical decision making, the quality of clinicians' interactions with patients, and patients' perceptions of bias.

Failures to Show a Link Between Stereotyping/Prejudice and Clinical Recommendations

While several studies have documented that stereotyping and prejudice influence physicians' treatment of patients, some have failed to find a direct effect of biases on treatment recommendations. For example, while several studies find that clinicians explicitly and implicitly stereotype their overweight patients as less adherent to treatment, some of these studies find that the extent to which clinicians hold this noncompliance stereotype has no predictive influence on treatment recommendations. Further, a subset of studies documenting explicit and implicit bias toward Black, Hispanic, and mentally ill patients failed to find effects of these biases on clinical decision making including physicians' treatment recommendations. This literature that documents bias but shows no impact of bias on treatment suggests that further study is needed to understand the specific conditions under which bias may be predictive of clinical decision making, as well as the more nuanced impact of bias on healthcare.

For example, although several studies do not show a direct effect of race (or other low-power group status, e.g., mental illness) on clinical recommendations, several show that clinicians' perceptions of uncooperativeness and doubts about adherence alter treatment recommendations. Namely, they show that lower perceived adherence is predictive of lower likelihood to recommend treatment. These results suggest that differences in treatment are not necessarily directly influenced by race or other group status but that they may be indirectly influenced through stereotypes, including lower perceptions of cooperativeness and adherence.

In addition, in the studies that fail to show a link between stereotyping and prejudice and treatment recommendations, there is often a standard of care that is typically followed. With little ambiguity and thus less discretion, it might be difficult to capture bias. Further, authors

of some studies that fail to show a link between explicit or implicit bias and treatment recommendations note that participants might have been aware and/or were suspicious of the study's purposes. With greater attention paid to race as a variable of interest, participants may have been particularly sensitive to make sure that their minority patients were given treatment as opposed to not. In Green et al.'s (2007) study, for example, 67 physicians were excluded from the analyses because they expressed awareness of the nature of the study. Yet, when these 67 "aware" physicians were included in the results, Green et al. found an interaction effect. As unaware physicians' pro-White bias increased, their likelihood of recommending thrombolysis to Black patients decreased. In contrast, increase in pro-White bias among aware physicians was associated with more thrombolysis recommendations for Black patients.

When Are Stereotyping and Prejudice More Likely?

The extant literature suggests situations in which stereotyping and prejudice are more likely to bias clinical decisions. First, situations marked with ambiguity are more likely to elicit bias. Research shows that in cases with clinical uncertainty, where a medical procedure is discretionary, or treatment guidelines are not well defined, the likelihood of clinician bias is greater. In addition, previous research shows that being under high cognitive load makes people, including physicians, more likely to behave according to stereotypes and prejudice. Similarly, when people are in time-pressured situations, which necessitate that they simultaneously attend to numerous tasks, they tend to be more likely to engage in stereotyping and to act according to their biases. Unfortunately, the medical context, which is often one marked by limited time and high cognitive load, may be especially likely to elicit bias.

Reducing the Occurrence and Impact of Stereotyping and Prejudice in Healthcare

To ameliorate the impact of stereotyping and prejudice in healthcare, the extant literature suggests several solutions. First, hiring a more diverse set of physicians may alleviate some of the observed disparities. Previous research finds that racially concordant medical interactions (those in which the physician and patient are the same race) are marked with less perceived discrimination, greater trust, greater satisfaction, and more effective communication (e.g., Cooper et al., 2003). Hence, increasing racial minority patients' access to racially concordant physicians may help to produce more productive medical visits for racial minority patients. The literature also suggests that workshops may help by making physicians, nurses, and medical students aware of their implicit bias and how it may influence their clinical decision making and their interactions with patients. Finally, bias-reduction strategies that have been successful in laboratory and some field settings may also be effective in the context of healthcare to reduce prejudice and stereotyping. These include taking the perspective of the minority patient (i.e., perspective taking), perceiving the patient as an individual as opposed to a member of their minority group (i.e., individuation), fostering a sense of common identity between the provider and the patient (i.e., common identity), and considering the ways in which the patient does not fit with stereotypes of his or her minority group (i.e., counterstereotyping). Individuation and perspective taking in particular have been shown to reduce biases in medical treatment, including pain treatment (see Chapman et al., 2013). Specifically, this research finds that inducing perspective taking increases physicians' empathy for their minority patients,

improving their ability to assess their patients' pain and, consequently, increase the provision of treatment. Importantly, these bias-reduction strategies may have a place in medicine to alleviate the impact of prejudice and stereotyping in healthcare.

Suggestions for Future Research

There are several directions for future research that will enhance our understanding of stereotyping and prejudice in healthcare. For example, while some bias-reduction strategies have been shown to be effective, further research is needed to understand how, and under what circumstances, these known strategies may be useful specifically in the healthcare context. In addition, given that some literature has failed to find a link between stereotyping and prejudice and clinical recommendations, further research is needed to determine the specific conditions under which stereotyping and prejudice will influence providers' recommendations and interactions with patients and the instances in which it will not.

Future research may also examine the role of clinicians' expectations on patient health outcomes. For example, if a clinician has low expectations (based on existing prejudice and stereotypes) that a patient will commit to therapy or treatment recommendations, some research suggests that they may be less likely to counsel the patient or to suggest a more challenging, yet perhaps more effective, course of treatment. As a result, the patient may experience worse health outcomes. Future research should more thoroughly examine the extent to which these expectations influence interactions with patients and how they influence the type and quality of treatment recommendations.

Further, research has focused more on the existence of stereotyping and prejudice among clinicians, but less on examining patients' own lived experience of stereotyping and prejudice. Not only would it be beneficial to obtain a qualitative understanding of these experiences, but it would also be beneficial to understand the cumulative effect of these experiences on patients, how these experiences may influence patients' perception of and participation in healthcare, and how these experiences impact patients' subsequent interactions with clinicians. Given that a great deal of work examines stereotyping and prejudice in short-term studies, future work may also include more long-term studies that examine how negative interactions with clinicians, and the healthcare system more generally, may influence patients' subsequent participation in healthcare.

Importantly, a more thorough investigation of patient-provider communication is warranted. Medical decisions are not made in a vacuum—rather, they derive from an interaction between the clinician and the patient. Future work may investigate how the nuances of these interactions shape the patients' attitudes and behavior toward the physician and how these interactions may influence the physician's clinical decision making. Research that investigates the more nuanced and reciprocal nature of patient-provider interaction (e.g., using non-obtrusive observation methods such as the Electronically Activated Recorder; Mehl & Robbins, 2012) may be particularly effective at understanding prejudice and stereotyping in the dynamic, and multifaceted, context of healthcare.

Author Biography

Dr. Elizabeth S. Focella is an assistant professor of psychology at the University of Wisconsin Oshkosh. Her research examines attitudes and behavior change, chiefly in the context of health

promotion. One major focus of her research examines prejudice and prejudice reduction, particularly in healthcare. Another line of her work, in cognitive dissonance, examines how people are motivated to change when they are made aware of the discrepancy between their pro-health attitudes and their unhealthy behavior.

References

- Bean, M. G., Focella, E. S., Covarrubias, R., Stone, J., Moskowitz, G. B., & Badger, T. A. (2014). Documenting nursing and medical students' stereotypes about Hispanic and American Indian patients. *Journal of Health Disparities Research and Practice*, 7(3), 14–22. Retrieved from <http://digitalscholarship.unlv.edu/jhdrp/vol7/iss4/2>
- Bean, M. G., Stone, J., Moskowitz, G. B., Badger, T. A., & Focella, E. S. (2013). Evidence of nonconscious stereotyping of Hispanic patients by nursing and medical students. *Nursing Research*, 62(5), 362–367. doi:10.1097/NNR.0b013e31829e02ec
- Bird, S. T., Bogart, L. M., & Delahanty, D. L. (2004). Health-related correlates of perceived discrimination in HIV care. *AIDS Patient Care and STDs*, 18(1), 19–26. doi:10.1089/108729104322740884
- Brener, L., Hippel, W. V., Kippax, S., & Preacher, K. J. (2010). The role of physician and nurse attitudes in the health care of injecting drug users. *Substance Use & Misuse*, 45(7-8), 1007–1018. doi:10.3109/10826081003659543
- Chapman, E. N., Kaatz, A., & Carnes, M. (2013). Physicians and implicit bias: How doctors may unwittingly perpetuate healthcare disparities. *Journal of General Internal Medicine*, 28(11), 1504–1510. doi:10.1007/s11606-013-2441-1
- Cooper, L. A., Roter, D. L., Carson, K. A., Beach, M. C., Sabin, J. A., Greenwald, A. G., & Inui, T. S. (2012). The associations of clinicians' implicit attitudes about race with medical visit communication and patient ratings of interpersonal care. *American Journal of Public Health*, 102(5), 979–987. doi:10.2105/ajph.2011.300558
- Cooper, L. A., Roter, D. L., Johnson, R. L., Ford, D. E., Steinwachs, D. M., & Powe, N. R. (2003). Patient-centered communication, ratings of care, and concordance of patient and physician race. *Annals of internal medicine*, 139(11), 907–915.
- Dovidio, J. F., & Fiske, S. T. (2012). Under the radar: How unexamined biases in decision-making processes in clinical interactions can contribute to health care disparities. *American Journal of Public Health*, 102(5), 945–952. doi:10.2105/ajph.2011.300601
- Drwecki, B. B., Moore, C. F., Ward, S. E., & Prkachin, K. M. (2011). Reducing racial disparities in pain treatment: The role of empathy and perspective-taking. *Pain*, 152(5), 1001–1006. doi:10.1016/j.pain.2010.12.005
- Foglia, M. B., & Fredriksen-Goldsen, K. I. (2014). Health disparities among LGBT older adults and the role of nonconscious bias. *Hastings Center Report*, 44(s4), S40–S44. doi:10.1002/hast.369
- Geiger, H. J. (2003). Racial and ethnic disparities in diagnosis and treatment: A review of the evidence and a consideration of causes. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*, 40(10), 417–454. doi:10.5860/choice.40-5843
- Green, A. R., Carney, D. R., Pallin, D. J., Ngo, L. H., Raymond, K. L., Iezzoni, L. I., & Banaji, M. R. (2007). Implicit bias among physicians and its prediction of thrombolysis decisions for Black and White patients. *Journal of General Internal Medicine*, 22(9), 1231–1238. doi:10.1007/s11606-007-0258-5
- Mehl, M. R., & Robbins, M. L. (2012). Naturalistic observation sampling: The Electronically Activated Recorder (EAR). In M. R. Mehl & T. S. Conner (Eds.), *Handbook of research methods for studying daily life* (pp. 176–192). New York, NY: Guilford Press.
- Moskowitz, G. B., Stone, J., & Childs, A. (2012). Implicit stereotyping and medical decisions: Unconscious stereotype activation in practitioners' thoughts about African Americans. *American Journal of Public Health*, 102(5), 996–1001. doi:10.2105/AJPH.2011.300591

- Penner, L. A., Dovidio, J. F., West, T. V., Gaertner, S. L., Albrecht, T. L., Dailey, R. K., & Markova, T. (2010). Aversive racism and medical interactions with Black patients: A field study. *Journal of Experimental Social Psychology*, 46(2), 436–440. doi:10.1016/j.jesp.2009.11.004
- Peris, T. S., Teachman, B. A., & Nosek, B. A. (2008). Implicit and explicit stigma of mental illness: Links to clinical care. *The Journal of Nervous and Mental Disease*, 196(10), 752–760. doi:10.1097/nmd.0b013e3181879dfd
- Persky, S., & Eccleston, C. P. (2011). Medical student bias and care recommendations for an obese versus non-obese virtual patient. *International Journal of Obesity*, 35(5), 728–735. doi:10.1038/ijo.2010.173
- Sabin, J. A., & Greenwald, A. G. (2012). The influence of implicit bias on treatment recommendations for 4 common pediatric conditions: Pain, urinary tract infection, attention deficit hyperactivity disorder, and asthma. *American Journal of Public Health*, 102(5), 988–995. doi:10.2105/ajph.2011.300621
- van Ryn, M., & Burke, J. (2000). The effect of patient race and socio-economic status on physicians' perceptions of patients. *Social Science & Medicine*, 50(6), 813–828. doi:10.1016/s0277-9536(99)00338-x

Suggested Reading

- Dovidio, J. F., & Fiske, S. T. (2012). Under the radar: How unexamined biases in decision-making processes in clinical interactions can contribute to health care disparities. *American Journal of Public Health*, 102(5), 945–952. doi:10.2105/ajph.2011.300601
- Nelson, A. (2002). Unequal treatment: confronting racial and ethnic disparities in health care. *Journal of the National Medical Association*, 94(8), 666–668.
- Shavers, V. L., Fagan, P., Jones, D., Klein, W. M., Boyington, J., Moten, C., & Rorie, E. (2012). The state of research on racial/ethnic discrimination in the receipt of health care. *American Journal of Public Health*, 102(5), 953–966. doi:10.2105/AJPH.2012.300773

The Prototype-Willingness Model

Frederick X. Gibbons¹, Michelle L. Stock²,
and Meg Gerrard¹

¹*Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA*

²*Department of Psychological and Brain Sciences, George Washington University,
Washington, DC, USA*

Basic Components

The prototype-willingness model (PWM) (Gerrard, Gibbons, Houlihan, Stock, & Pomery, 2008; Gibbons, Gerrard, & Lane, 2003; Gibbons, Stock, Gerrard, & Finneran, 2015) was created with a goal of improving the predictive and explanatory power of existing health behavior theories but with a specific target in mind: health *risk* behavior among *adolescents*. It is a modified dual-process model. Like other dual-process theories, the PWM maintains that health decision making involves two kinds of information processing. The two types are operative in the two pathways to health behavior described in the model.

The reasoned pathway involves analytic processing. It is the deliberative route to behavior reflecting the fact that some risky behaviors are the result of a consideration of options and expected outcomes associated with those behaviors. The proximal antecedent in this path is behavioral intention, which is defined as an intention (plan) to engage in a particular behavior. *The social reaction path* reflects a belief that certain behaviors, especially among adolescents, are more reactive than reasoned—that is, a response to a set of circumstances that may not have been sought or even anticipated. This path involves more heuristic processing: Deliberation is truncated, especially consideration of consequences; affect has more impact, and cognition less impact; images are influential; and heuristics play an important role. Both pathways include attitudes and subjective norms as distal antecedents; the social reaction path, however, also includes two constructs that are unique to the model, willingness and prototypes.

Behavioral willingness is defined as an openness to risk opportunity. It captures the reactive aspect of behavior, which is considerable for adolescents. It differs from an intention—it is not a plan of action or a goal state. Very few people plan on driving under the influence of drugs, for example, but some are willing to do so. Whether the behavior happens, then, is largely

dependent on whether the risk opportunity occurs (Gibbons et al., 2004). Willingness is influenced by affect and social influence (Gibbons et al., 2004), as well as heuristics (e.g., prototypes). Outcomes associated with the social reaction path typically have included risky health behaviors. However, there is no theoretical reason why the behavior has to be negative; as long as the behavior has a reactive component and an associated image, the PWM is potentially appropriate.

Assessment of willingness includes a hypothetical scenario; for example, “Imagine you’re at a party sometime in the next month and there are some drugs there you can have if you want. We’re not suggesting you would ever be in this kind of situation, but try to imagine it happening.” Next, the respondent is asked how *willing* she/he would be under these circumstances to do each of several behaviors that increase in risk (e.g., “try some ... use enough to get high ... take some to use later”). Responses are aggregated. A basic assumption behind the construct of willingness is that many adolescents are curious about risky behaviors. Peer pressure can be important, but there is more to substance use or risky sex than an inability to “just say no”; willingness is different from resistance efficacy. When individuals are asked about their willingness, various heuristics are instantiated. This happens quickly, but it does require some thought. Thus, the social reaction pathway—and this is true for much health decision making—relies heavily on heuristics, but it is a conscious more than an unconscious process, and it is seldom automatic.

Prototypes are the images that individuals have of the type of person who engages in a behavior. They include two primary dimensions: favorability and similarity. The most common method of favorability assessment involves adjective descriptors. For example, wording for adolescents (age ~12–14) would be:

We want to ask about your images of people who do different behaviors. We’re not talking about any particular person, and we’re not saying these people are all alike. We want to know what you think these people are like. Please think about the type of person your age who _____. How much do you think each of these adjectives describes that type of person?

This prompt is followed by a list of adjectives, preferably a mix of positive and negative (e.g., popular, selfish, considerate). These prototype (and willingness) items have consistently produced valid and reliable indices, even among children as young as 8 (Andrews, Hampson, Barckley, Gerrard, & Gibbons, 2008).

Research: Age and Experience

Two recent meta-analyses have reviewed some of the studies prompted by the PWM. One provided an analysis of the general model (Todd, Kothe, Mullan, & Monds, 2016), whereas the second examined a more specific issue: prototype similarity versus favorability (van Lettow, de Vries, Burdorf, & van Empelen, 2016). Both analyses reached essentially the same two general conclusions: PWM constructs, specifically prototypes and willingness, add significantly to the prediction of health behaviors, and the model has utility and potential in terms of interventions. Below are some issues raised in those meta-analyses.

Adolescents

A number of studies have indicated that willingness is usually a better predictor than intention among adolescents for risky behaviors, including substance use (e.g., cigarettes and drinking).

This varies with age, however. Two prospective studies examined the relative predictive power of willingness versus intention annually from age 12 through 18 and found the anticipated pattern for smoking: controlling for previous behavior, willingness exceeded intention through age 16, at which point the smoking became more intentional for the next 2 years (Pomery, Gibbons, Reis-Bergan, & Gerrard, 2009). From then on, smoking was mostly habitual, and neither willingness nor intention predicted significant variance beyond previous behavior. However, another analysis in the same paper with nonsmokers age 12–19 showed that *initiation* of smoking was also predicted better by willingness up to age 19. Consistent with these results, Todd et al. (2016) found that age moderated the prototype-to-willingness and willingness-to-behavior relations, leading them to conclude that the PWM works better for adolescents than adults.

Adults

Some behaviors are risky or rare enough that the image associated with them is pronounced and impactful; consequently, they are less likely than drinking or smoking to become intentional or habitual, even into adulthood. These behaviors include sexual “cheating,” drunk driving, and heavy drug use, among others. More generally, many risk behaviors are intentional up to a certain level, but people have their limits, and risk above that threshold often becomes willingness based. More PWM studies with adults are likely in the future.

Type of Behavior

Health Risk

Todd et al.’s list of behaviors examined in PWM studies included health promotion (e.g., exercise, vaccination) and health risk. The model did best in predicting substance use: Willingness accounted for 20% of the variance in behavior, whereas intention accounted for only 8%. As Todd et al. suggested, substance use is a risky behavior with a significant social reaction component. On the other hand, willingness explained less variance than intention in cigarette use, which has much less of a social component. This pattern varies considerably by age, however—much more reactive in early adolescence and then more reasoned in late adolescence and early adulthood (Pomery et al., 2009).

Health Promotion

Gerrard et al. (2002) examined the prospective effects of a healthy prototype, the adolescent *nondrinker*, and the more common, unhealthy prototype, the adolescent drinker, on adolescents’ willingness to drink and their actual consumption. As expected, the drinker image predicted drinking indirectly, via willingness to drink, and the abstainer image predicted directly. However, there was also an indirect path from the abstainer image to drinking through an image contemplation measure (“How often have you thought about this type of person?”). These results are consistent with a dual-process account. First, nondrinkers had a favorable nondrinker image, and that image directly predicted their abstinence, suggesting that the nondrinker image was a goal for them. Second, a favorable nondrinker image was associated with more image contemplation, which also suggests more reasoned processing; in contrast, the drinker image involved heuristic processing. Other healthy images that have been examined include condom users, exercisers, healthy eaters, sun protectors, and (substance) abstainers.

Comparing Willingness and Intention

The distinction between willingness and intention is central to the PWM and to its status as a dual-process model. Clearly, many health behaviors have a reasoned component to them, and although the intention–behavior correlation is often less than predicted, it is reliable. For this reason, the PWM maintains that for many risky behaviors, analyses that include both willingness and intention are more likely to effectively predict behavior than intention alone. By the same token, for many health-related behaviors, willingness is not relevant, especially among adults (e.g., drinking or smoking).

Antecedents

In spite of their overlap, intention and willingness have distinct antecedents. In survey research, for example, Gibbons et al. (2004) found that parents' substance use predicted their children's intentions and especially their behavioral *expectations* to use substances (e.g., "My parents smoke, I probably will also"), but it did not predict willingness to use. Social influence (i.e., perception of friends' use) and risk images, on the other hand, predicted both willingness and intention, but the relations were stronger for willingness. Other studies have found that endorsement of the heuristic "If it's illegal, it must be effective" predicted willingness but not intention to use performance-enhancing drugs and also that positive and negative alcohol portrayals by actors in movies affected adolescents' willingness to drink but not their intentions (Gibbons et al., 2016).

Manipulations

Several studies have also demonstrated differences in how intention and willingness are affected by experimental manipulations. Two such studies examined a behavior with a significant willingness–intention distinction: casual sex. One study found that men who initially reported no intention to engage in casual sex increased in willingness (but not intention) to engage in the behavior after exposure to tempting photos (women posing provocatively in bikinis; Roberts, Gibbons, Kingsbury, & Gerrard, 2013). A second study replicated this effect for "cheating" using supraliminal primes (provocative photos interspersed with control photos). This study found that those in committed relationships reported lower willingness to engage in casual sex than those who were single, as expected, but these committed men and women had significantly higher willingness than intention. Apparently, for some people with no intention to cheat but some willingness, whether that willingness turns into casual sex behavior may depend on "risk opportunity."

Prototypes

Dimensions

Both favorability and similarity are important aspects of risk images (prototypes); when both are high, willingness is most likely. In their meta-analysis, van Lettow et al. (2016) found that in a majority of studies that assessed both prototype similarity and favorability, similarity was a stronger predictor for willingness. However, studies that also included a similarity by favorability interaction term showed that the interaction was a stronger predictor than either dimension by itself. In fact, the "diagonals"—those who are high in similarity but low in favorability, or vice-versa—are an interesting group. Presumably, those in the latter category are considering

starting the behavior (“contemplators”). On the other hand, those in the high-similarity/low-favorability category clearly do not approve of the behavior and, presumably, are either concerned they may start engaging or are already engaging and want to stop.

Relations With Intention and Willingness

Some studies have found evidence of a prototype-to-intention path. Although rare when both willingness and intention are included in analyses, this relation is not inconsistent with the model. Health behavior often is goal based, and one goal can be image acquisition. Exercising is an obvious example of a healthy behavior with an image that can act as a goal. But (normatively) negative images can also be goals: admission into a desired social group, “dirts” or “druggies,” for example. In this case, the group has a negative image that can be attractive (for some) and therefore increase the image-to-intention relationship. Finally, prototype influence is moderated by social comparison tendencies. Those who compare a lot are more responsive to images, so the prototype/willingness relationship is stronger for them.

Subjective Norms and Attitudes

Norms

Some form of subjective norm construct can be found in most health behavior models, sometimes divided into *descriptive* and *injunctive* (people’s perceptions of what others are doing versus what others want them to do). We have found that peer pressure (injunctive norms) is often not a significant factor in terms of health risk willingness, whereas perceived prevalence (descriptive norms) usually is. The model describes paths from subjective norms to both intention and willingness, but that may not be true for both types of norms. Engaging in a behavior because you believe others want or expect you to do it requires some thought and reasoning (what do they want? will this please them?), whereas doing a behavior mostly for conformity reasons (i.e., “everyone’s doing it”) requires less reasoning and more reacting. Future research examining whether injunctive norms lead to intentions whereas descriptive norms lead to willingness would be informative.

Attitudes

A similar dual-process argument could be made for attitudes. Using perceived risk as an example, perceptions of danger (e.g., “driving after drinking is dangerous”) are usually higher than perceptions of personal vulnerability, which often reflect some optimistic bias (e.g., “I could have several drinks and drive home; I’ve done it before”). Perceived danger should be more strongly related to intention, and personal vulnerability more to willingness. Similarly, attitudes include both cognitive and affective components; the latter should be more strongly related to willingness than the former.

Interventions: Laboratory Studies

Perceived Risk

The PWM has been the theoretical basis for several interventions and preventive interventions as well as laboratory analogue studies examining the utility of components of the model, mostly

prototypes and willingness, for possible future interventions. One recent example of this involved the “absent-exempt” heuristic, which is a perception that some people develop if they have been engaging in a risk behavior for some time and nothing bad has happened to them that they may be somehow immune to the consequences. A series of studies showed that absent-exempt thinking is enhanced among high-risk individuals by (downward) social comparison with a low-risk individual who *has* experienced the associated negative consequence (e.g., an STD), even though they had engaged in very little risk behavior (Stock, Gibbons, Beekman, & Gerrard, 2015). Most important, this increase in absent-exempt thinking was accompanied by an *increase* in willingness to have casual sex among high-risk individuals. This finding has intervention implications: PSAs that include the theme “It only takes once!” are common (especially for sexual behavior), which creates a real possibility of iatrogenic effects—increases in risk-taking after the intervention, due to absent thinking, among those already at high risk.

Dual Processing

There was also evidence of dual processing in these studies. Risk willingness increased significantly in all three studies, but risk intention did not change. Also, when reasoned processing was instantiated before the comparison opportunity, it eliminated the absent-exempt effect. Most interesting, however, was the fact that in addition to reporting lower perceived vulnerability and higher willingness (but not intention), high-risk participants also reported significantly more intention to get tested for an STD. In other words, the absent-exempt heuristic was associated with an increase in risk willingness *and* an increase in health promotion intentions—an example of reasoned action accompanying social reaction in the same individuals simultaneously.

Manipulating Prototypes

Safe Sex Images

Many studies have manipulated components of the PWM to test prototype malleability and the impact of these manipulations on willingness and behavior. They have addressed a range of behaviors besides substance use and UV exposure, including exercise, diet, sleeping, safe sex, and nonprescription drug use. Most have focused on risk prototypes, but a sizable number were prompted by Gerrard et al. (2002) in showing that images of people who engage in healthy behavior can also motivate change. In the first prototype manipulation study, college students read a bogus newspaper article reporting the results of a personality survey, indicating that the typical person who does not use condoms (experimental group) or does not vote (control) is “more selfish” and “less responsible” than those who do (Blanton et al., 2001). As expected, students in the condom condition reported less willingness to have unprotected sex than did those in the voter condition.

Prototype Visualization

Another prototype paradigm uses a visualization strategy. Ouellette, Hessling, Gibbons, Reis-Bergan, and Gerrard (2005) had participants write about typical exercisers and non-exercisers. As expected, exerciser prototypes were much more favorable; more important, a follow-up revealed that those in the exerciser condition significantly increased their exercising, relative to those in the non-exerciser condition. An extension of this study based on terror management

theory employed a mortality salience induction to examine *why* prototypes affect behavior and found that making mortality salient, combined with contemplating an exerciser prototype, increased the extent to which participants said they valued exercising.

Interventions

UV Protection

A series of UV protection lab studies has also successfully altered prototypes. These studies employed a UV filter camera that produces a facial photo that graphically depicts existing skin damage due to previous UV exposure. The prediction was that the photos would make images of tanning and tanners more negative. Thus, the same motivation behind the risk behavior (appearance) was used to motivate abandoning it. One study showed that the photographs reduced prototype favorability and tanning willingness while increasing perceived vulnerability to skin damage; these changes mediated subsequent declines in tanning booth use among those who received the photos (Gibbons et al., 2005). A more recent study examined the impact of the UV photos from a dual-processing perspective by asking women to focus on either their thoughts or their feelings when they viewed the UV photos in order to encourage analytic or heuristic processing about the risk (Walsh, Stock, Peterson, & Gerrard, 2014). As expected, older women in the cognitive focus condition had lower sun exposure willingness and higher perceived skin cancer vulnerability than those in the affective focus group, suggesting that altering type of processing regarding risk can lead to a reduction in risk behavior. Another UV intervention focused on a high-risk population: road maintenance workers (Stock et al., 2009). Participants were shown UV facial photographs and then watched an educational video on UV risk and protection. There were two significant paths from the intervention to the outcome measures (changes in self-reported UV protection and tanning assessed objectively with a spectrophotometer): a direct path *and* an indirect path mediated by changes in risk cognitions suggested by the PWM. Thus, the most effective strategy for this high-risk population included both reasoned (educational) and heuristic components.

Strong African American Families (SAAF)

Work on family process by Gene Brody and colleagues led to the development of the SAAF Program, which is a family-centered alcohol preventive intervention based partly on the PWM. It includes two components. One component, which involved the parents, targeted the reasoned path by promoting parent–child communication. The second, more heuristic, arm was directed at the children. It was designed to make their drinker images more negative and educate them on the differences between planned and reactive behavior, thereby reducing their willingness to drink. The intervention was successful in decreasing the favorability of the children’s drinker images and their intention and willingness to drink while increasing reports of parent–child communication. The desired effect on alcohol consumption at a 24-month follow-up also was significant (Gerrard et al., 2006).

Dual Processing

The SAAF intervention also demonstrated the efficacy of combining a heuristic *and* an analytic approach to shaping adolescents’ decision making. In the reasoned path, the effect of changes in parenting on adolescents’ drinking was mediated by changes in their intention; in

the social reaction path, the effect of changes in the children's prototypes on their drinking were mediated by changes in willingness. The social reaction path was stronger, mostly because the willingness/behavior relationship was stronger than the intention-behavior relationship. Importantly, however, a structural equation model that included both the reasoned *and* the social reaction paths provided a significantly better fit of the data than models with either path alone, once again illustrating the utility of a dual-processing approach to intervention.

Smoking Prevention

Click City is an online prevention program that targets adolescents' prototypes and willingness and intentions to use tobacco. One study demonstrated that the intervention was most efficacious for children who were most at risk, that is, those who had tried smoking at baseline and/or had a family member who smoked. A follow-up replicated these results: Students in the program reported smaller increases in the favorability of their smoker prototypes and their willingness and intentions to smoke than those in the control condition (Andrews et al., 2014).

Future Research Directions

Smoker Prototypes and Smoking Cessation

The first PWM prototype study examined the relation between smoker images and success at smoking cessation (Gibbons, Gerrard, Lando, & McGovern, 1991). Results indicated that smokers' images of other smokers became more negative during the course of their quit group, and the more negative they became, the more likely the smokers were to quit. A subsequent study found that a negative smoker image was a better predictor of quitting than was a positive *former smoker* image (Gibbons & Eggleston, 1996). Along similar lines, overweight images have been shown to be stronger predictors of weight loss efforts than are thin images, and negative smoker prototypes are more motivating for nonsmokers to avoid smoking than positive smoker prototypes are for nonsmokers to start. These results suggest the desire to stop being, or avoid becoming, a negative exemplar may be a stronger motivator than the desire to become a positive (healthy) exemplar. More such cessation studies are needed.

Willingness and Prototype Development

Finally, examining factors that affect the development of images and willingness would have value from both an applied and theoretical perspective. We know that risk images are influential for children as young as 8, and we know that the media (especially movies) are an important source for these images (Gibbons et al., 2010). We also know that by age 13 or 14, adolescents can articulate the difference between intention and willingness. Further examination of this developmental process is called for; the same is true for the study of factors that can effect change in willingness and prototypes.

Conclusion

Several conclusions can be drawn from the PWM literature. First, willingness and intention are clearly related, but there is considerable evidence suggesting that they are different constructs,

with discriminable antecedents and consequences. Second, analogue studies have demonstrated that risk and nonrisk prototypes are malleable and that reducing risk prototype favorability and/or increasing nonrisk prototype favorability can reduce risk willingness and actual risk behavior. Finally, there is evidence of increasing acceptance of the dual-processing perspective upon which the PWM is based, along with enthusiasm for the utility of this perspective for explaining, predicting, and changing health behavior, especially among adolescents.

Author Biographies

Frederick X. Gibbons is a health-social psychologist and a professor in the Department of Psychological Sciences at the University of Connecticut. He received his PhD in psychology from the University of Texas. His research interests focus on the application of social psychology theory and principles to the study of health behavior. This research has examined disparities in health status and factors associated with onset and cessation of health risk behavior. It has included survey and lab (experimental) studies, along with interventions and preventive interventions.

Michelle L. Stock is an associate professor in the Department of Psychology at The George Washington University. She received her PhD in psychology from Iowa State University. Her program of research includes a focus using the prototype-willingness model to provide a framework for understanding cognitive (heuristic and reasoned), affective, and situational factors that influence risky health decisions.

Meg Gerrard is a research professor in the Department of Psychological Sciences at the University of Connecticut. She received her PhD in psychology from the University of Texas. Throughout her career, her research has focused on the prediction of adolescent risk behaviors (i.e., alcohol and substance use, risky sexual behaviors, and smoking) and the prevention of these behaviors. She and her colleague (Rick Gibbons) developed the prototype-willingness model of adolescent health behavior in an effort to explain and explore non-intentional adolescent risk behavior.

References

- Andrews, J. A., Gordon, J. S., Hampson, S., Gunn, B., Christiansen, S., & Slovic, P. (2014). Long-term efficacy of Click City® tobacco: A school-based tobacco prevention program. *Nicotine & Tobacco Research, 16*, 33–41. doi:10.1093/ntr/ntt106
- Andrews, J. A., Hampson, S. E., Barckley, M., Gerrard, M., & Gibbons, F. X. (2008). The effect of early cognitions on cigarette and alcohol use during adolescence. *Psychology of Addictive Behaviors, 22*, 96–106. doi:10.1037/0893-164X.22.1.96
- Blanton, H., Vanden-Eijnden, R., Buunk, B. P., Gibbons, F. X., Gerrard, M., & Bakker, A. (2001). Accentuate the negative: Social images in the prediction and promotion of condom use. *Journal of Applied Social Psychology, 31*, 274–295. doi:10.1111/j.1559-1816.2001.tb00197.x
- Gerrard, M., Gibbons, F. X., Brody, G. H., Murry, V. M., Cleveland, M. J., & Wills, T. A. (2006). A theory-based dual-focus alcohol intervention for preadolescents: The Strong African American Families Program. *Psychology of Addictive Behaviors, 20*, 185–195. doi:10.1037/0893-164X.20.2.185
- Gerrard, M., Gibbons, F. X., Houlihan, A. E., Stock, M. L., & Pomery, E. A. (2008). A dual-process approach to health risk decision making: The prototype-willingness model. *Developmental Review, 28*, 29–61. doi:10.1016/j.dr.2007.10.001

- Gerrard, M., Gibbons, F. X., Reis-Bergan, M., Trudeau, L., VandeLune, L., & Buunk, B. (2002). Inhibitory effects of drinker and nondrinker prototypes on adolescent alcohol consumption. *Health Psychology, 21*, 601–609. doi:10.1037/0278-6133.21.6.601
- Gibbons, F. X., & Eggleston, T. J. (1996). Smoker networks and the ‘typical smoker’: A prospective analysis of smoking cessation. *Health Psychology, 15*, 469–477. doi:10.1037/0278-6133.15.6.469
- Gibbons, F. X., Gerrard, M., Lando, H. A., & McGovern, P. G. (1991). Social comparison and smoking cessation: The role of the “typical smoker”. *Journal of Experimental Social Psychology, 27*, 239–258. doi:10.1016/0022-1031(91)90014-W
- Gibbons, F. X., Gerrard, M., & Lane, D. J. (2003). A social-reaction model of adolescent health risk. In J. M. Suls & K. A. Wallston (Eds.), *Social psychological foundations of health and illness* (pp. 107–136). Oxford, England: Blackwell.
- Gibbons, F. X., Gerrard, M., Lane, D. J., Mahler, H. I. M., & Kulik, J. A. (2005). Using UV photography to reduce use of tanning booths: A test of cognitive mediation. *Health Psychology, 24*, 358–363. doi:10.1037/0278-6133.24.4.358
- Gibbons, F. X., Gerrard, M., VandeLune, L. S., Wills, T. A., Brody, G., & Conger, R. D. (2004). Context and cognition: Environmental risk, social influence, and adolescent substance use. *Personality and Social Psychology Bulletin, 30*, 1048–1061. doi:10.1177/0146167204264788
- Gibbons, F. X., Kingsbury, J. H., Wills, T. A., Finneran, S. D., Dal Cin, S., & Gerrard, M. (2016). Impulsivity moderates the effects of movie alcohol portrayals on adolescents’ willingness to drink. *Psychology of Addictive Behaviors, 30*(3), 325–334.
- Gibbons, F. X., Pomery, E. A., Gerrard, M., Sargent, J. D., Weng, C. Y., Wills, T. A., ... Yeh, H. C. (2010). Media as social influence: Racial differences in the effects of peers and media on adolescent alcohol cognitions and consumption. *Psychology of Addictive Behaviors, 24*, 649–659. doi:10.1037/a0020768
- Gibbons, F. X., Stock, M. L., Gerrard, M., & Finneran, S. (2015). The prototype-willingness model. In M. Conner & P. Norman (Eds.), *Predicting and changing health behaviour: Research and practice with social cognition models* (3rd ed., pp. 189–224). Berkshire, UK: Open University Press.
- Ouellette, J. A., Hessling, R., Gibbons, F. X., Reis-Bergan, M., & Gerrard, M. (2005). Using images to increase exercise behavior: Prototypes vs. possible selves. *Personality and Social Psychology Bulletin, 31*, 610–620. doi:10.1177/0146167204271589
- Pomery, E. A., Gibbons, F. X., Reis-Bergan, M., & Gerrard, M. (2009). From willingness to intention: Experience moderates the shift from reactive to reasoned behavior. *Personality and Social Psychology Bulletin, 35*, 894–908. doi:10.1177/0146167209335166
- Roberts, M. E., Gibbons, F. X., Kingsbury, J. H., & Gerrard, M. (2013). Not intending but somewhat willing: The influence of visual primes on risky sex decisions. *British Journal of Health Psychology, 19*, 553–565. doi:10.1111/bjhp.12055
- Stock, M. L., Gerrard, M., Gibbons, F. X., Dykstra, J. L., Mahler, H. I. M., Walsh, L. A., & Kulik, J. A. (2009). Sun protection intervention for highway workers: Long-term efficacy of UV photography and skin cancer information on men’s protective cognitions and behavior. *Annals of Behavioral Medicine, 38*, 225–236. doi:10.1007/s12160-009-9151-2
- Stock, M. L., Gibbons, F. X., Beekman, J., & Gerrard, M. (2015). It only takes once: The absent-exempt heuristic and reactions to comparison-based sexual risk information. *Journal of Personality and Social Psychology, 109*, 35–52. doi:10.1037/a0039277
- Todd, J., Kothe, E., Mullan, B., & Monds, L. (2016). Reasoned versus reactive prediction of behavior: A meta-analysis of the prototype willingness model. *Health Psychology Review, 10*(1), 1–24. doi:10.1080/17437199.2014.922895
- van Lettow, B., de Vries, H., Burdorf, A., & van Empelen, P. (2016). Quantifying the strength of the associations of prototype perceptions with behaviour, behavioural willingness and intentions: A meta-analysis. *Health Psychology Review, 10*(1), 25–43. doi:10.1080/17437199.2014.941997
- Walsh, L. A., Stock, M. L., Peterson, L., & Gerrard, M. (2014). Women’s sun protection cognitions in response to UV photography: The role of age, cognition, and affect. *Journal of Behavioral Medicine, 37*, 553–563. doi:10.1007/s10865-013-9512-y

Suggested Reading

- Gerrard, M., Gibbons, F. X., Brody, G. H., Murry, V. M., Cleveland, M. J., & Wills, T. A. (2006). A theory-based dual-focus alcohol intervention for preadolescents: The Strong African American Families Program. *Psychology of Addictive Behaviors*, *20*, 185–195. doi:10.1037/0893-164X.20.2.185
- Gibbons, F. X., Stock, M. L., Gerrard, M., & Finneran, S. (2015). The prototype-willingness model. In M. Conner & P. Norman (Eds.), *Predicting and changing health behaviour: Research and practice with social cognition models* (3rd ed., pp. 189–224). Berkshire, UK: Open University Press.

Psychosocial Factors in Coronary Heart Disease

Elizabeth J. Vella

Department of Psychology, University of Southern Maine, Portland, ME, USA

Cardiovascular disease represents the leading cause of death globally, which includes mortality due to stroke and coronary heart disease (CHD); of these two forms of cardiovascular disease, CHD accounts for more deaths annually (World Health Organization, 2015). The primary features of CHD include plaque development in the coronary arteries (atherosclerosis), heart attack (myocardial infarct), and acute chest pain (angina; Labarthe, 1998). The traditional risk factors for CHD include age, obesity, high cholesterol, high blood pressure, inactive lifestyle, smoking, excessive alcohol consumption, and family history of the disease (World Heart Federation, 2015). Epidemiologic evidence suggests that traditional risk factors of CHD may account for 58–75% of new cases (Beaglehole & Magnus, 2002). Other predictors of CHD may include stress-related psychosocial factors at a person level (e.g., dispositional hostility and depression) and/or environmental level (e.g., chronic work-related stress and lack of social support; Albus, 2010).

What Is a Psychosocial Risk Factor?

Simply stated, a psychosocial risk factor should be conceptualized as a psychological attribute that impacts social behaviors in a way that elevates likelihood of illness, such as emotion dispositions of anger that increase antagonistic social interactions, thereby leading to exacerbated stress levels and social isolation, eventually giving rise to disease onset. The impact of psychosocial risk factors on illness onset is often both direct (e.g., stress-induced elevations in markers of inflammation that hasten atherosclerotic plaque formation) and indirect (e.g., stress-induced changes in lifestyle behaviors that contribute to unhealthy diet, smoking, and excessive alcohol consumption as maladaptive means of coping). When studying psychosocial risk factors for CHD, researchers must measure and control for the presence of traditional risk factors in their

statistical models to effectively be able to ascertain that the psychosocial risk factors in question are predictive of CHD above and beyond the traditional risk factors aforementioned.

Early Research on the Type A Behavior Pattern and Trait Hostility

In the 1950s, cardiologists Friedman and Rosenman began to take notice that a pattern of personality characteristics seemed to typify their CHD patients. These physicians termed the personality style as the Type A behavior (TAB) pattern, characterized by a strong sense of time urgency, hard-driving competitiveness, ambitiousness, hostility, and tendencies to experience anger and become aggressive. They conducted a wide-scale assessment of TAB entitled the Western Collaborative Group Study (WCGS) to determine if a reliable relationship exists between this set of personality traits and the severity of coronary dysfunction; 3,154 men aged 39–59 were administered the structured interview to assess TAB and then followed for 8.5 years (Rosenman et al., 1975). Results indicated that Type A individuals were more than twice as likely to subsequently develop clinically significant CHD symptoms than Type B (essentially calm demeanor, even tempered) counterparts.

However, the Multiple Risk Factor Intervention Trial (MRFIT) called the TAB construct into question by failing to replicate WCGS results. Reliable differences in the administration and coding of the structured interview to assess TAB have been reported between WCGS and MRFIT, which may account for these discrepant results (Sherwitz & Brand, 1990). Further, the multifaceted nature of the Type A construct may increase the probability of inconsistencies in the literature. Importantly, subsequent analyses of data from WCGS and MRFIT indicated that the structured interview-derived subscale score entitled “potential for hostility,” an interactive style of expressive speech and experience of anger, was a better predictor of CHD in both datasets than the global Type A score (Dembroski, MacDougall, Costa, & Grandits, 1989; Hecker, Chesney, Black, & Frautschi, 1988). Therefore, the toxic elements of TAB appear to concern a particular interactive style separate from the qualities of ambitiousness, competition, and time urgency.

More recently, evidence suggests a reliable association between trait hostility/anger and CHD outcomes. For example, a meta-analytic report across 21 articles longitudinally examining 71,606 initially healthy participants and 18 articles longitudinally studying 8,120 CHD patients found that elevated ratings of anger/hostility were linked to an average 20% increase in disease risk, in terms of risk of eventual diagnosis amid initially healthy participants and risk of additional coronary events/CHD mortality for the patient samples (Chida & Steptoe, 2009). Interestingly, the risk profile was more pronounced amid the latter samples of participants already diagnosed with CHD, suggesting that the mere diagnosis and experience of CHD as a condition may promote bouts of anger and hostility in a way that contributes to poorer prognosis. Specifically, anger/hostility ratings predicted a 24% increased risk of additional coronary events/CHD mortality amid patients compared with a 19% increased risk of CHD diagnosis amid initially healthy participants.

Currently, the hostility construct remains a point of controversy as a risk factor for the development of CHD, yielding a mixture of results claiming significant and insignificant associations. A clear problem in the literature that may explain many of these discrepancies across studies concerns the multidimensionality of the hostility construct (cognitive mistrustfulness, emotional anger, and behavioral aggression). This problem is compounded by variability in measurement instruments: some assessment tools for measuring hostility are more reliably predictive of disease onset than others (e.g., cynical mistrustfulness and the experience of

anger versus anger expression through aggression), a quality that adds confusion to the medical science literature.

Depression

Clinical depression is diagnosed based upon criteria specified in the Diagnostic and Statistical Manual published by the American Psychological Association, including such symptoms as depressed mood, feelings of worthlessness, fatigue, changes in sleep patterns, and suicidal ideation. The relationship between clinical depression and CHD diagnosis/mortality is more reliable and robust than the associations apparent for hostility/TAB. For example, a recent meta-analysis across 30 prospective cohort studies including a total of 893,850 participants indicated that depression was associated with a 30% increased risk of CHD diagnosis (Gan et al., 2014). Similar to the hostility construct, the timeframe of depression onset has been found to be meaningfully related to CHD prognosis: patients who experienced recurrent–persistent depression pre-CHD diagnosis and maintained depression levels post-CHD diagnosis were 1.5–2 times more likely to be re-hospitalized for a coronary event or experience coronary mortality, respectively, than their non-depressed counterparts (Leung et al., 2012). These findings indicate that clinically depressed adults are at an elevated risk for CHD diagnosis and that they furthermore exhibit poorer CHD prognoses compounded by a higher mortality risk due to coronary complications.

Environmental Factors: Work Stress

A long-standing history of research has illustrated work-related stress to contribute to disease outcomes, ranging the full gamut from acute bouts of insomnia to chronic medical conditions, such as CHD (Ganster & Rosen, 2013). A variety of theoretical orientations have been offered to organize research assessments, including the job strain model, the effort–reward imbalance model, and the organizational injustice model (e.g., Kivimäki et al., 2006). The job strain model proposes the demand–control hypothesis that occupations characterized as high demand–low control represent a toxic combination in terms of giving rise to chronically elevated stress levels that degrade immune function and lead to adverse health outcomes. In short, this model stipulates that employees who are burdened with oftentimes unpredictable waves of high volume responsibilities (elevated demand) without appropriate decision latitude for managing the workload (poor control) are at risk for work-related illness and eventual burnout. Along similar lines, the effort–reward imbalance model stipulates that work environments whereby employees are disproportionately challenged relative to compensation and recognition are likewise at risk for stress-induced illness. Finally, the organizational injustice model argues that stress-induced illnesses may arise in workplaces whenever employees reliably report being treated unfairly by their supervisors. Each model grapples with different ways by which workers may experience heightened stress levels on a daily basis.

Evidence supports all three models of conceptualizing the work stress–CHD relationship, as indicated by a meta-analytic report of 14 prospective cohort studies involving 83,014 employees, whereby elevations in work stress predicted an average 50% increase in CHD diagnosis/mortality (Kivimäki et al., 2006). Although all three models were supported in this meta-analysis, the evidence was significantly stronger in support of the effort–reward imbalance and organizational injustice models relative to the job strain model, after controlling for

the variance pertaining to traditional CHD risk factors. Irrespective, there is a clear and strong pattern of evidence that elevated stress in the workplace predicts adverse coronary outcomes over time.

Environmental Factors: Social Support

Having the opportunity to discuss stressful life experiences with a trusted confidant can be helpful in reducing emotional strain and promoting problem-focused coping, envisioning potential resolutions to the circumstances underlying the challenge/threat in question. Social support resources elicit the tendency to perceive that a weight has been lifted from one's shoulders through self-disclosure, thereby enhancing the sensation of self-efficacy (ability to effectively handle the stressful situation) and reducing the maladaptive tendency of catastrophizing (rumination of difficulties that leads to perceived hopelessness—common when people feel overwhelmed by their life stressors and alone in their path of coping). If chronically elevated stress levels increases risk of CHD diagnosis/mortality, it stands to reason that stress-reducing social environments would confer protective benefit.

Sources of social support are typically defined in the health sciences literature in terms of being “functional” or “structural” in scope. Types of functional support include the following categories: emotional, informational (e.g., receiving advice), financial, appraisal (someone helping to evaluate a situation), and instrumental (e.g., receiving assistance in completing a project), whereas structural support pertains to the number of individuals in one's social network in terms of marital status, proximity of friends and family, community memberships, and the frequency of social contacts (e.g., number of times visiting with friends per month).

The pattern of evidence in both etiologic (initially healthy samples) and prognostic (samples of CHD patients) longitudinal studies has been significant for functional social support but insignificant for structural social support, whereby low functional support predicted elevated relative risk of heart attack amid initially healthy samples and, in particular, a 59% increased risk of mortality amid CHD patients after controlling for traditional risk factors in a meta-analysis (Barth, Schneider, & von Känel, 2010). These findings highlight the prominence of functional social support with regard to the quality of supportive social interactions, rather than the structural quantity of social interactions, when predicting health outcomes.

Environmental Factors: Socioeconomic Status

Typically assessed by annual income and/or years of education, socioeconomic status (SES) represents an environmental attribute worthy of consideration as a psychosocial risk factor for CHD. There are a variety of putative mechanisms that may connect low SES to CHD outcomes, such as indirect effects of inadequate resources to maintain a healthy lifestyle and direct effects of stress-induced influences on coronary dysfunction. For example, individuals of a lower class standing may consume a cheaper, highly processed diet conducive to the development of obesity, type 2 diabetes, hypertension, and elevated cholesterol. Likewise, the economic burden of poverty often compels individuals to work two to three jobs to make ends meet, thereby exacerbating perceptual stress levels as a function of workload and reducing time spent with family and friends—effectively degrading social support resources for stress management. Thus, lower SES tends to co-occur with elevated work stress and poor social support, eliciting potential synergistic effects on disease outcome, suggesting a need for

theoretical orientations designed to guide research investigations involving psychosocial risk factors for CHD morbidity and mortality.

Meta-analytic evidence involving 70 studies has indicated significant effects across income, occupation, and education whereby individuals in the lowest socioeconomic positions exhibited elevated risk of incident heart attack relative to their more affluent counterparts, with the effects most apparent in terms of income (a staggering 71% increase in relative risk of heart attack; Manrique-Garcia, Sidorchuk, Hallqvist, & Moraldi, 2011). Further, prospective evidence following 66,500 initially healthy adults across 8 years found low SES predicted CHD mortality after controlling for traditional risk factors. SES was measured in terms of three occupational classes (professional/managerial, skilled manual/non-manual workers, and semi-routine/unskilled workers) with each step down from professional/managerial associated with a 24% increase in CHD mortality (Lazzarino, Hamer, Stamatakis, & Steptoe, 2013). Moreover, a synergistic effect was observed whereby psychological distress augmented the adverse effects of low SES: the combination of low SES and high psychological distress was linked to a 33% increase in CHD mortality relative to high SES and low psychological distress counterparts (Lazzarino et al., 2013).

Piecing Together the Puzzle: Integrative Theoretical Frameworks

The evidence to date reveals that a variety of psychosocial risk factors predict elevated CHD morbidities and mortalities to a varying degree after controlling for the presence of traditional risk factors, including dispositional factors such as hostility and depression (e.g., Chida & Steptoe, 2009; Gan et al., 2014) and environmental factors such as occupational strain and low social support (e.g., Barth et al., 2010; Kivimäki et al., 2006). However, emergent findings increasingly show that these psychosocial risk factors do not exist in a vacuum, but rather dynamically interact with one another, thereby requiring more comprehensive theoretical models to guide future research. For example, the transactional model of hostility stipulates that the cynical mistrustfulness associated with this disposition serves as a driving force to create a social environment conducive to antagonistic behaviors that degrade social support resources and exacerbates emotional strain (e.g., Vella, Kamarck, Flory, & Manuck, 2012). Finally, the reserve capacity model was designed to explain associations between SES and health outcomes, whereby lower SES directly increases perceptual stress levels while simultaneously reducing social support resources for coping (the reserve capacity); in turn, both of these SES-induced developments of increased stress and decreased coping reserve are thought to directly elevate daily negative emotions/cognitions and reduce positive emotions/cognitions, collectively altering physiological processes that underlie CHD risk (Gallo & Matthews, 2003).

Numerous issues pervade this literature and are worthy of mention as limitations for consideration to improve subsequent research. First, whether dispositional or environmental in scope, all psychosocial risk factors reviewed here feature the same common denominator of elevated stress levels that may impact upon health status via direct and indirect pathways. As such, researchers should focus their attention on incorporating standardized measures of perceptual stress levels in concert with the psychosocial risk factors in question to disentangle the pathways leading to CHD outcome. Second, most of these psychosocial risk factors are multifaceted in scope, suggesting a need to concentrate efforts on measurement issues to reliably delineate the relative degrees to which psychosocial risk factor attributes predict the development and prognosis of CHD. Finally, just as it is known that functional social support may

improve one's ability to effectively cope with stressful life experiences, reducing risk of CHD morbidity and mortality, researchers should also counterbalance their investigations by incorporating other psychosocial "protective" factors that may confer significant health benefit as part of their theoretical models (e.g., frequency of positive emotions and trait optimism). Future research on psychosocial risk factors of CHD should make targeted efforts to ascertain interactive pathways that include traditional risk factors to maximally predict health outcomes and enhance scientific understanding of these provocative associations.

Author Biography

Dr. Elizabeth J. Vella is an associate professor of psychology at the University of Southern Maine. Her research interests include the link between psychosocial factors and cardiovascular risk and the physiological mechanisms that may explain these associations, as well as the implications for stress management interventions in improving quality of life and reducing physiological responses to stressors among at risk populations. She has authored numerous articles in scientific journals and presented her research at academic conferences.

References

- Albus, C. (2010). Psychological and social factors in coronary heart disease. *Annals of Medicine*, 42, 487–494. doi:10.3109/07853890.2010.515605
- Barth, J., Schneider, S., & von Känel, R. (2010). Lack of social support in the etiology and prognosis of coronary heart disease: A systematic review and meta-analysis. *Psychosomatic Medicine*, 72, 229–238. doi:10.1097/PSY.0b013e3181d01611
- Beaglehole, R., & Magnus, P. (2002). The search for new risk factors for coronary heart disease: Occupational therapy for epidemiologists? *International Journal of Epidemiology*, 31, 1117–1122. doi:10.1093/ije/31.6.1117
- Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology*, 53(11), 936–946. doi:10.1016/j.jacc.2008.11.044
- Dembroski, T. M., MacDougall, J. M., Costa, P. T., & Grandits, G. A. (1989). Components of hostility as predictors of sudden death and myocardial infarction in the Multiple Risk Factor Intervention Trial. *Psychosomatic Medicine*, 51, 514–522.
- Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin*, 129(1), 10–51. doi:10.1037/0033-2909.129.1.10
- Gan, Y., Gong, Y., Tong, X., Sun, H., Cong, Y., Dong, X., ... Lu, Z. (2014). Depression and risk of coronary heart disease: A meta-analysis of prospective cohort studies. *BMC Psychiatry*, 14, 371–382. doi:10.1186/s12888-014-0371-z
- Ganster, D. C., & Rosen, C. C. (2013). Work stress and employee health: A multidisciplinary review. *Journal of Management*, 39(5), 1085–1122. doi:10.1177/0149206313475815
- Hecker, H. L., Chesney, M. A., Black, G. W., & Frautschi, N. (1988). Coronary-prone behaviors in the Western Collaborative Group Study. *Psychosomatic Medicine*, 50, 153–164.
- Kivimäki, M., Virtanen, M., Elovainio, M., Kouvonen, A., Väänänen, A., & Vahtera, J. (2006). Work stress in the etiology of coronary heart disease: A meta-analysis. *Scandinavian Journal of Work Environment Health*, 32(6), 431–442. doi:10.5271/sjweh.1049
- Labarthe, D. R. (1998). Coronary heart disease. In J. Colilla (Ed.), *Epidemiology and prevention of cardiovascular diseases: A global challenge* (pp. 43–72). Gaithersburg, MD: Aspen Publishers, Inc.

- Lazzarino, A. I., Hamer, M., Stamatakis, E., & Steptoe, A. (2013). Low socioeconomic status and psychological distress as synergistic predictors of mortality from stroke and coronary heart disease. *Psychosomatic Medicine*, 75, 311–316. doi:10.1097/PSY.0b013e3182898e6d
- Leung, Y. W., Flora, D. B., Gravely, S., Irvine, J., Carney, R. M., & Grace, S. L. (2012). The impact of premorbid and postmorbid depression onset on mortality and cardiac morbidity among patients with coronary heart disease: Meta-analysis. *Psychosomatic Medicine*, 74, 786–801. doi:10.1097/PSY.0b013e31826dddbd
- Manrique-Garcia, E., Sidorchuk, A., Hallqvist, J., & Moraldi, T. (2011). Socioeconomic position and incidence of acute myocardial infarction: A meta-analysis. *Journal of Epidemiology and Community Health*, 65(4), 301–309. doi:10.1136/jech.2009.104075
- Rosenman, R. H., Brand, R. J., Jenkins, C. D., Friedman, M., Straus, R., & Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study: Final follow-up of 8.5 years. *Journal of the American Medical Association*, 233, 872–877. doi:10.1001/jama.1975.03260080034016
- Sherwitz, L., & Brand, R. (1990). Interviewer behaviors during structured interviews to assess Type A/B behavior in the Western Collaborative Group Study and the Multiple Risk Factor Intervention Trial. *Journal of Psychopathology and Behavioral Assessment*, 12(1), 27–47.
- Vella, E. J., Kamarck, T. W., Flory, J. D., & Manuck, S. (2012). Hostile mood and social strain during daily life: A test of the transactional model. *Annals of Behavioral Medicine*, 44(3), 341–352. doi:10.1007/s12160-012-9400-7
- World Health Organization. (2015). Cardiovascular diseases fact sheet. Retrieved from <http://www.who.int/mediacentre/factsheets/fs317/en/>.
- World Heart Federation. (2015). Cardiovascular disease risk factors. Retrieved from <http://www.world-heart-federation.org/cardiovascular-health/cardiovascular-disease-risk-factors/>.

Suggested Reading

- Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health*, 26, 469–500. doi:10.1146/annurev.publhealth.26.021304.144542
- Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical review of research on coronary heart disease. *Annual Review of Psychology*, 53, 341–369. doi:10.1146/annurev.psych.53.100901.135208
- Matthews, K. A. (2005). Psychological perspectives on the development of coronary heart disease. *American Psychologist*, 60(8), 783–796. doi:10.1037/0003-066X.60.8.783

Relationship Dissolution and Health

Karen Hasselmo, Kyle J. Bourassa, and David A. Sbarra

Department of Psychology, University of Arizona, Tucson, AZ, USA

Humans are inherently social beings, and intimate relationships are a particularly important part of our lives. The beneficial effects of intimate relationships may operate directly by encouraging healthy behaviors (e.g., high-quality sleep or a healthy diet) or more indirectly by buffering against the effects of psychological stress. Conversely, the absence of relationships, or being in a low-quality relationship, is associated with a variety of negative health outcomes. This entry focuses on health outcomes associated with romantic separations. Specifically, we report on the epidemiological data connecting romantic separations with morbidity and mortality, explore the potential behavioral physiological mechanisms linking these outcomes with the dissolution of the relationship (e.g., cardiovascular and immune system processes, health behaviors), and consider the psychological variables that may explain the associations of interest, including forgiveness, rumination, and psychological distancing. We end with future directions for research and intervention development.

When intimate relationships end, most people are resilient and cope well over time. The end of an intimate relationship can, however, set the stage for adverse effects on well-being—whether it is divorce after decades of marriage or the breakup with a first love.¹ For the subset of people who *are* affected negatively by a separation experience, these effects can exert their negative influence across a range of health-relevant physiological systems.

Morbidity and Mortality Following Dissolution

The bird's-eye view of intimate relationship dissolution offers one perspective on the connection between separations and poor health. In large samples of separated and/or divorced people, average findings indicate an increased risk for all-cause morbidity and mortality (Sbarra, Law, & Portley, 2011). For example, in a long-term study of the Uppsala birth cohort, being divorced placed both men and women in the highest risk groups for early mortality, with a 46 and 27% higher relative mortality risk than their married counterparts, respectively (Donrovich, Drefahl, & Koupil, 2014).

Research has begun to connect specific disease processes to marital status changes. In a sample of over 3.5 million participants, for example, married participants had lower odds of any cardiovascular disease ($OR = 0.95$), whereas divorced participants had higher odds ($OR = 1.05$) relative to single adults (Alviar, Rockman, Guo, Adelman, & Berger, 2014). Losses in bone density are also connected to divorce or separation; divorced men, relative to those who are married, demonstrated a 0.33 standard deviation reduction in lumbar spine-bone mass index. This number was similar (0.36) even for those men who remarried.

Infectious diseases are also associated with marital status. In a cohort of over 5.5 million men and women followed for hospital contacts over three decades, divorced participants had a 48% greater chance of contracting a hospital-diagnosed infectious disease when compared with married participants (Nielsen, Davidsen, Hviid, & Wohlfahrt, 2014). This increased infection risk was sustained in a 15-year follow-up. Other research demonstrates that rates of illness peak around the time of divorce and may not return to their baseline levels. Beyond physical health, marital status is predictive of mood and anxiety disorders across the world. In a study conducted by the World Mental Health Survey Initiative on a sample of 35,500 participants, the odds ratio of any mood or anxiety disorder increased 23% for divorced participants relative to married adults, and this effect was much stronger for women ($OR = 2.6$; McDowell et al., 2014).

These epidemiological findings demonstrate the importance of studying health outcomes following a separation but offer few hints regarding what processes might underlie the association between dissolution of intimate relationships and negative health outcomes.

Health-Relevant Responses to Relationship Dissolution

A thorough account of how relationship breakups can ultimately affect physical health links psychological responses to separations with biologically plausible mechanisms of action (cf. Miller, Chen, & Cole, 2009). Physiological responding can be studied to investigate emotions and emotional responses, as well as an intermediate health-relevant index used to link emotional reactions with long-lasting, systemic changes in the body. For example, cortisol may play a role in the distress response following a social stressor, but it may also function as the mediator of the relationship between stress and a pathological inflammatory response. Thus, care must be taken in discussing physiological responses in general versus physiological responses that have clear implications for health.

Miller et al. (2009) have advanced a conceptual approach to studying how biobehavioral responses to life events are associated with and may regulate (and be regulated by) disease-relevant molecular and cellular responses. This approach highlights the role of biological intermediaries, or, as Miller et al. (2009) stated, “Once a robust linkage between a psychosocial factor and a clinical health outcome is identified, the next step is to determine what biological processes convey those effects into the physical environment of disease pathogenesis” (p. 504).

Related to divorce, Miller et al.’s (2009) first criterion is satisfied. Beyond the meta-analytic findings we described above, other studies demonstrate that divorce can result in a proliferation of stress that mediates the association between the end of marriage and poor self-rated health up to a decade later. We do not yet know, however, the biological intermediaries that translate the psychosocial stress of divorce into poor physical health. We have some clues as to how these effects might operate but lack clear-cut, disease-relevant pathways. In the following sections, we review what is known with respect to cardiovascular, immune, and endocrine outcomes. We also discuss the role of relationship dissolution in shaping people’s health behaviors, as these behaviors are associated with changes in each of these systems.

Cardiovascular Responding

Cardiovascular processes are one possible link between intimate relationship dissolution and negative health outcomes. A dysregulated cardiovascular response to stress is thought to be an indicator of—if not causally linked to—the development of cardiovascular disease. It is well established that an exaggerated response to environmental stress leads to rises in future blood pressure, and there is evidence to suggest hyperreactivity is reliably associated with a host of other cardiovascular issues, such as atherosclerosis (i.e., buildup of plaque in the arteries) and increased cardiac hypertrophy (i.e., thickening of heart muscles; Chida & Steptoe, 2010). Conversely, hyporeactivity, or a blunted response to stress, is also linked with negative health outcomes, including obesity, depression, and poor self-rated health, possibly due to its association with behaviors driven by disordered motivation, such as heightened focus on rewarding stimuli. As a result, both hyper- and hyporeactivity may indicate health risk following a stressful separation experience. For example, when asked to reflect over their relationship history and recent separation experience, divorced adults who scored high in anxious attachment style and who spoke in a verbally immediate way (i.e., indicating a pattern of continual engagement with the dissolved relationship) evidenced significantly higher blood pressure at the start of a divorce-related experimental task (Lee, Sbarra, Mason, & Law, 2011). In a similar investigation, high levels of divorce-related emotional intrusion were associated with elevated blood pressure, and men who find thinking about their separation especially difficult evidence substantial blood pressure increases when asked to do so (Sbarra, Law, Lee, & Mason, 2009).

Heart rate variability, as indexed by respiratory sinus arrhythmia (RSA) (Porges, 1995), is a second active area of research in cardiac responding. RSA represents the action of the vagus nerve, which exerts inhibitory control over the heart. Higher RSA is adaptive in certain situations, as it helps prepare the body to free-metabolic resources for coping responses driven by the sympathetic nervous system. Low RSA is predictive of greater cardiovascular disease risk; moreover, context inappropriate changes in RSA are thought to index poor self-regulation (Porges, 1995). Regarding RSA reactivity in divorced adults, Sbarra and Borelli (2012) found that adults who reported having avoidant attachment styles, conceptualized as an ability to achieve emotional distance from their relationships, and who were successful at regulating their emotions during a divorce-related task, as indexed by high levels of RSA reactivity during the task, evidence the greatest improvements in their self-concept over the course of three months (Sbarra & Borelli, 2012). Further research involving RSA and adults' adjustment following divorced considered a genetic polymorphism in the serotonin transporter gene (SERT). The "short" variation of this allele results in less available serotonin in the brain, and adults with the genetic polymorphism who also reported difficulty adjusting to their separation experience had significantly decreased levels of RSA during a task that asked them to reflect on their divorce experience (Hasselmo, Sbarra, O'Connor, & Moreno, 2015).

Immune Functioning

A second health-relevant psychophysiological pathway through which the end of an intimate relationship may exert its negative effects is via the immune system. Much like the cardiovascular pathway, the immune system has important functions for survival and health, and higher levels of social support are generally associated with an increase in physiological functioning in both the cellular and humoral divisions of the immune system (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Conversely, divorced/separated adults experience higher rates of mortality due to infectious diseases compared with their married counterparts. Psychoneuroimmunologists

investigate the immune correlates of social influence by looking at both numbers of important immune cells and functionality of the system itself, both of which are required to mount an appropriate immune response (Uchino et al., 1996). Intimate relationship dissolution may influence the immune system through both of these routes. For example, when compared with married adults on various measure of immune system functioning, recently divorced participants showed decreased functioning on several immune parameters (Kiecolt-Glaser et al., 1987). A weakened immune system following the chronic stress of divorce or separation may lead to the higher rate of infectious diseases in this population. The mechanism that links the psychosocial effects of divorce or separation and the immune system is not yet understood, but two candidate pathways have emerged.

One route through which the stress of separation from a partner influences immune parameters is the endocrine system, specifically the various hormones this system releases in response to stress. Cortisol, a steroid hormone, has a particularly diverse range of effects within the body, including the ability to bind to the white blood cells of the immune system and regulate both their distribution throughout the body and their functional responses (Segerstrom & Miller, 2004). Cortisol plays an important role in preparing the body to respond to stress, but if levels are elevated chronically, it can lead to the development of chronic inflammatory diseases such as arthritis and asthma, among others. Preliminary data from Powell et al. (2002) investigated 20 women who were undergoing a separation event and 20 who were in stable marriages. They found that separating women had elevated salivary cortisol, suggesting a link between relationship dissolution and cortisol levels. Although this data is encouraging, more specific studies linking the cortisol pathway to divorce in particular are needed.

An exaggerated cortisol response can be deleterious in and of itself, but it can also set the stage for other problematic immune responses. In particular, it can permit inflammation over time. Similar to the hyperactivation of the cardiovascular system following the dissolution of an intimate relationship, the immune system can be over-activated in response to the chronic psychological stress associated with a breakup. The immune response to stress often initiates with the release of chemical messengers, known as cytokines, which alert the rest of the body to the presence of an intruder. If these cytokines are pro-inflammatory, they set off a cascade of processes in the body that promote inflammation. Although these pro-inflammatory cytokines are adaptive at first, they can become dysregulated during the chronic stress following a distressing event. One model suggests that glucocorticoid receptors in the brain, which receive the inflammatory message of these cytokines and then downregulate the signal, become desensitized to the pro-inflammatory cytokines when they are chronically proliferating (Miller, Cohen, & Ritchey, 2002). Without this glucocorticoid regulation, the inflammatory response persists, elevating risk for a range of chronic inflammatory diseases, including Type 2 diabetes, various autoimmune diseases, and cardiovascular disease. This pro-inflammatory pathway receives considerable attention in the broad literature on stress and health but has yet to be studied as people adapt to a divorce or a romantic breakup.

Health Behaviors

Although it is possible that the psychological stress of divorce is associated directly with changes in the aforementioned physiological systems, it is important to recognize that lifestyle habits following a breakup also play a powerful role in shaping the responses in these systems. In this way, health behaviors may be important in understanding the intermediate steps from relationship dissolution to morbidity and mortality. For example, social relationships promote

important behaviors like seeking medical care and adherence to care while also encouraging self-care like exercising, healthy eating, and avoiding illicit drug use. A recent study of adherence to doctor-provided cancer prevention guidelines noted that the number one indicator of reductions in all-cancer incidence and cancer mortality was following the prescribed guidelines (Kabat, Matthews, Kamensky, Hollenbeck, & Rohan, 2015). As being married increases adherence to medical advice, this may affect health at multiple levels for divorced or separated adults. Similarly, the loss of this social pressure in the form of a spouse or partner may disrupt these healthy lifestyle behaviors. Furthermore, there is epidemiological evidence that divorce or separation promotes the adoption of unhealthy habits like substance abuse. Women in particular are vulnerable to decreases in body mass, increased likelihood of smoking relapse, and decreases in the amount of healthy fruits and vegetables consumed following divorce. The cessation of positive health behaviors and the development of unhealthy ones result in an increased risk for the promotion and maintenance of cardiovascular problems and deleterious changes in the immune functioning.

Sleep is an additional lifestyle factor that may affect health following the end of marriage. Disturbances in sleep can lead to dysregulated endocrine and autonomic nervous system functioning and even directly to higher mortality risk. Research suggests that divorced people have a higher incidence of sleep disturbance than partnered individuals. An early study of 70 subjects undergoing a separation period revealed these participants exhibited less delta wave sleep during their separation experience than their age-matched, married counterparts, indicating lighter overall sleep (Cartwright & Wood, 1991). Epidemiological studies reveal that being separated or divorced is associated with an elevated risk for severe insomnia and sleep maintenance problems (Hajak, 2001). Another study focusing on 367 middle-aged women's relationship histories over 6–8 years noted that continuously married or cohabitating participants have better sleep quality (measured both by self-report and a number of objective measures) than women who were unpartnered or gained/lost a partner during this same time period (Troxel et al., 2010). More recently, research on divorced adults suggests that sleep disturbances following separation predict future increases in blood pressure (Krietsch, Mason, & Sbarra, 2014). These increases were especially noteworthy in participants who continued to report disturbances 10 weeks after the separation and who evidenced the greatest increases in future blood pressure (Krietsch et al., 2014). Sleep highlights another pathway through which cardiovascular functioning may increase morbidity and mortality following a separation experience.

Integrating Across Multiple Domains: Allostatic Load

If the presence of indicators of health risk across a variety of health-relevant physiological systems are chronic and/or severe enough, their effects may accrue to produce accumulated wear and tear on the body, termed *allostatic load* (McEwen, 2000). *Allostasis* describes a physiological or behavioral adaptation to environmental changes in order to restore normality, or *homeostasis* (McEwen, 2000). Excessive cardiovascular, neuroendocrine, and immune activations in response to stress can promote vascular remodeling, initiate atherosclerotic plaque growth, and alter gene expression in a manner that contributes to disease pathogenesis (Miller et al., 2009). These processes may lead to the increased morbidity and mortality rates noted among populations affected by divorce and separation, though this area requires future research. To the extent that the cardiovascular and immune systems stay activated over time, that wear and tear may lead to illness and early death. What psychological processes are associated with the prolonged, hyperactivation of these physiological systems? We consider this question in the final section of the chapter.

Psychological Processes and Health Outcomes

In terms of psychological characteristics associated with adjustment to divorce, it is well known that individual differences in attachment anxiety are associated with poor outcomes when people perceive a threat to their relationship and/or their security within the relationship. In a study mentioned above in relation to blood pressure responses, Lee et al. (2011) studied language as a behavioral manifestation of attachment-related hyperactivation. People higher in attachment anxiety who spoke about their separation experience in a highly immediate, experiential, and self-focused manner demonstrated greater increases in systolic and diastolic blood pressure when thinking about their relationship history and separation experience relative to those people lower in attachment anxiety. People at high risk for poor outcomes following marital separation appear to employ coping strategies that are associated with a high degree of physiological activation, and this study, focused on blood pressure reactivity, provides an example of the ways in which emotion regulation strategies around attachment themes can provide insights into processes that confer risk for poor distal health outcomes.

Other research on emotion regulation following divorce supports this idea. For example, an intervention for recently divorced people (Sbarra, Boals, Mason, Larson, & Mehl, 2013) focused on the use of expressive writing (EW; writing about emotional feelings) compared with control writing (continuously writing about nonemotional issues). The results indicated that EW increased distress related to the divorce 3-month follow-up for people high on rumination when compared with low ruminators who were in the EW condition and all people in the control writing condition. After 8 months, however, all people in the EW condition and low ruminators in the control writing condition were equivalent in their distress, whereas high ruminators, purportedly at the most risk for negative outcomes following divorce, evidenced the lowest distress. The results suggest that allowing people who are at risk for rumination to focus on actively structuring their time (rather than “digging more deeply” into the emotions associated with their separation) reduces their psychological distress following marital separation.

Together, the findings related to attachment anxiety and rumination suggest potential psychological mechanisms linking marital separation to poor health outcomes. People who have a hard time distancing themselves from their psychological experiences show excessive cardiovascular responding, which, as noted above, is associated with the development of cardiovascular disease. Conceptually, this work fits well with the larger literature on self-distanced reflection and evidence indicating that people who *recount* their experiences in a blow-by-blow manner rather than *reconstrue* their experiences to find meaning are at heightened risk for mood disorders (see Kross & Ayduk, 2011).

In addition to attachment anxiety and rumination, forgiveness is another important psychological variable associated with adults' recover from divorce. Rye et al. (2005) developed an intervention focused on facilitating forgiveness for an ex-spouse using previous research indicating that forgiveness predicted improved well-being, as well as reduced depressive symptoms and both state and trait anger. The intervention was based on a prior intervention designed to increase forgiveness for previous wrongs in a romantic relationship in college women, which resulted in higher levels of forgiveness and well-being for participants. Among divorcing adults, this team tested two versions of their forgiveness intervention (secular versus religious forgiveness) and a no-treatment control using a randomized control trial (RCT) over 8 weeks of treatment and a 6-week follow-up period. Participants in both the secular and religious intervention conditions showed improvements in forgiveness of their ex-partner compared

with control, though only the secular condition evidenced decreased levels of depression. This program of research and evidence from a laboratory experiment that less forgiveness results in higher skin conductance, heart rate, and blood pressure compared with baseline (van Oyen Witvliet, Ludwig, & Vander Lann, 2001) suggests that interventions designed to target forgiveness following divorce and romantic breakups may yield benefits in health outcomes.

Beyond self-distanced reflection and forgiveness, other variables and processes may serve as potential explanatory pathways leading to health-relevant biological changes. In a prospective study of breakups following nonmarital dissolution (Mason, Law, Bryan, Portley, & Sbarra, 2012), improvements in self-concept clarity (knowing who you are as a person after a separation) were associated with increases in future psychological well-being. There was no evidence in this study that people begin to feel better, who then report a greater sense of who they are after their breakup; instead, the direction of the effect seems to operate from self-concept clarity to improved psychological well-being. In a recent experimental study, Larson and Sbarra (2015) found that people who simply reflected on their separation experience multiple times over nine weeks increased their sense of self-concept clarity, which, in turn, explained decreases in loneliness and breakup-related emotional distress over the entire study period. Self-concept clarity was a key variable in early accounts of the psychological response to divorce (Weiss, 1975), yet no studies to date have examined this variable with respect to biomarkers of interest.

Conclusions

Intimate relationships are an important part of people's lives and can be beneficial for psychological and physical health. When relationships end, however, people are broadly vulnerable to morbidity and mortality from all causes, potentially due to changes in cardiovascular and immune responding. Alterations in these so-called biological intermediaries appear to be the result of changes in health behaviors, including sleep, medical adherence, self-care, healthy eating, and exercise. There are several potential psychological mechanisms that may link the end of an intimate relationship to distal health outcomes. Examples provided in this entry include experiential over-involvement, rumination, forgiveness, and self-concept clarity. Because of the health risks conferred by romantic separations, it seems essential to target those most at greatest risk for poor outcomes. Future interventions after romantic separation and divorce should focus on affecting the health behaviors and psychological processes proposed to mediate the link between romantic separation and health outcomes, with the goal of reducing the health burden associated with separation. In short, romantic separation and divorce are common stressful events, and these experiences are associated with negative, health-relevant consequences for a subset of people.

Author Biographies

Karen Hasselmo, MA, is a doctoral candidate at the University of Arizona, and her research centers on psychological and physiological outcomes of relationship dissolution.

Kyle J. Bourassa, MA, is a doctoral candidate at the University of Arizona, and his research centers on dyadic processes in intact relationships, as well as how prior relationship characteristics might affect people's well-being following romantic separation.

David A. Sbarra, PhD, is an associate professor of psychology at the University of Arizona. His research focuses on social relationships and health, as well as how people recover from difficult social transitions.

Note

- 1 Although there are obvious differences between the end of marriage and a nonmarital breakup, in this entry, we consider literatures relevant to both. Where the category matters, we make the distinction clear.

References

- Alviar, C. L., Rockman, C., Guo, Y., Adelman, M., & Berger, J. (2014). Association of marital status with vascular disease in different arterial territories: A population based study of over 3.5 million subjects. *Journal of the American College of Cardiology*, 63(12), A1328. doi:10.1016/S0735-1097(14)61328-0
- Cartwright, R. D., & Wood, E. (1991). Adjustment disorders of sleep: The sleep effects of a major stressful event and its resolution. *Psychiatry Research*, 39, 199–209. doi:10.1016/0165-1781(91)90088-7
- Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status a meta-analysis of prospective evidence. *Hypertension*, 55(4), 1026–1032. doi:10.1161/HYPERTENSIONAHA.109.146621
- Donrovich, R., Drefahl, S., & Koupil, I. (2014). Early life conditions, partnership histories, and mortality risk for Swedish men and women born 1915–1929. *Social Science & Medicine* (1982), 108, 60–67. doi:10.1016/j.socscimed.2014.02.036
- Hajak, G. (2001). Doxepin shows promise in primary insomnia. *Inpharma*, 1302, 25. doi:10.2165/00128413-200113020-00032
- Hasselmo, K., Sbarra, D. A., O'Connor, M. F., & Moreno, F. A. (2015). Psychological distress following marital separation interacts with a polymorphism in the serotonin transporter gene to predict cardiac vagal control in the laboratory. *Psychophysiology*. doi:10.1111/psyp.12409
- Kabat, G. C., Matthews, C. E., Kamensky, V., Hollenbeck, A. R., & Rohan, T. E. (2015). Adherence to cancer prevention guidelines and cancer incidence, cancer mortality, and total mortality: A prospective cohort study. *American Journal of Clinical Nutrition*, 101(3), 558–569. doi:10.3945/ajcn.114.094854
- Kiecolt-Glaser, J. K., Fisher, L. D., Ogrocki, P., Stout, J. C., Speicher, C. E., & Glaser, R. (1987). Marital quality, marital disruption, and immune function. *Psychosomatic Medicine*, 49(1), 13–34.
- Krietsch, K. N., Mason, A. E., & Sbarra, D. a. (2014). Sleep complaints predict increases in resting blood pressure following marital separation. *Health Psychology*, 33(10), 1204–1213. doi:10.1037/hea0000089
- Kross, E., & Ayduk, O. (2011). Making meaning out of negative experiences by self-distancing. *Current Directions in Psychological Science*, 20, 187–191. doi:10.1177/0963721411408883
- Larson, G. M., & Sbarra, D. A. (2015). Participating in research on romantic breakups promotes emotional recovery via changes in self-concept clarity. *Social Psychological and Personality Science*, 6(4), 399–406. doi:10.1177/1948550614563085
- Lee, L. A., Sbarra, D. A., Mason, A. E., & Law, R. W. (2011). Attachment anxiety, verbal immediacy, and blood pressure: Results from a laboratory-analogue study following marital separation. *Personal Relationships*, 18(2), 285–301. doi:10.1111/j.1475-6811.2011.01360.x. doi:10.1016/j.ijpsycho.2004.11.007

- Mason, A. E., Law, R. W., Bryan, A. E., Portley, R. M., & Sbarra, D. A. (2012). Facing a breakup: Electromyographic responses moderate self-concept recovery following a romantic separation. *Personal Relationships, 19*(3), 551–568.
- McDowell, R. D., Ryan, A., Bunting, B. P., O'Neill, S. M., Alonso, J., Bruffaerts, R., ... Tomov, T. (2014). Mood and anxiety disorders across the adult lifespan: A European perspective. *Psychological Medicine, 44*(04), 707–722. doi:10.1017/S0033291713001116
- McEwen, B. S. (2000). The neurobiology of stress: from serendipity to clinical relevance. *Brain Research, 886*(1), 172–189. doi:10.1016/S0006-8993(00)02950-4
- Miller, G., Chen, E., & Cole, S. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524. doi:10.1146/annurevpsych.60.110707.163551
- Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: A glucocorticoid-resistance model. *Health Psychology, 21*(6), 531–541. doi:10.1037/0278-6133.21.6.531. doi:10.1111/j.1532-5415.2000.tb03901.x
- Nielsen, N. M., Davidsen, R. B., Hviid, A., & Wohlfahrt, J. (2014). Divorce and risk of hospital-diagnosed infectious diseases. *Scandinavian Journal of Public Health, 42*(7), 705–711. doi:10.1177/1403494814544398
- Porges, S. W. (1995). Cardiac vagal tone: A physiological index of stress. *Neuroscience and Biobehavioral Reviews, 19*(2), 225–233. doi:10.1016/0149-7634(94)00066-A
- Powell, L. H., Lovallo, W. R., Matthews, K. A., Meyer, P., Midgley, A. R., Baum, A., ... Ory, M. G. (2002). Physiologic markers of chronic stress in premenopausal, middle-aged women. *Psychosomatic Medicine, 64*(3), 502–509.
- Rye, M. S., Pargament, K. I., Pan, W., Yingling, D. W., Shogren, K. A., & Ito, M. (2005). Can group interventions facilitate forgiveness of an ex-spouse? A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 73*(5), 880. doi:10.1037/0022-006X.73.5.880
- Sbarra, D. A., Boals, A., Mason, A. E., Larson, G. M., & Mehl, M. R. (2013). Expressive writing can impede emotional recovery following marital separation. *Clinical Psychological Science, 1*(2), 120–134. doi:10.1055/s-0029-1237430.Imprinting
- Sbarra, D. A., & Borelli, J. L. (2012). Heart rate variability moderates the association between attachment avoidance and self-concept reorganization following marital separation. *International Journal of Psychophysiology, 88*(3), 253–260. doi:10.1016/j.ijpsycho.2012.04.004
- Sbarra, D. A., Law, R. W., Lee, L. A., & Mason, A. E. (2009). Marital dissolution and blood pressure reactivity: Evidence for the specificity of emotional intrusion-hyperarousal and task-rated emotional difficulty. *Psychosomatic Medicine, 71*(5), 532–540. doi:10.1097/PSY.0b013e3181a23eee
- Sbarra, D. A., Law, R. W., & Portley, R. M. (2011). Divorce and death: A meta-analysis and research agenda for clinical, social, and health psychology. *Perspectives on Psychological Science, 6*(5), 454–474. doi:10.1177/1745691611414724
- Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601–630. doi:10.1037/0033-2909.130.4.601
- Troxel, W. M., Buysse, D. J., Matthews, K. A., Kravitz, H. M., Bromberger, J. T., Sowers, M., & Hall, M. H. (2010). Marital/cohabitation status and history in relation to sleep in midlife women. *Sleep, 33*(7), 973–981.
- Uchino, B., Cacioppo, J., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*(3), 488–531. doi:10.1037/0033-2909.119.3.488
- van Oyen Witvliet, C., Ludwig, T. E., & Vander Laan, K. L. (2001). Granting forgiveness or harboring grudges: Implications for emotion, physiology, and health. *Psychological Science, 12*(2), 117–123. doi:10.1111/1467-9280.00320
- Weiss, R. S. (1975). *Marital separation*. New York, NY: Basic Books.

Suggested Reading

- Miller, G., Chen, E., & Cole, S. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, 60, 501–524. doi:10.1146/annurev.psych.60.110707.163551
- Sbarra, D. A., Hasselmo, K., & Bourassa, K. J. (2015). Divorce and health: Beyond individual differences. *Current Directions in Psychological Science*, 24(2), 109–113. doi:10.1177/0963721414559125
- Sbarra, D. A., Law, R. W., & Portley, R. M. (2011). Divorce and death: A meta-analysis and research agenda for clinical, social, and health psychology. *Perspectives on Psychological Science*, 6(5), 454–474. doi:10.1177/1745691611414724

Risk Perception

Vera Hoorens

*Center for Social and Cultural Psychology, Katholieke Universiteit Leuven,
Leuven, Belgium*

Risk perception is people's judgment of future outcomes that may occur if they or other people follow a given course of action. Although risks may involve desirable as well as undesirable outcomes, the term is mostly used to describe undesirable outcomes such as health and safety hazards or natural or man-made disasters. Some researchers distinguish between risk perception and risk evaluation. By risk perception they mean the estimation of the risk's magnitude; by risk evaluation they mean judgments of risk acceptability, assumed to depend not only on the perceived magnitude of the risk but also on other risk characteristics. Other researchers use the term "risk perception" in a broader sense, including judgments of risk magnitude as well as of any other characteristic that might affect the risk's acceptability. Risk perception in both senses is to be distinguished from risk assessment, which is a formal and systematic risk analysis undertaken by risk experts (such as actuaries) in the context of financial or policy decisions.

Understanding how people perceive and evaluate risks is important to understand health and safety behavior and responses to risks of man-made and natural disasters. To explain why people are willing (or unwilling) to pay for insurance, for instance, or to explain why they freely enter objectively dangerous situations but feel unsafe in objectively non-dangerous ones, one needs to understand how people perceive the risks involved. Not surprisingly, risk perception and evaluation have been studied by researchers from a wide variety of disciplines, including psychology, economics, criminology, and sociology.

Early research often started with the assumption (sometimes implicit) that people intuitively follow normative statistical or logical rules while judging risk magnitude. From a normative point of view, the magnitude of a given individual's risk is a joint function of the perceived valence and extremity of the outcomes that he or she may experience (in more technical terms, outcomes' expected subjective utility) and these outcomes' perceived likelihood. Although many studies have focused on such individual risk perception (e.g., a vacationer judging her risk of skiing accidents), people may also form impressions of collective risks (e.g., a travel guide judging the risk of skiing accidents in her group of vacationers). Normatively, judgments

of the magnitude of overall risk should then also depend on the number of individuals who are likely to be affected. Assuming that people can and sometimes do intuitively follow normative rules, researchers in the domain of individual risk perception have studied how people estimate event likelihoods, how they arrive at expected subjective utilities, and how they combine likelihood and utility information toward an overall judgment of a risk's magnitude. In addition to these questions, researchers in the domain of collective risk perception have also examined how people perceive the number of individuals affected and how they integrate this information in their general risk perception.

More recent studies on risk perception are rooted in the view that people can apprehend reality in two fundamentally distinct manners: in an analytical manner, or in an experiential manner. From this perspective, people are thought to sometimes base their evaluation of the risks that are associated with a given activity on a systematic consideration of the associated outcomes and their likelihoods, but in other cases they base their risk evaluation on the affective state they experience while considering the activity. The analytical manner is deliberative, verbal, systematical, and slow, whereas the experiential manner is affective, nonverbal, intuitive, and quick. Although researchers who support the dual-process approach continue to value research on likelihood estimation, expected subjective utility, and the combination of the two, they also examine how affective states determine risk perception and strive to identify the circumstances in which either systematic risk analysis or affect-driven risk perception occurs. Among the circumstances that have been shown to favor affect-driven risk perception are strong emotional reactions, time pressure, and reduced cognitive resources (e.g., Finucane, Alhakami, Slovic, & Johnson, 2000).

Likelihood Estimation

One can estimate the likelihood of an event by observing how often, out of the cases in which it might have occurred, it has indeed occurred. People sometimes try to engage in this exercise, but in many cases they do so incompletely or use a totally different approach.

When relative frequencies are used to estimate probabilities, one should weight each nonoccurrence as heavily as each occurrence. Five occurrences of an infection clearly represent a different likelihood of infection if 20 individuals were exposed than if 200 individuals were exposed. However, people focus on occurrences to the neglect of nonoccurrences. When likelihood or frequency information is presented to them in the form of a proportion or a fraction (e.g., a 1 out of 100 chance, a 10 out of 1,000 chance), they tend to focus on the numerator (in the examples just given, 1 or 10, respectively) to the neglect of the denominator (in the examples just given, 100 or 1,000, respectively). Thus, a 10 out of 1,000 chance seems larger than a 1 out of 100 chance even though the probability is identical in each case. One implication of this phenomenon is that likelihoods that are expressed in a probability format (i.e., as a value from 0 to 1 or as a percentage) seem smaller than likelihoods that are expressed in a frequency format with a denominator above 1.

One impressive demonstration of this phenomenon was produced by Slovic, Monahan, and MacGregor (2000). They asked mental health professionals to judge if a given patient should be released from the hospital given the risk of the patient committing acts of violence. Half of the participants read that according to another expert, "20 out of 100" comparable patients committed acts of violence, whereas the other half read that according another expert, comparable patients had a "20% likelihood" of committing acts of violence. Participants were much less willing to release the patient in the former case than in the latter, indicating that they considered the likelihood of violent actions higher in the former case than in the latter.

Even if people try to follow normative statistical rules, their estimates of how often events happen versus not happen is relatively easy to influence. For instance, support theory implies that people derive an estimate of an event's likelihood from the support that they can find for the hypothesis that the event will occur relative to support for the alternative hypothesis that it will not occur (Rottenstreich & Tversky, 1997). The support for the focal or the alternative hypothesis depends, among other variables, on the ways in which people can imagine that the event will happen (or not happen). Events seem more likely when specific instances are provided (when the events are "unpacked") than when the event is merely described through a general label. For instance, when people estimate their likelihood of contracting a disease on their vacation, their estimate will be higher when they consider Dengue, typhoid fever, cholera, diarrhea, hepatitis, and malaria than when they merely consider the general condition of "diseases that one may contract while on vacation."

One robust finding in the domain of likelihood estimation is that people often disregard normative rules of probability estimation and instead use simple rules of thumb, generally called heuristics (Furnham & Boo, 2011; Nisbett & Ross, 1980; Tversky & Kahneman, 1974). The availability heuristic implies that people base their judgment of the likelihood of an outcome on the ease with which they can remember or imagine that outcome. The representativeness heuristic implies that people base their judgment of the likelihood of a given action leading to a given outcome on the resemblance between the action and the potential outcome. For instance, people tend to believe that extreme or impressive actions provoke severe outcomes and that small, simple actions provoke no more than mundane outcomes.

A third heuristic, the anchoring-and-adjustment heuristic, implies that people start from an initial value (the "anchor") that they adjust to accommodate the specific risk information they may have. The anchor is whatever numerical value is active in working memory and hence may or may not be related to the specific risk at hand. Heuristics allow quick likelihood estimations that in many cases are sufficiently accurate to allow well-balanced decisions. In many other cases, however, they lead to systematic error. These errors tend to occur when factors other than outcome frequency critically determine an outcome's availability or its representativeness for the action under consideration, or when the anchor that is being used is unrelated to, or no more than remotely related to, the risk under consideration. Outcomes do not always resemble the actions that provoke them (e.g., seemingly unimpressive actions may have weighty consequences), such that the resemblance between actions, and outcomes does not reflect the strength of the association between them. Similarly, the ease by which people can remember or imagine an outcome depends on a variety of outcome characteristics, such as the vividness and the extensiveness with which they have been covered in the media or in the arts. People therefore overestimate the likelihood of outcomes that receive vivid media coverage. Finally, the anchor may be unrelated to the risk at hand. Because adjustments from initial anchors tend to be insufficient, irrelevant values may thus unduly affect likelihood perceptions.

Expected Subjective Utility

People do not judge events by comparing their valence and extremity to some absolute zero level, but to a psychological reference point. The choice of a particular reference point thus crucially determines if, and to which extent, an outcome will be experienced as a loss or as gain. The reference point may be the current state of affairs, a previously experienced or expected state of affairs, or any other baseline that happens to be cognitively available. "Objective" gains may even be perceived as losses and "objective" losses as gains

if they fall below the reference point that happens to be active. For instance, a patient suffering from a chronic disease may view the need for further treatment as a loss when comparing it the status quo but as a gain when comparing it to the deterioration that may occur without any treatment or when comparing it to the health status of other patients who are even worse off.

The expected subjective utility of an outcome is not a strictly increasing monotonic function of the distance between that outcome and the reference point. The further an outcome is from the reference point, the less a further departure will enhance the extremity of its utility. For instance, the difference between paying \$500 versus \$1,000 for a medical treatment “feels” larger than the difference between paying \$5,000 versus \$5,500. In addition, losses are generally weighted heavier than gains, and the subjective utility function of losses is generally steeper than the subjective utility function of gains. Unexpectedly being refunded \$500 feels like a gain, and unexpectedly having to pay an additional \$500 feels like a loss, but the expected subjective utility of the loss will be more extreme than the expected subjective utility of the gain.

One additional determinant of the expected subjective utility of an outcome is how easily people can imagine the relevant outcomes. Thus, vivid information about potential outcomes does not only enhance the subjective likelihood of these outcomes but may also make them seem more extreme. For instance, people are willing to pay more for flight insurance protecting them against losses caused by “terrorism” than for flight insurance protecting them against all losses, regardless of their cause, because terrorist attacks are easier to imagine than “any cause of loss” (Johnson, Hershey, Meszaros, & Kunreuther, 1993). The unpacking effect, which occurs in likelihood estimation, also has its equivalent in expected subjective utility, with instance-based descriptions of events leading to more extreme judgments than general event labels (Van Boven & Epley, 2003). In one study, for instance, people read about an oil refinery that released toxins in the atmosphere and thereby caused an increase in health problems in the surrounding community. The participants’ task was to recommend the magnitude of compensation awarded to each victim and to advise on how long the refinery should be closed. Participants recommended larger compensations and a longer closure if the health hazards were described as “asthma, lung cancer, throat cancer, and all other varieties of respiratory diseases” rather than as “all varieties of respiratory diseases” (Van Boven & Epley, 2003, Study 1).

Combining Likelihoods and Utilities

Inspired by normative expectancy-value models, many researchers have assumed that a multiplicative function describes how people combine their estimates of outcome likelihood and outcome severity to arrive at a general estimation of risk magnitude. However, this multiplicative function has seldom been observed. One reason seems to be that people are rather insensitive to differences among relatively high probabilities. When comparing risks involving outcomes that are moderately to highly likely, they tend to solely focus on differences in severity despite differences between the outcomes’ likelihood (Weinstein, 2000). Similarly, people seem insensitive to differences among very low probability events (Kunreuther, Novemsky, & Kahneman, 2001).

People seem to particularly disregard probabilities when the outcomes provoke strong emotions. In one study, participants indicated how much they would be willing to pay to eliminate a cancer risk due to arsenic in drinking water (Sunstein, 2003). The likelihood of getting

cancer due to arsenic pollution was described as being either 1 in 100,000 or 1 in 1,000,000. The cancer risk was described either in nonemotional terms or in a vivid and therefore frightening manner. In the former case, the number of participants willing to pay was considerably higher in the 1 in 100,000 risk condition than in the 1 in 1,000,000 risk condition. In the latter case, the participants were equally willing to pay in both conditions.

When events seem very serious or provoke fear, people solely focus on the outcome's subjective utility to the neglect of these outcomes' probability (Sunstein, 2003). In one demonstration of "probability neglect," participants imagined an experiment involving either a chance of experiencing a painful electric shock or having to pay a penalty of \$20. Importantly, some participants were told that the chance of having to undergo the shock or to pay the penalty was 1%; others were told that the chance was 99%. When asked how much they would be willing to pay to avoid participating in the experiment, participants were willing to pay much more to avoid a 99% chance of having to pay \$20 than to avoid a 1% chance of having to pay it (\$18 versus \$1). In contrast, they were not willing to pay much more to avoid a 99% chance of undergoing a painful shock than to avoid a 1% chance of undergoing it (\$10 versus \$7).

Finally, some evidence suggests that likelihood and extremity estimations are not independent from each other. A trade-off may occur, in that people assume that rare health hazards are serious and that more common health hazards are not so serious (Jemmott, Ditto, & Croyle, 1986). Of course, this rule of thumb may in many circumstances be accurate (if extremely serious hazards were very common, little would be left of humanity by now).

Perceiving the Numbers Involved

The manner in which people consider the number of potential victims in their judgments resembles the manner in which they arrive at expected subjective utilities. Perhaps the best documented phenomenon in this context is "psychic numbing" (also called "psychophysical numbing"), meaning that the subjective value of an additional life saved or lost diminishes as more lives are involved (e.g., Slovic, 2007). For instance, if people find it reasonable to spend a fixed amount of money to save one life, they do not find it reasonable to spend X times that amount to save X lives. Saving one life feels important, but saving an additional life given many others already saved does not seem to make such a difference.

Reference points also play a role in the judgment of numbers involved. One reference point that seems to be of psychological relevance is the population to which the individuals at risk belong. A given number of individuals at risk may seem smaller when the potential victims come from a large population than when they come from a small population, so that people are less willing to take action to save the potential victims in the former case than in the latter. For instance, Fetherstonhaugh, Slovic, Johnson, and Friedrich (1997, Study 1) had people express their preference for a program that would provide drinkable water to refugee camp and thus save 4,500 lives in a camp inhabited by 11,000 refugees or in a camp inhabited by 250,000 refugees. Participants showed a clear-cut preference for the program in the smaller camp than for the program in the larger camp, even though the absolute number of lives saved was identical. Fetherstonhaugh et al. (1997, Study 3) also asked a group of participants how many lives would need to be saved to warrant an investment of 10 million dollars in life-saving medical treatments. When the population at risk consisted of, say, 15,000 individuals, the median number of lives needed was 9,000. When the population at risk consisted of 290,000 individuals, the median number of lives needed was 100,000.

From Risk Magnitude to Risk Acceptability

The role of a large variety of outcome characteristics other than severity and likelihood has been examined as potential determinants of risk evaluation. These include, but are not limited to, how voluntarily people engage in the risky course of action; how much control they can still exert on the action's outcome given that it occurs; how well the potential outcome is known to the individuals who are at risk for it, to scientists, or to society as a whole; and how many people are exposed to the risk or may fall victim to it. Risk evaluation furthermore involves an evaluation of the expected or potential beneficial outcomes of the risky action. These additional determinants of risk evaluation, above and beyond the perception of risk magnitude, explain why people sometimes respond very differently to equally large risks. For instance, people tend to find risks that they can control more acceptable than risks they cannot control and tend to find the risks of actions that benefit them more acceptable than the risks of action that do not benefit them.

Researchers in the domain of risk evaluation have tried not only to examine the influence of each of these factors on risk evaluation but also to identify the underlying risk dimensions. Their studies have yielded different risk structures, partly due to differences in the selections of risks being included, but among the dimensions that were frequently found are the number of people exposed, the familiarity of the risk, and the extent to which the outcome is dreaded (e.g., because it cannot be controlled or because it is fatal when it occurs; for a succinct review, see Breakwell, 2014, pp. 37–43). The familiarity dimension suggests that people may start out by judging new and unknown risks as unacceptable but gradually come to accept these risks as they develop a feeling of familiarity with them—even if they is no “objective” reason to assume that characteristics of the risk other than its subjective familiarity have changed (Lima, Barnett, & Vala, 2005).

Risk perception is supposed to be among the key determinants of risk, health, and safety behaviors. One debated issue in this context is whether people mainly act upon absolute risk perceptions (their perceptions of their own personal risks) or upon comparative risk perceptions (their perception of how their own personal risks compare with other people's risks).

The Affect Heuristic

As noted above, the dual-process approach suggests that people do not always engage in systematic likelihood estimations, subjective utility judgments, or judgments of other risk dimensions. Instead, they sometimes simply rely on how they feel toward a potential outcome or to the risky activity that entails the risk at hand. When the outcome provokes strong affect in them, they perceive the risk as large. When they hold favorable feelings toward an activity, they view the risks that are associated with the activity as small and/or acceptable and the benefits that are associated with it as large. When they have unfavorable feelings toward an activity, they view the risks that are associated with the activity as large and/or unacceptable and the benefits that are associated with it as small. This manner of appraising risks has been coined the “affect heuristic” (Finucane et al., 2000).

One characteristic of affect-based risk perception is that the qualitative (rather than quantitative) nature of feelings renders this type of risk perception relatively insensitive to the scope and likelihood of the relevant outcomes. Although the experiential system seems highly sensitive to the difference between the occurrence or nonoccurrence of an outcome, and to the

difference between certainty and uncertainty, it is rather insensitive to further variations in utility and likelihood. Extreme cases of probability neglect may therefore also be considered evidence of risk perceptions sometimes being affect-based rather than based on a likelihood-utility calculation (Hsee & Rottenstreich, 2004). In one study, for instance, participants were asked to indicate how much they would be willing to pay for a set of second-hand Madonna CDs. People in whom a “calculation mindset” had been induced were willing to pay significantly more for a 10-CD box than for a 5-CD box, whereas people in whom a “feeling mindset” had been induced were insensitive to the number of CDs that the box allegedly held (Hsee & Rottenstreich, 2004, Study 1).

One consequence of the “affect heuristic” is that people often view risks and benefits as being negatively correlated (with higher risks associated with small benefits and lower risks associated with large benefits). This perception is remarkable because risks and benefits are in many cases positively correlated. Activities that entail large potential benefits tend to carry large risks, and activities that carry no more than small risks often produce no more than limited benefits.

Another implication of the affect heuristic is that any intervention that changes how an activity makes people feel will affect the perceived magnitude of the risks that are associated with it, as well as the activity’s perceived benefits. Because this type of intervention may be related to the perceived benefits or to the magnitude of the risks themselves, enhancing the perceived benefits of an activity may reduce people’s perception of the risks associated with it, while reducing the perceived risks of the activity may enhance its perceived benefits. For instance, Finucane et al. (2000, Study 2) provided participants with information stressing either the high benefit, the low benefit, the low risk, or the high risk of various technologies (food additives, natural gas, nuclear power). Before and after reading the information, participants rated the benefits and the risks of the technologies. When the information said that the benefits of the technologies were high, it lowered participants’ ratings of the risks associated with these technologies. When the information said that the risks were low, it enhanced participants’ ratings of the technologies’ benefits. Information saying that the risks were low generally enhanced ratings of the technologies’ benefits.

Coda: Your Risk and Mine Are Not Created Equal

One intriguing phenomenon in risk perception through analytical processes is that systematic differences exist between people’s perceptions of their own risks and the risks of others. These differences occur both in perceptions of likelihood and in perceptions of outcome severity. Intriguingly, they seem to counteract each other. Likelihood estimations are characterized by comparative optimism, also called unrealistic optimism, with most people assuming that they are less likely than others to experience negative outcomes and more likely to experience positive events (for a brief introduction, see Shepperd, Waters, Weinstein, & Klein, 2015). Comparative optimism also occurs when people spontaneously imagine events that may happen in their or someone else’s future, rather than estimating the likelihood of outcomes that are presented to them (Hoorens, Smits, & Shepperd, 2008). Severity estimations, in contrast, seem characterized by comparative pessimism, with most people assuming that if negative outcomes happen to them, they will suffer more than others would (Blanton, Axsom, McClive, & Price, 2001). It is as yet an open question whether self-other differences characterize affect-driven risk perception as much as likelihood-utility-based risk perception.

Author Biography

Vera Hoorens is a full professor of social psychology in the Centre for Social and Cultural Psychology of Leuven University, Belgium. Her research in the field of risk perception and risk communication mainly focuses on perceived self-other differences in the likelihood and experience of life events. She also studies and publishes more broadly on social comparison, verbal communication, and the perception of groups and individuals. She has a professional guilty pleasure, namely, sixteenth-century history.

References

- Blanton, H., Axson, D., McClive, K. P., & Price, S. (2001). Pessimistic bias in comparative evaluations: A case of perceived vulnerability to the effects of negative life events. *Personality and Social Psychology Bulletin*, 27, 1627–1636. doi:10.1177/01461672012712006
- Breakwell, G. M. (2014). *The psychology of risk*. Cambridge, UK: Cambridge University Press.
- Fetherstonhaugh, D., Slovic, P., Johnson, S., & Friedrich, J. (1997). Insensitivity to the value of human life: A study of psychophysical numbing. *Journal of Risk and Uncertainty*, 14, 283–300. doi:10.1023/A:1007744326393
- Finucane, M. L., Alhakami, A., Slovic, P., & Johnson, S. M. (2000). The affect heuristic in judgments of risks and benefits. *Journal of Behavioral Decision Making*, 13, 1–17.
- Furnham, A., & Boo, H. C. (2011). A literature review of the anchoring effect. *The Journal of Socio-Economics*, 40, 35–42. doi:10.1016/j.soc.2010.10.008
- Hoorens, V., Smits, T., & Shepperd, J. A. (2008). Comparative optimism in the spontaneous generation of future life-events. *British Journal of Social Psychology*, 47, 441–451. doi:10.1348/014466607X236023
- Hsee, C. K., & Rottenstreich, Y. (2004). Music, pandas, and muggers: On the affective psychology of value. *Journal of Experimental Psychology: General*, 133, 23–30. doi:10.1037/0096-3445.133.1.23
- Jemmott, J. B., Ditto, P. H., & Croyle, R. T. (1986). Judging health status: Effects of perceived prevalence and personal relevance. *Journal of Personality and Social Psychology*, 50, 899–905. doi:10.1037/0022-3514.50.5.899
- Johnson, E. J., Hershey, J., Meszaros, J., & Kunreuther, H. (1993). Framing, probability distortions, and insurance decisions. *Journal of Risk and Uncertainty*, 7, 35–51. doi:10.1007/978-94-011-2192-7_3
- Kunreuther, H., Novemsky, N., & Kahneman, D. (2001). Making low probabilities useful. *Journal of Risk and Uncertainty*, 23, 103–120. doi:10.1023/A:1011111601406
- Lima, M. L., Barnett, J., & Vala, J. (2005). Risk perception and technological development at a societal level. *Risk Analysis*, 25, 1229–1239. doi:10.1111/j.1539-6924.2005.00664.x
- Nisbett, R. E., & Ross, L. (1980). *Human inference: Strategies and shortcomings of social judgment*. Englewood Cliffs, NJ: Prentice-Hall.
- Rottenstreich, Y., & Tversky, A. (1997). Unpacking, repacking, and anchoring: Advances in support theory. *Psychological Review*, 104, 406–415. doi:10.1037/0033-295X.104.2.406
- Shepperd, J. A., Waters, E. A., Weinstein, N. D., & Klein, W. M. P. (2015). A primer on unrealistic optimism. *Current Directions in Psychological Science*, 24, 232–237. doi:10.1177/0963721414568341
- Slovic, P. (2007). “If I look at the mass I will never act”: Psychic numbing and genocide. *Judgment and Decision Making*, 2, 79–95.
- Slovic, P., Monahan, J., & MacGregor, D. G. (2000). Violence risk assessment and risk communication: The effects of using actual cases, providing instruction, and employing probability versus frequency formats. *Law and Human Behavior*, 24, 271. doi:10.1023/A:1005595519944
- Sunstein, C. R. (2003). Terrorism and probability neglect. *Journal of Risk and Uncertainty*, 26, 121–136. doi:10.1023/A:1024111006336

- Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *Science*, 185, 1124–1131. doi:10.1126/science.185.4157.1124
- Van Boven, L., & Epley, N. (2003). The unpacking effect in evaluative judgments: When the whole is less than the sum of its parts. *Journal of Experimental Social Psychology*, 39, 263–269. doi:10.1016/S0022-1031(02)00516-4
- Weinstein, N. D. (2000). Perceived probability, perceived severity, and health-protective behavior. *Health Psychology*, 19, 65–74. doi:10.1037/0278-6133.19.1.65

Suggested Reading

- Breakwell, G. M. (2014). *The psychology of risk*. Cambridge, UK: Cambridge University Press.
- Finucane, M. L., Alhakami, A., Slovic, P., & Johnson, S. M. (2000). The affect heuristic in judgments of risks and benefits. *Journal of Behavioral Decision Making*, 13, 1–17.
- Kahneman, D. (2003). A perspective on judgment and choice: mapping bounded rationality. *American Psychologist*, 58, 697. doi:10.1037/0003-066X.58.9.697
- Slovic, P. E. (2000). *The perception of risk*. London, UK: Earthscan Publications.

Rumination

Andrew W. Manigault and Peggy M. Zoccola

Psychology Department, Ohio University, Athens, OH, USA

Interest in the role of rumination and related repetitive thought constructs in mental and physical illness and health has grown exponentially in the last several decades. Initial research focused on the role of ruminative thinking in the etiology and symptomatology of mental health disorders. More recently, researchers and clinicians have extended their focus to the physical health parameters associated with rumination, including physiological processes and biomarkers, somatic symptoms, and health-relevant behaviors. Overall, rumination can be considered a risk factor for a variety of detrimental mental and physical health outcomes. This entry first reviews several prominent theoretical models and definitions of rumination and then presents a conceptual model in an effort to organize the understanding of rumination's role in health. Afterward, we review the evidence for the relationship between rumination and health-relevant physiological systems and biomarkers, including blood pressure, cortisol, and immune functioning. The links between rumination and somatic symptoms, health behaviors, and other disease-relevant outcomes are also presented. The entry concludes with a discussion of intervention and treatment efforts aimed at reducing rumination and suggestions for future directions in research and practice.

Models and Definitions of Rumination

It is imperative that those who find themselves interested in rumination and its rich literature bear in mind that the meaning of rumination may change based upon theoretical context. Multiple theoretical models and definitions of rumination appear across disparate literatures. Among such theoretical models of rumination are the response styles theory, stress-reactive model, attentional scope model, and goal process theory. Although there are important distinctions between these models, as reviewed below, there are also important unifying dimensions and some conceptual overlap. For instance, many models highlight the repetitive nature of ruminative thought that is often past focused and characterized by negative valence or content.

One of the most prominent and widely adopted models of rumination is Nolen-Hoeksema's response styles theory. According to this theory, rumination is repetitive thinking about the "causes, consequences, and symptoms of one's negative affect" (p. 117; Smith & Alloy, 2009). Response styles theory posits that individuals who ruminate on the causes of their depressive symptoms tend to think more negatively, which consequently interferes with psychological functioning and maintains depressive symptoms. The theory also proposes that distraction from symptoms may alleviate the effects of rumination on depression. Supporting this account, some research shows that nondepressed individuals are more likely to experience a depressive episode if they report a tendency to ruminate over depressive symptoms relative to distraction from symptoms (Just & Alloy, 1997). Although response styles theory has received empirical support from a wide body of literature, some criticism persists. For example, rumination research grounded in this theory often makes use of questionnaires that have been criticized for their overlap with measures of depression, worry, and even positive forms of rumination (Smith & Alloy, 2009). Despite some criticism and mixed findings regarding the role of rumination in depressive symptoms, response styles theory remains one of the major approaches to health-related rumination research.

A theoretical extension of response styles theory is the stress-reactive model of rumination (Robinson & Alloy, 2003). According to this model, rumination, or repetitive thoughts pertaining to negative inferences, following a stressful life event puts individuals at risk for developing depression or having depressive episodes of longer duration. Thus, in this model, the content of rumination pertains specifically to thoughts (including negative affect) associated with a stressor. A minor departure from response styles theory, the stress-reactive model also captures ruminative thoughts before those that co-occur with negative affect. Although evidence supports the role of stress-reactive rumination in depression, this theoretical model of rumination has been criticized for being too specific (e.g., not capturing non-stressor-related ruminative thoughts; Smith & Alloy, 2009).

Inspired by cognitive findings, the attentional scope model of rumination focuses on the cognitive mechanisms of rumination that contribute to its repetitiveness and negative valence (Whitmer & Gotlib, 2013). This model suggests that negative mood can affect not only what thoughts are readily available but also how likely one is to exert control over these thoughts. Specifically, negative mood is thought to narrow one's attention such that it will be more difficult to inhibit the target of one's attentional focus or pay attention to novel stimuli. This model also makes a distinction between the cognitive impairments associated with dispositional rumination and state rumination. For example, although ruminative tendencies are associated with difficulties with inhibiting irrelevant information and updating working memory, state rumination seems to be associated with widespread deficits in cognitive control. A major advantage of this model is that it attempts to reconcile some of the seemingly contradictory findings of the rumination literature by discriminating between types of attention that may promote repetitive thinking and those that do not.

Other approaches to rumination have focused on the role of goals—in particular, frustrated and blocked goals—in prompting ruminative thinking. For example, goal process theory posits that ruminative thoughts emerge as a result of one's failure to progress toward a goal (Martin, Tesser, & McIntosh, 1993). In this framework, rumination is defined as a class of conscious thoughts concerning one's goal, which recur in the absence of immediate demands requiring the thoughts. This theory suggests that the experience or threat of failure is more important for rumination to occur than the feeling of sadness. A benefit of this approach is that it emphasizes conditions that lead to rumination as well as the cognitions that maintain it. Furthermore, goal process theory is more inclusive than other accounts;

stressful events and depressed mood may both constitute blocked goals and thus fit within this framework.

In sum, this brief review of rumination models highlights the need for a more comprehensive definition of rumination and perhaps more discriminatory use of terminology. Although the name may be shared, all types of rumination are not equivalent. The aforementioned conceptual models lack agreement with regard to the specific content and causes of rumination. Further, each model aims to predict different outcomes (e.g., response styles theory aims to predict the occurrence and duration of depressive episodes; the attentional scope model aims to identify the underlying cognitive mechanisms of rumination). Thus, they fail to provide a unified definition of rumination because they were never meant to do so. Despite theoretical discrepancies, it is important to note that the conceptual models reviewed here are not in full contradiction; across models, rumination is generally considered to be negatively valenced, repetitive, conscious, and past oriented. When considering rumination's relationship to psychological and physical health disorders, it may be useful to take a comprehensive conceptual approach while also stipulating how rumination differs from other related constructs (e.g., negative affect, sadness). A broader definition of rumination, or one that examines it across psychological disorders (and among individuals with and without psychopathology), may be valuable for identifying common causes, mechanisms, and thus points of intervention that have wider leverage.

Rumination and Health

The effects of rumination (broadly defined) extend beyond psychological processes. In particular, the link between rumination and physical health has been the subject of growing scientific interest. For example, the perseverative cognition hypothesis suggests that rumination may exacerbate or perpetuate physiological changes and symptoms that occur in response to stressors (Brosschot, Gerin, & Thayer, 2006). That is, normally occurring responses to stressors (e.g., rises in heart rate, blood pressure, and hormone secretion) may be accentuated or occur for longer periods of time if accompanied by ruminative thinking. Research on rumination and stress-related physiological parameters has yielded especially interesting results pertaining to stress recovery (i.e., a return to physiological resting states in the absence of the stressor). Given that poor cardiovascular recovery has been linked to serious health threats such as hypertension, some have proposed that the way in which one recovers from a stressful event is a better or at least equivalent predictor of long-term health outcomes than stress reactivity (i.e., the magnitude of the physiological changes that occur when the stressor is present). In other words, although it is adaptive to react in stressful situations, negative health outcomes may result from prolonged reactions in the absence of a stressor. Rumination may therefore impact health by maintaining the stress response beyond its adaptive range.

In addition, rumination may influence other cognitions, emotions, and behaviors that are critical for health and well-being. For example, if rumination maintains negative mood or depression, it may also negatively affect exercise and diet. In addition, rumination might also exacerbate the perception of negative symptoms (e.g., greater pain, poor sleep quality). In sum, the relationship between rumination and health is varied and operates through numerous pathways. A broad, conceptual overview of the effects of rumination on physical health is presented in Figure 1. The following section presents a non-exhaustive summary of studies that have tested some of the pathways outlined in this conceptual model linking rumination to physical health.

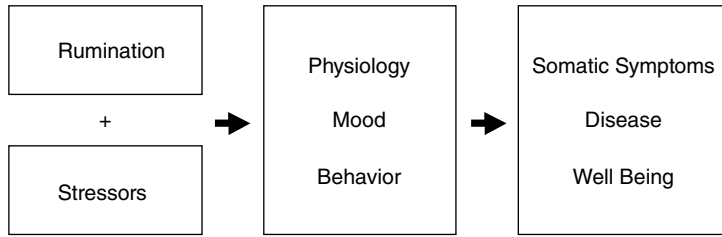


Figure 1 A conceptual model of the effects of rumination on physical health.

Rumination and Physiological Parameters

Autonomic Nervous System and Cardiovascular Activity

The study of the effect of rumination on autonomic and cardiovascular activity is robust and growing. Among others, angry, stress-related, and goal-dependent rumination measures have been examined in relation to blood pressure, heart rate, heart rate variability, and endothelin-1. Although there is some experimental and correlational evidence that rumination may lead to alterations in these parameters, empirical findings tend to vary depending on how rumination and cardiovascular outcomes are measured. Thus again, as is the case for definitions of rumination, the effect of rumination on cardiovascular outcomes depends to some degree on theoretical context. Such a relationship is logical given that any given definition of rumination may or may not capture relevant aspects of the construct relating to cardiovascular outcomes.

Several lines of research show that stress-related rumination predicts delayed cardiovascular recovery from stress. For example, undergraduates who report ruminating over a stressful arithmetic task take longer to return to baseline levels of blood pressure (Radstaak, Geurts, Brosschot, Cillessen, & Kompier, 2011). Rumination has also been linked to heart rate variability, or the variation in time between heartbeats, in some instances. Heart rate variability is a marker of physiological arousal that predicts cardiovascular health outcomes such as myocardial infarctions. In one study of heart rate variability, women without depressive rumination tendencies who engaged in state rumination following a stressful recall task had impaired heart rate variability recovery compared with those who did not ruminate following the task (Key, Campbell, Bacon, & Gerin, 2008). Thus, there is some consensus that stress-related and anger-related measures of rumination predict poor cardiovascular recovery.

Rumination may also affect other parameters that are important for cardiovascular health, including endothelin-1. Endothelin-1 is a vasoconstrictor, which can contribute to atherosclerosis, plaque rupture, and acute coronary events. For example, one study demonstrated that angry rumination tendencies predict greater increases in endothelin-1 in response to an anger recall task among individuals suffering from coronary heart disease (Fernandez et al., 2010). This finding seemingly holds after controlling for numerous other health covariates. Taken together, multiple lines of research point to an association between rumination and cardiovascular activation.

Neuroendocrine/Immune Activity

The relationship between rumination and health correlates extends to neuroendocrine and immune systems. Among other stress hormones, cortisol is often used in research because it is

easy to sample (e.g., via saliva) and affects the body in a variety of ways (e.g., metabolism, analgesia, immune/digestive/reproductive function). Cortisol reallocates resources from biological processes like digestion to prepare one's body to react in a stressful situation. Although adaptive in the short term, the effects of cortisol (e.g., suppressing non-specific immune responses) can be degenerative in the long term (e.g., increasing susceptibility to infections). Thus, demonstrating an effect of rumination on cortisol responses has important implications for physiological health.

The literature on the relationship between cortisol and rumination is relatively new and inconsistent, but growing. Inconsistent findings regarding this relationship can be attributed, at least in part, to variation in measures of rumination (e.g., stress related vs. depressive, state vs. trait) and types of stressor tasks used (e.g., social evaluative vs. nonevaluative; Zoccola & Dickerson, 2012). Nevertheless, recent findings provide strong evidence in favor of a causal link between stress-related rumination and cortisol recovery (Zoccola, Figueroa, Rabideau, Woody, & Benencia, 2014). In this experiment, participants were randomly assigned to a guided rumination or distraction condition following a stressful speech task. Participants who were guided through ruminative thinking produced higher concentrations of salivary cortisol 40 min and 1 hr after the stressful speech task relative to participants who were distracted. In sum, stress-related rumination may prolong cortisol exposure, but effects seem to depend on study design issues.

Rumination may also impact the immune system, but research on this topic is in its infancy. Using a sample of older adults, one study demonstrated that rumination is positively associated with several immune measures including leukocytes count, lymphocytes count, CD19⁺ B-lymphocyte count, and polyclonal T-cell activation (Thomsen et al., 2004). Given that high leukocyte counts are associated with increased risk of ischemic disease, rumination may negatively impact physical health via its effect on the immune system. Experimental evidence also links rumination to acute increases in inflammatory markers (Zoccola et al., 2014). Much more work is needed to confirm the role of rumination in influencing immune function.

Rumination and Somatic Complaints

The effects of rumination extend beyond physiology to symptom perception and somatic complaints. Mounting research suggests that rumination affects how one perceives, experiences, and reports health symptoms. Ruminative thinking may amplify the symptoms that one perceives and contribute to distress. For example, some research indicates that rumination is associated with health anxiety in college students, and this cognitive style predicts greater health anxiety above and beyond negative affect (as cited in Sansone & Sansone, 2012). Dispositional rumination also predicts increased somatic complaints (e.g., fatigue, nausea, and pain) in children and adolescents (as cited in Sansone & Sansone, 2012), as well as physical symptoms and overall subjective health ratings in young adults (Thomsen et al., 2004). Rumination has also been linked to hypochondriasis, which is characterized by a dysfunctional preoccupation that one has a severe illness despite medical reassurance to the contrary (as cited in Sansone & Sansone, 2012). Perceived and experienced somatic complaints can greatly influence quality of life and are highly relevant to health behaviors, such as treatment seeking and adherence, and health outcomes. Thus, the perception of symptoms can be as important as physiological processes with regard to health outcomes.

Within the pain perception framework in particular, rumination often has been integrated with other types of thoughts, such as feelings of helplessness and pessimism, which aggravate

symptom perception. The sum of these aggravating cognitions is referred to as “catastrophizing.” Pain catastrophizing is commonly measured with self-report questionnaires that include pain-focused rumination items or subscales. Several lines of research indicate that those who score higher on measures of pain catastrophizing report greater emotional distress, greater pain intensity, and more negative attitudes toward pain. Edwards, Bingham, Bathon, and Haythornthwaite (2006) suggest that catastrophizing leads to such outcomes by increasing attention to pain, amplifying pain processing in the central nervous system, and interfering with pain-coping behaviors.

When rumination is examined independently of the other components of pain catastrophizing, results are generally similar. For instance, findings from a recent experiment suggest that rumination is the best predictor of pain perception and distress in healthy adults (as cited in Sansone & Sansone, 2012). In this experiment, participants who scored high in pain-related rumination tendencies reported the greatest pain intensity resulting from inflation of a blood pressure cuff. In other studies, rumination predicted postoperative pain in athletes undergoing knee surgery, disability in chronic pain patients, pain levels in fibromyalgia and lower back pain patients, and psychological distress for palliative care patients (as cited in Sansone & Sansone, 2012). Thus, rumination and its related constructs (e.g., catastrophizing) are related to a wide spectrum of pain-related outcomes that are important for health and well-being.

Rumination has also been studied in relation to sleep. Sleep is a physiologic recuperative state that entails significant health repercussions when disturbed (e.g., cognitive impairments, depressed mood, impaired cardiovascular, neuroendocrine and immune function). Given that rumination is associated with an inability to disengage from irrelevant information, it is possible that those who tend to ruminate maintain high cognitive arousal before falling asleep and experience delayed sleep onset, insomnia, and lower-quality sleep. Results from several studies support this notion. For instance, one study in which participants were asked to report sleep quality and dispositional rumination demonstrated that rumination is negatively associated with sleep quality even after controlling for negative mood (Thomsen, Yung Mehlsen, Christensen, & Zachariae, 2003). In another study of college students, both state and trait measures of rumination may predict sleep onset latency, or time it takes to fall asleep (Zoccola, Dickerson, & Lam, 2009). Taken together, this body of work indicates that repetitive thinking generally predicts poor subjective sleep quality. Further, this relationship appears stable across types of ruminative thinking and thus constitutes an area of consensus for the rumination literature.

Health Behaviors

Logically, if ruminators experience more severe symptoms and are less likely to seek medical attention, then one should find empirical evidence for the relationship between rumination and healthcare utilization. Supporting this claim, Lyubomirsky, Kasri, Chang, and Chung (2006) found that individuals who ruminate may take longer than non-ruminators to seek help when experiencing symptoms of illness. In this study, women were asked to imagine finding a change in their breast and reported their intentions to seek medical attention. Women with ruminative tendencies were less likely to call a doctor immediately relative to women who do not ruminate. Furthermore, an independent sample of breast cancer survivors was asked to report the time lapse between noticing breast cancer symptoms and diagnosis. On average, women who ruminate took more than twice as long to visit a healthcare professional for breast cancer symptoms relative to non-ruminators. Given that the progression of a tumor into

metastatic cancer is partly a factor of time, seeking medical attention for signs of cancer early greatly increases one's chance of survival. Thus, for diseases such as cancer, rumination may increase the likelihood of fatalities by delaying diagnosis.

Although some evidence suggests that rumination negatively affects intent to seek medical attention and delays diagnosis, the relationship between rumination and health behaviors is not consistent across age groups and physical ailments. For instance, a study by Thomsen et al. (2004) showed that, among the elderly, the tendency to ruminate predicted telephone consultations with healthcare providers but is unrelated to in-person visits. Furthermore, rumination was unrelated to healthcare utilization in the young sample. The authors suggest that the positive relationship between rumination and health behaviors is more pronounced in individuals that are likely to need medical attention (e.g., the elderly). Further, given that rumination predicted phone call consultations but not in-person consultations in the elderly sample, rumination may lead individuals to seek reassurance from healthcare providers (e.g., are physical ailments indicative of serious problems?). As such, rumination may in some cases be beneficial. For specific populations, rumination appears to increase healthcare utilization. One should note that this area of research is still in infancy and thus does not allow clear conclusions regarding the relationship between rumination and healthcare utilization behaviors. Future research in this area would gain from investigating the interplay of relevant moderators.

Beyond healthcare utilization, rumination may also affect emotion-focused coping behaviors, which can be detrimental to physical health outcomes (e.g., drug use). In a longitudinal study, ruminating adolescents were more likely to misuse substances in response to a negative event relative to non-ruminators (Skitch & Abela, 2008). Additionally, individuals who experience work-related affective rumination generally report eating more unhealthy foods after work relative to non-ruminators (Cropley, Michalianou, Pravettoni, & Millward, 2012). Taken together, this area of research indicates that rumination may impact physical health by deterring healthcare utilization and promoting unhealthy coping behaviors.

Physical Illness and Disease

Rumination ultimately may play a role in disease development. As previously mentioned, there are various pathways through which rumination may contribute to physical illness and well-being. As a result of rumination, individuals may engage in fewer pro-health behaviors, exhibit increased and prolonged physiologically stress responses, experience aggravated symptoms leading to treatment delays or avoidance, sleep poorly, or engage in unhealthy coping behaviors. All of these possible effects of rumination entail consequences of their own, which are relevant to the progression of physical illness. For example, it is thought that the prolonged stress recovery associated with rumination may lead to weakened arteries, plaque buildup, and other risk factors of cardiovascular disease (Larsen & Christenfeld, 2009).

Some research supports a direct link between rumination and related repetitive thoughts (worry) and clinical disease-related outcomes (e.g., cancer, coronary heart disease). For example, worry—future-oriented repetitive thinking—is associated with coronary heart disease (Kubzansky et al., 1997). In a longitudinal study of over 2000 healthy men, participants reported at the beginning of the study the extent to which they worry. Over the subsequent 20 years, incidences of coronary heart disease and myocardial infarction were recorded. This study showed that men who tended to worry about social conditions were more likely to develop coronary heart disease and to experience a nonfatal myocardial infarction relative to those who reported little to no worry about social conditions. It is not clear from this finding

if worry can lead to coronary heart disease or if risk factors for coronary heart disease lead one to report more worry, hence this avenue of research deserves further inquiry.

Rumination may also play an important role in cancer prognosis. For example, Thomsen et al. (2013) found that in a sample of individuals who recently received a colon cancer diagnosis, rumination predicted increased depressive, intrusive, and avoidance symptoms at baseline and 8-week follow-up. These results further support the notion that rumination may negatively affect disease prognosis.

Prevention and Intervention

As reviewed above, the potential impact of rumination on health is broad and consequential. Thus, some researchers have focused on identifying means to reduce the extent to which individuals ruminate or engage in other perseverative cognitions. A recent meta-analysis by Querstret and Cropley (2013) suggests that both cognitive behavioral and mindfulness-based interventions may be effective in reducing both rumination and worry. Cognitive behavioral interventions focus on changing one's thinking style so that it may be more concrete, constructive, and active. Mindfulness-based interventions aim to teach coping skills such as meditation while promoting the generalization these skills (e.g., acceptance, present moment awareness) to daily experiences. A recent experiment shows that relative to an inactive control, women with cancer who underwent an 8-week mindfulness-based stress reduction program showed a decrease in self-reported rumination (Campbell, Labelle, Bacon, Faris, & Carlson, 2011). In sum, mindfulness-based and cognitive behavioral interventions may effectively reduce ruminative thinking.

Summary and Conclusions

As discussed in relation to psychopathology, biological parameters, somatic health, and illness, rumination may exacerbate or perpetuate the inherent components of each of these areas so as to result in negative health-related processes and outcomes. In addition to extending physiological responses beyond their adaptive range of action, rumination may magnify one's perception of poor health and thus aggravate psychological distress resulting from normally occurring symptoms. Via these pathways, rumination can promote pathogenesis and impair well-being.

Nevertheless, each of the pathways linking rumination to health entails important moderators; not all populations or measures of rumination are equivalent in relation to health outcomes. Specifically, the multitude of moderators that contribute to empirical findings linking rumination to health should not be ignored. A promising yet relatively untouched venue of research lies in a systematic review of such moderators (e.g., definitions of rumination, state vs. trait, age, gender). Although some outcomes have been reviewed across moderators (e.g., cortisol recovery), others have not (e.g., utilization of healthcare facilities). We encourage such research.

Specific to rumination definitions, researchers would benefit from identifying "active ingredients" of rumination contributing to its potentially negative impact on physical health. For example, the content (e.g., stress, anger, and sadness), temporal focus, intentionality (e.g., voluntary vs. involuntary), level of construal (abstract vs. concrete), and other characteristics of ruminative thinking may not contribute equally to its impact on health. Examining the correlates and consequences of rumination across multiple kinds of ruminative thinking and

multiple outcomes may be valuable for identifying common causes, mechanisms, and thus points of intervention that have wider leverage. For example, it is unclear if cognitive behavioral and mindfulness interventions are equally efficient in reducing stress-related, depressive, and angry rumination.

In sum, future rumination research should maximize generalizability while balancing the complex interplay of moderators that contribute to rumination's relationship to health. Rumination is worthwhile research subject due to its broad impact. As reviewed in the present entry, our everyday experiences, our physical health, and our well-being are at the mercy of our repetitive thoughts.

Author Biographies

Andrew W. Manigault is a doctoral student of experimental health psychology at Ohio University. Andrew Manigault studies the effects of mindfulness and other psychological factors on stress responses. His research focuses on the potential for mindfulness and meditation to affect the frequency of repetitive thoughts and ultimately physiological processes, with specific emphasis on the identifying underlying components of mindfulness, which may drive its positive health effects. He studies at Ohio University under the tutelage of Dr. Peggy Zoccola.

Peggy M. Zoccola is an assistant professor of psychology at Ohio University. Dr. Zoccola studies mechanisms contributing to persistent stress-related physiological activation and associated health consequences. Questions addressed by her research include the following: Do individuals who ruminate have increased cortisol and inflammation in response to psychosocial stressors? Are some individuals at increased risk for rumination? Are certain kinds of stressors or contexts more likely to elicit ruminative thinking and cortisol? How can we best measure or manipulate ruminative thought?

References

- Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, 60(2), 113–124. doi:10.1016/j.jpsychores.2005.06.074
- Campbell, T. S., Labelle, L. E., Bacon, S. L., Faris, P., & Carlson, L. E. (2011). Impact of Mindfulness-Based Stress Reduction (MBSR) on attention, rumination and resting blood pressure in women with cancer: A waitlist-controlled study. *Journal of Behavioral Medicine*, 35(3), 262–271. doi:10.1007/s10865-011-9357-1
- Cropley, M., Michalianou, G., Pravettoni, G., & Millward, L. J. (2012). The relation of post-work ruminative thinking with eating behaviour. *Stress and Health*, 28(1), 23–30. doi:10.1002/smi.1397
- Edwards, R. R., Bingham, C. O., Bathon, J., & Haythornthwaite, J. A. (2006). Catastrophizing and pain in arthritis, fibromyalgia, and other rheumatic diseases. *Arthritis Care & Research*, 55(2), 325–332. doi:10.1002/art.21865
- Fernandez, A. B., Soufer, R., Collins, D., Soufer, A., Ranjbaran, H., & Burg, M. M. (2010). Tendency to angry rumination predicts stress-provoked endothelin-1 increase in patients with coronary artery disease. *Psychosomatic Medicine*, 72(4), 348–353. doi:10.1097/PSY.0b013e3181d71982
- Just, N., & Alloy, L. B. (1997). The response styles theory of depression: Tests and an extension of the theory. *Journal of Abnormal Psychology*, 106(2), 221–229. <http://doi.org/10.1037/0021-843X.106.2.221>

- Key, B. L., Campbell, T. S., Bacon, S. L., & Gerin, W. (2008). The influence of trait and state rumination on cardiovascular recovery from a negative emotional stressor. *Journal of Behavioral Medicine*, 31(3), 237–248. doi:10.1007/s10865-008-9152-9
- Kubzansky, L. D., Kawachi, I., Spiro, A., Weiss, S. T., Vokonas, P. S., & Sparrow, D. (1997). Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the normative aging study. *Circulation*, 95(4), 818–824. doi:10.1161/01.CIR.95.4.818
- Larsen, B. A., & Christenfeld, N. J. S. (2009). Cardiovascular disease and psychiatric comorbidity: The potential role of perseverative cognition. *Cardiovascular Psychiatry and Neurology*, e791017. doi:10.1155/2009/791017
- Lyubomirsky, S., Kasri, F., Chang, O., & Chung, I. (2006). Ruminative response styles and delay of seeking diagnosis for breast cancer symptoms. *Journal of Social and Clinical Psychology*, 25(3), 276–304. doi:10.1521/jscp.2006.25.3.276
- Martin, L. L., Tesser, A., & McIntosh, W. D. (1993). Wanting but not having: The effects of unattained goals on thoughts and feelings. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 552–572). Englewood Cliffs, NJ: Prentice-Hall, Inc.
- Querstret, D., & Cropley, M. (2013). Assessing treatments used to reduce rumination and/or worry: A systematic review. *Clinical Psychology Review*, 33(8), 996–1009. doi:10.1016/j.cpr.2013.08.004
- Radstaak, M., Geurts, S. A. E., Brosschot, J. F., Cillessen, A. H. N., & Kompier, M. A. J. (2011). The role of affect and rumination in cardiovascular recovery from stress. *International Journal of Psychophysiology*, 81(3), 237–244. doi:10.1016/j.ijpsycho.2011.06.017
- Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research*, 27(3), 275–291. doi:10.1023/A:1023914416469
- Sansone, R. A., & Sansone, L. A. (2012). Rumination: Relationships with physical health. *Innovations in Clinical Neuroscience*, 9(2), 29.
- Skitch, S. A., & Abela, J. R. Z. (2008). Rumination in response to stress as a common vulnerability factor to depression and substance misuse in adolescence. *Journal of Abnormal Child Psychology*, 36(7), 1029–1045. doi:10.1007/s10802-008-9233-9
- Smith, J. M., & Alloy, L. B. (2009). A roadmap to rumination: A review of the definition, assessment, and conceptualization of this multifaceted construct. *Clinical Psychology Review*, 29(2), 116–128. doi:10.1016/j.cpr.2008.10.003
- Thomsen, D. K., Jensen, A. B., Jensen, T., Mehlsen, M. Y., Pedersen, C. G., & Zachariae, R. (2013). Rumination, reflection and distress: An 8-month prospective study of colon-cancer patients. *Cognitive Therapy and Research*, 37(6), 1262–1268. doi:10.1007/s10608-013-9556-x
- Thomsen, D. K., Mehlsen, M. Y., Hokland, M., Viidik, A., Olesen, F., Avlund, K., ... Zachariae, R. (2004). Negative thoughts and health: Associations among rumination, immunity, and health care utilization in a young and elderly sample. *Psychosomatic Medicine*, 66(3), 363–371.
- Thomsen, D. K., Yung Mehlsen, M., Christensen, S., & Zachariae, R. (2003). Rumination: Relationship with negative mood and sleep quality. *Personality and Individual Differences*, 34(7), 1293–1301. doi:10.1016/S0191-8869(02)00120-4
- Whitmer, A. J., & Gotlib, I. H. (2013). An attentional scope model of rumination. *Psychological Bulletin*, 139(5), 1036–1061. doi:10.1037/a0030923
- Zoccola, P. M., & Dickerson, S. S. (2012). Assessing the relationship between rumination and cortisol: A review. *Journal of Psychosomatic Research*, 73(1), 1–9. doi:10.1016/j.jpsychores.2012.03.007
- Zoccola, P. M., Dickerson, S. S., & Lam, S. (2009). Rumination predicts longer sleep onset latency after an acute psychosocial stressor. *Psychosomatic Medicine*, 71(7), 771–775. doi:10.1097/PSY.0b013e3181ae58e8
- Zoccola, P. M., Figueroa, W. S., Rabideau, E. M., Woody, A., & Benencia, F. (2014). Differential effects of poststressor rumination and distraction on cortisol and C-reactive protein. *Health Psychology*, 33(12), 1606–1609. doi:10.1037/hea0000019

Suggested Reading

- Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, 60(2), 113–124. doi:10.1016/j.jpsychores.2005.06.074
- Segerstrom, S. C., Stanton, A. L., Alden, L. E., & Shortridge, B. E. (2003). A multidimensional structure for repetitive thought: What's on your mind, and how, and how much? *Journal of Personality and Social Psychology*, 85(5), 909–921. doi:10.1037/0022-3514.85.5.909
- Smith, J. M., & Alloy, L. B. (2009). A roadmap to rumination: A review of the definition, assessment, and conceptualization of this multifaceted construct. *Clinical Psychology Review*, 29(2), 116–128. doi:10.1016/j.cpr.2008.10.003
- Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, 134(2), 163. doi:10.1037/0033-2909.134.2.163

Screening Behavior

Leona S. Aiken

Department of Psychology, Arizona State University, Tempe, AZ, USA

Medical screening refers to the identification of undetected disease or risk for disease among asymptomatic, apparently healthy individuals. The driving force in medical screening from a public health perspective is the reduction of morbidity and mortality through early detection and treatment. More broadly, the identification of risk for disease may lead to health behavior change. Of the 10 great public health achievements of 2001–2010 in the United States (Centers for Disease Control and Prevention, 2011), two encompass screening across the lifespan: life-saving treatment and intervention for newborns through “improvements in technology and endorsement of a uniform newborn-screening panel of diseases” (p. 620) and reduction in colorectal, female breast, and cervical cancer through improved cancer screening.

This entry targets the main social factors that relate to screening uptake, among them social norms, close interpersonal relationships, social networks, and racial/ethnic disparities. Genetic testing, with its impact on families, is considered. An overview of screening as a broad public phenomenon is provided to contextualize the review of social forces. The drive for universal and repeated screening emanating from health-related federal agencies and private medical organizations, operationalized through screening guidelines, is long standing. Yet recent writing in the medical community has raised questions about the risks versus benefits of screening, and ethical issues abound. These issues are included here as critical background for health psychology, as the field engages in both the study of screening behavior and in intervention research to increase screening. (A limitation was imposed on number of references per entry in this volume. A more complete reference list for this entry is available from the author.)

Varieties and Targets of Screening

Foci of screening are many and varied. *Genetic screening* has a long history. In newborns and infants, the earliest essentially universal genetic screening was and is for phenylketonuria (PKU), which results in severe mental retardation. Screening for PKU, begun in the 1960s, is now carried

out throughout the United States. Since PKU is treatable if detected very early, it is an exemplar of human biochemical genetics applied to health. Newborn infants in the United States are genetically screened for 29 disorders recommended by the American College of Medical Genetics. The US federal Newborn Screening Saves Lives Reauthorization Act of 2014 extends appropriations for infant genetic screening through 2019. Genetic screening of the fetus targets incurable conditions like cystic fibrosis and Huntington's disease. Genetic screening and counseling of future parents inform decisions about whether to have children in the face of possible genetic disease transmission. Screening of children and adults is available for multitude of genetic disorders that place one at high risk of developing a disease. Given sequencing of the genome and new technologies that make genetic analysis accessible, private companies now offer direct-to-consumer genetic screening for multiple genes that might have some implication for disease risk.

Cancer screening has been a central focus since the 1980s, when mammography screening became increasingly available and the National Cancer Institute and American Cancer Society recommended universal screening. Now melanoma and colorectal screening are public health targets; controversial prostate cancer screening is now in the public focus. Screening for a host of other conditions and diseases is available, among them alcohol and substance abuse cardiovascular disorders, metabolic, nutritional, and endocrine conditions, sexually transmitted diseases, and musculoskeletal disorders.

The US Preventive Services Task Force (USPSTF), convened by the Agency for Healthcare Research and Quality (AHRQ), provides evidenced-based recommendations for clinical preventive services, including over 60 screening tests. The USPSTF employs a grading system for screening test utility: A and B (recommends, high certainty of substantial/moderate net benefit), C (recommends selective provision for individual patients), D (recommends against, no net benefit or harms outweigh benefits), and I (insufficient current evidence). Only a third of reviewed screening tests reviewed receive A or B grades. The C grade places a substantial shared decision burden on physicians and patients.

Health Psychology and Screening

Health psychology has long been active in research on individual, social, and system-wide factors associated with screening uptake, as well as interventions to encourage screening for multiple diseases among diverse populations. This involvement dovetails with advances in medical screening methodologies. The *American Psychologist* (February–March 2015) devoted a complete issue to cancer and psychology, considering both traditional screening for common cancers and newer screening approaches for genetic risk. Psychology has been active for over 40 years in cancer screening for common cancers, including breast, cervical, colorectal, and prostate cancer (Wardle, Robb, Vernon, & Waller, 2015), but only little involved in newer genetic screening (McBride, Birmingham, & Kinney, 2015). Genetic screening raises psychological issues ranging from the decision to undergo screening to the impact of positive screening outcomes for both the individual and their relatives.

Intraindividual Factors and Screening: Perceived Risk and Attitudes

As previously indicated, this overview focuses mainly on social factors that influence screening uptake and social impacts associated with screening outcomes. Much health psychology-based research on both the correlates of screening uptake and interventions to increase screening

uptake has been driven by classic models of health behavior—the health belief model (HBM), the theories of reasoned action (TRA) and planned behavior (TPB), protection motivation theory (PMT), the health action process approach (HAPA), the transtheoretical model of change (TTM), and social cognitive theory (SCT). Overall, these models focus on intraindividual, mainly cognitive factors that relate to screening—perceived risk, attitudes (benefits, barriers), outcome expectancies, self-efficacy, and perceived behavioral control and planning.

Perceived Risk

Perceived risk for disease is a central driving force in screening uptake and a core component in interventions to increase screening. Health behavior models treat perceived risk for disease as a distal driving force in a chain of constructs that result in health protective behavior (Aiken, Gerend, Jackson, & Ranby, 2012). Aiken et al. (2012) provide an extensive treatment of the relationship of perceived risk to screening, both from the perspective of perceived risk as a correlate of screening and the role of perceived risk in interventions to increase screening.

Attitudes

Attitudes toward screening are represented by multiple constructs across health models, including perceived benefits versus barriers in the HBM and positive versus negative outcome expectations in the TRA/TPB. In general, attitudes constitute a more proximal factor than perceived risk in motivating health behavior. Many older women have been socialized into having regular cervical cancer screening as they became sexually active, into having regular mammograms beginning in their early 40s, and, along with men, into having colorectal screening by age 50. These individuals have already weathered winds of change in screening recommendations over the years—for example, the ongoing debate since the early 1990s as to whether women in their 40s should receive mammograms. For these individuals, ceasing screening requires an active decision, whereas continuing screening sustains their ongoing practice through adulthood (Torke, Schwartz, Holtz, Montz, & Sachs, 2013).

Many elderly individuals continue screening, valuing peace of mind and sensing a moral obligation to continue screening to care for their health. Physicians are more likely to discuss the pros than the cons of screening with older adults (Torke et al., 2013). In the United States, Medicare funding for senior healthcare includes regular mammograms and regular screening for cervical and vaginal, colorectal, and prostate cancer. In contrast, the USPSTF, which operates independent of US Department of Health and Human Services, recommends against PSA testing for prostate cancer, against routine colonoscopy above age 75, and against routine cervical screening over age 65 and reports insufficient evidence of benefits versus risks of mammography screening above age 75.

Barriers to Screening: An Overview

Myriad barriers to screening exist. In a review of barriers to mammography screening, Sarma (2015) provided a broadly applicable social ecological model to capture the interplay between the individual and the environment in relation to screening. The model employed a three-tier organization of barriers: healthcare system-level, social, and individual barriers. At the healthcare system level, nonadherence to screening recommendations was associated with lack of a healthcare provider (HCP) recommendation, lack of a usual HCP, and economic barriers,

including lack of insurance coverage, lack of access to screening services in terms of both transportation and insufficient facilities, difficulty in getting a mammogram appointment, and language barriers. Social barriers included lack of social support for screening from others and nonadherence to guidelines in the social network; broader cultural impacts included fatalism with regard to contracting and dying from cancer. On the individual level, a lack of accurate knowledge about screening, negative expectations about screening, including the receipt of a cancer diagnosis, distrust of the medical system, and competing priorities all were negatively associated with screening.

Social Factors and Screening

Collectively across health behavior models, social factors are far less well represented than intraindividual factors. Social factors represented in current models include subjective norms in TRA and TPB and socio-structural factors in SCT.

Social Norms and Screening

Broadly conceived, two classes of social norms operate on behavior: descriptive norms that characterize predominant behavior by others in a given situation and injunctive norms that reflect behavior that is approved versus disapproved by society. Injunctive norms are most often represented in screening literature as subjective norms in TRA/TPB—specifically, what significant others believe that a target individual should do, weighted by the target individual's wish to comply. How accurately subjective norms predict screening intention and actual screening uptake varies widely across studies. In a meta-analysis of prediction of screening by the TRA/TPB (Cooke & French, 2008), attitudes toward screening exhibited a stronger relationship to screening intention than subjective norms. In contrast, Smith-McLallen and Fishbein (2008) found subjective norms to be the strongest predictor of screening intentions, above and beyond attitudes and perceived behavioral control. One complexity of research on subjective norms and screening is that multiple sources of norms may be folded into a single scale of subjective norms, including the physician, friends and family, and, specifically, an individual's spouse; these sources relate differentially to screening uptake. In addition, screening behaviors are performed in private, in contrast to observable health protective behaviors like exercise, which are subject to social evaluation. For further discussion of norm theory in relation to health behavior, see Reid, Cialdini, and Aiken (2010).

Given the extensive literature on breast and cervical screening, much of what is known about cancer screening uptake is based on women. PsycINFO contains over five times the number of peer-reviewed articles on breast and cervical screening (beginning in 1958) than on prostate screening (beginning in 1994). What this literature collectively illustrates is the complexity of the relationship of subjective norms (i.e., injunctive norms of what significant others believe one should do). Influence from significant others has been observed to support screening uptake on the one hand but to induce reactance and resistance to screening on the other hand. In a study of men's uptake of colorectal and prostate screening, subjective norms that aggregated partners, families, and people in general were positively related to whether men had never, intermittently, or regularly been screened (Sieverding, Matterné, & Ciccarello, 2010). However, subjective norms were negatively related to screening in the next year among those who had previously never or intermittently been screened. This result was interpreted in terms of reactance to pressure for screening from significant others, particularly wives. This finding highlights the complexity of the relationship of injunctive norms to behavior.

The descriptive norm of what others actually do contributes to the prediction of screening intentions above and beyond injunctive norms (Smith-McLallen & Fishbein, 2008). Yet descriptive norms have complex relationships to behavior. While the descriptive norm of high perceived prevalence of a particular behavior may encourage the behavior, low perceived prevalence may well discourage behavior. Hence, social norms-based marketing that bemoans the fact that few people are engaged in an important health protective behavior (a common theme in public health messaging) actually discourages the behavior. For example, communication addressing the low prevalence of cancer screening discouraged men's intention for screening (Sieverding, Decker, & Zimmerman, 2010).

Spousal Influence on Screening

Spouses exhibit high concordance in health behaviors, attributed in part to assortative mating but also to mutual influence on health behavior change (see review by Jackson, Steptoe, & Wardle, 2015). Manne, Kashy, Weinberg, Boscarino, and Bowen (2012) noted substantial concordance between spouses in screening practices. They employed an interdependence model that characterized spouses' mutual influence on colorectal screening based on each spouse's perceived risk of colorectal cancer (CRC), perceived benefits of versus barriers to colorectal screening (i.e., decisional balance), and marital relationship quality.

Apparent spousal impact on screening uptake varies across populations. Among elderly participants in the national Medical Expenditure Panel Survey, for example, residing with one's spouse was positively associated with screening uptake (Lau & Kirby, 2009). Yet the complexity of the spousal association is evident in considering diverse populations. Among immigrant Latinas, for example, being married has been associated with higher uptake of routine medical checkups, a factor positively related to screening. In contrast among Latinas from a range of countries including Puerto Rico and Mexico and countries in Central and South America, attending an educational intervention on mammography and cervical screening with their husbands was associated with lower screening uptake, attributable to patriarchal control of women's health decisions (Shelton, Jandorf, Thelemaque, King, & Erwin, 2012).

Social Network and Screening

Social networks play a role in adult health (Martire & Franks, 2014). Research on social networks and screening addresses diverse populations, among whom Mexican Americans and Asians residing in the United States are major foci. Early on, social networks were positively associated with screening among older Mexican American women. Yet more recent research indicates weaker association of social integration with screening uptake that varies across Latina groups in the United States (Suarez et al., 2000). Among Vietnamese American women, who are at high risk for cervical cancer and have low screening rates, recommendation from friends for cervical screening was associated with increased screening (Nguyen-Truong et al., 2012). Structural characteristics of communities, specifically community cohesion characterized by residential stability and available community ties, are positively related to peer referrals for mammography (Southwell et al., 2010).

Genetic Screening

As characterized by the National Institutes of Health, genetic screening associated with disease is organized into six broad classes related to health: (a) newborn testing for disorders that are

treatable early in life, (b) diagnostic testing to confirm or disconfirm the presence of a genetic or chromosomal condition, (c) carrier testing to identify individuals who have a gene mutation that can be passed to offspring, (d) prenatal testing of the fetus in service of decision making about pregnancy continuation, (e) predictive and presymptomatic testing for diseases that may appear later in life, and (f) preimplantation testing of the embryo for procedures such as *in vitro* fertilization. There is a large expanding literature on factors associated with the decision to be screened (see review by Sweeny, Ghane, Legg, Huynh, & Andrews, 2014) and on the impact of test results on interpersonal relationships, particularly within the family.

The inheritance pattern of most cancer genetic mutations is autosomal dominant; there is a 50% risk of offspring inheriting the mutation if one parent is a carrier. Whether the disease itself is manifest in a carrier depends on gene penetrance. For BRCA1 and BRCA2 carriers, an oft-cited estimate of cumulative risk of breast cancer development is 57 and 49%, respectively, by age 70; for ovarian cancer, 40 and 18%, respectively (Chen & Parmigiani, 2007). Given high familial risk, family factors operate in both the decision to be screened for genetic mutations and in communication of screening results (see Katapodi, Northouse, Milliron, Liu, & Merajver (2013) for a summary). Discussion of genetic screening is positively associated with family cohesiveness and hardiness and negatively related to family conflict. Whether the decision to undergo screening is considered a personal or family matter differs across families.

Multiplex genetic susceptibility testing (MGST) is a form of genetic testing in which a single genetic sample is used to test for multiple gene variants that *may* alter the chances of developing particular diseases. A notion underlying such testing is that personalized medicine can be based on personal genetic profiles. “The future for the field is proffered as one in which individuals and health care providers might use genomic risk information to facilitate decision-making, personalize treatment approaches and motivate lifestyle improvements and adherence to screening recommendations” (Alford et al., 2011, p. 85). In 2007, the National Institutes of Health implemented the Multiplex Initiative to assess public response to genetic testing. Healthy young adults, all of whom had access to healthcare, were offered web-based information about genetic testing, free genetic multiplex testing for eight common diseases, and a personalized risk report. In all, 35% of those reached at baseline refused to participate; another 13% were ineligible. Only 6% of those initially reached at baseline actually received screening, having completed a complex multistep educational protocol required before screening. Consistent with previous findings concerning participation in genetic research, men had lower participation than women, and African Americans had lower participation than Whites (Alford et al., 2011).

A vision for health psychology is that advances in genomic technology, including understanding of epigenetic alterations of genetic material by environment and behavior, will lead to translational research that lowers the threat of genetically associated disease. McBride et al. (2015) identify three important research areas for health psychologists in the genetic testing sphere: (a) effective communication about gene–environment interactions, (b) provider–patient communication about the impact of genetic risk for disease development, and (c) design of behavioral risk-reduction interventions for those at genetic risk. The field of genetic counseling is already actively involved in these areas of research.

Social Stigma, Discrimination, and Screening

Social stigma may operate as a deterrent to screening or disclosure of screening—for example, screening for sexually transmitted diseases (Balfe & Brugh, 2010) and for blood diseases such

as hepatitis B transmitted via bodily fluid from an infected person. Earnshaw, Bogart, Dovidio, and Williams (2013) provide a comprehensive model to account for racial/ethnic disparities in HIV. While HIV testing among Blacks and Hispanics exceeds that for Whites, minority individuals are tested at a later stage of disease progression. Avoidance of testing is attributed to anticipated discrimination as a result of a positive HIV diagnosis, as well as to the expectation that the fact of being tested itself will lead to stigmatization.

DiMillo et al. (2015) identified distinct spheres of discreditable stigma (that is, stigma that is not immediately perceivable by others) that are produced by genetic screening. Women carrying the BRCA1 or BRCA2 gene feared that they would be viewed by others as weak and unable to carry out work responsibilities and that their children might suffer stigmatization due to their potential for also carrying the genetic mutation. Discrimination in the workplace and discrimination in access to insurance are two spheres of serious concern with regard to genetic testing. In the United States, the federal Genetic Information Nondiscrimination Act of 2008 (GINA) contains both employment and health insurance provisions but leaves much uncovered. Employment provisions apply only to companies with 15 or more employees. Access to life insurance, disability insurance, and long-term care insurance and the use of genetic test results to determine health insurance payments are not covered.

Racial/Ethnic Disparities

Racial/ethnic disparities are evident in screening uptake in the United States. According to the 2010 National Health Interview Survey (NHIS), almost identical percentages of White and Black women received mammograms (73% each) and Pap tests (83, 85%, respectively) in accord with USPSTF recommendations. Hispanic women lagged behind both White and Black women for mammograms (70%) and Pap tests (79%). Overall, Asian women fell substantially behind White, Black, and Hispanic women for mammograms (64%) and Pap tests (75%). CRC screening uptake was higher among Whites than Blacks (60% versus 55%) and substantially lower among Hispanics and Asians (47% each). For all tests, having health insurance, higher education, a usual source of healthcare, and being US born were associated with higher screening rates. Among immigrants, being in the United States for fewer than 10 years was associated with lower screening (Centers for Disease Control and Prevention, 2012; see also Wardle et al., 2015).

Physician Recommendation

Physician recommendation is a gatekeeper for access to screening, among the most powerful correlates of screening uptake. Physician recommendation is also a marker variable for barriers to screening, including lack of health insurance and lack of access to regular healthcare. Physician recommendation has been scrutinized as a putative cause of lower screening rates among racial/ethnic minorities. Discrepancies in rate of physician recommendation across racial/ethnic groups vary across studies, and close attention to the databases and case selection on which outcomes are based is warranted. The NHIS has been employed over time to assess differences in physician referral for screening tests as a function of race/ethnicity. Based on the 2005 NHIS, Wallace, Baltrus, Wallace, Blumenthal, and Rust (2013) reported a lower rate of physician recommendation for colorectal screening among Black than among White study participants, all of whom had no family history of CRC. With age, gender, education, income,

health insurance, and number of doctor visits in the past year controlled, the probability of recommendation was less than half for Blacks than for Whites.

Current Issues in Screening

Screening research in health psychology has for decades followed the medical community's endorsement of screening, associated with ever-refined technology for detection of disease before it is manifested in observable symptoms. Health psychology has researched putative determinants of and deterrents to screening uptake and has developed and evaluated interventions to increase screening rates. Yet now a new theme has emerged in the international medical community: that some screening procedures have health risks and screening yields false positives, over-diagnosis, and over-treatment for diseases that may never become life threatening.

The screening message to the public has been that early detection leads to better treatment outcomes and longer survival. Yet lead-time bias in screening refers to the detection of disease earlier than it would be diagnosed as a function of symptoms, while the course of disease remains the same. Thus, oft-cited 5-year survival rates after detection are not an accurate measure of the impact of screening on saving lives. In the current medical climate, joint decision making between physician and patient is the model. There are great emotional issues in screening decisions (Rosenbaum, 2014) coupled with a call for greater understanding of the interplay between affective science and cancer control (Ferrer, Green, & Barrett, 2015). Genetic screening brings a host of ethical issues—informed consent, ownership of genetic samples, disclosure of genetic findings, the impact of genetic screening on access to health insurance, and discrimination in the workplace. These issues are made more complex by direct-to-consumer marketing. The concern with over-screening is reflected in a recent funding initiative the National Cancer Institute to reduce over-screening older adults for breast, cervical, and colorectal cancers. In sum, as health psychology continues to participate in screening research, the complexity of screening from individual, public health, social, and economic perspectives appears to increase exponentially over time.

Author Biography

Leona S. Aiken is president's professor of psychology at Arizona State University (ASU); she chairs the PhD concentration in quantitative methods in psychology. She is coauthor of *Multiple Regression: Testing and Interpreting Interactions* and of *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences*. Her methodological work considers measured and latent continuous variable interactions. Her substantive research focuses on women's health protective behaviors across the lifespan. She is past president of the Society of Multivariate Experimental Psychology.

References

- Aiken, L. S., Gerend, M. A., Jackson, K. M., & Ranby, K. W. (2012). Subjective risk and health protective behavior. In A. Baum, T. Revenson, & J. Singer (Eds.), *Handbook of health psychology* (2nd ed., pp. 113–145). New York, NY: Taylor & Francis.

- Alford, S. H., McBride, C. M., Reid, R. J., Larson, E. B., Baxevanis, A. D., & Brody, L. C. (2011). Participation in genetic testing research varies by social group. *Public Health Genomics*, 14, 85–93. doi:10.1159/000294277
- Balfe, M., & Brugha, R. (2010). Disclosure of STI testing activities by young adults: The influence of emotions and social networks. *Sociology of Health & Illness*, 32, 1041–1058. doi:10.1111/j.1467-9566.2010.01281.x
- Centers for Disease Control and Prevention. (2011). Ten great public health achievements—United States, 2001–2010. *Morbidity and Mortality Weekly Report*, 60, 619–623.
- Centers for Disease Control and Prevention. (2012). Cancer screening—United States, 2010. *Morbidity and Mortality Weekly Report*, 61, 41–45.
- Chen, S., & Parmigiani, G. (2007). Meta-analysis of BRCA1 and BRCA2 penetrance. *Journal of Clinical Oncology*, 25, 1329–1333. doi:10.1200/JCO.2006.09.1066
- Cooke, R., & French, D. P. (2008). How well do the theory of reasoned action and planned behavior predict intentions and attendance at screening programmes? A meta-analysis. *Psychology and Health*, 23, 745–765. doi:10.1080/08870440701544437
- DiMillo, J., Samson, A., Thériault, A., Lowry, S., Corsini, L., Verma, S., & Tomiak, E. (2015). Genetic testing: When prediction generates stigmatization. *Journal of Health Psychology*, 20, 393–400. doi:10.1177/1359105313502566
- Earnshaw, V. A., Bogart, L. M., Dovidio, J. F., & Williams, D. R. (2013). Stigma and racial/ethnic HIV disparities: Moving toward resilience. *American Psychologist*, 68, 225–236. doi:10.1037/a0032705
- Ferrer, R. A., Green, P. A., & Barrett, L. F. (2015). Affective science perspectives on cancer control: Strategically crafting a mutually beneficial research agenda. *Perspectives on Psychological Science*, 10, 328–345. doi:10.1177/1745691615576755
- Jackson, S. E., Steptoe, A., & Wardle, J. (2015). The influence of partner's behavior on health behavior change. *JAMA Internal Medicine*, 175, 385–392. doi:10.1001/jamainternmed.2014.7554
- Katapodi, M. C., Northouse, L. L., Milliron, K. J., Liu, G., & Merajver, S. D. (2013). Individual and family characteristics associated with BRCA1/2 genetic testing in high-risk families. *Psycho-Oncology*, 22, 1336–1343. doi:10.1002/pon.3139
- Lau, D. T., & Kirby, J. B. (2009). The relationship between living arrangement and preventive care use among community-dwelling elderly persons. *American Journal of Public Health*, 99, 1315–1321. doi:10.2105.AJPH.2008.151142
- Manne, S., Kashy, D., Weinberg, D. S., Boscarino, J. A., & Bowen, D. J. (2012). Using the interdependence model to understand spousal influence on colorectal cancer screening intentions: A structural equation model. *Annals of Behavioral Medicine*, 43, 320–329. doi:10.1007/s12160-012-9344-y
- Martire, L. M., & Franks, M. M. (2014). The role of social networks in adult health: Introduction to the special issue. *Health Psychology*, 33, 501–504. doi:10.1037/hea0000103
- McBride, C. M., Birmingham, W. C., & Kinney, A. Y. (2015). Health psychology and translational genomic research: Brining innovation to cancer-related behavioral interventions. *American Psychologist*, 72, 91–104. doi:10.1037/a0036568
- Nguyen-Truong, C. K. Y., Lee-Lin, F., Leo, M. C., Gedaly-Duff, V., Nail, L. M., Wang, P., & Tran, T. (2012). A community-based approach to understanding pap testing adherence among vietnamese American Immigrants. *Journal of Obstetrics, Gynecologic, and Neonatal Nursing*, 41, E26–E40. doi:10.1111/j.1552-6909.2012.01414.x
- Reid, A. E., Cialdini, R. B., & Aiken, L. S. (2010). Social norms and health behavior. In A. Steptoe (Ed.), *Handbook of behavioral medicine research: Methods and application* (pp. 263–275). New York, NY: Springer. doi:10.1007/978-0-387-09488-5
- Rosenbaum, L. (2014). Invisible risks, emotional choices: Mammography and medical decision making. *New England Journal of Medicine*, 371, 1549–1552. doi:10.1056/NEJMrns1409003
- Sarma, E. A. (2015). Barriers to screening mammography. *Health Psychology Review*, 9, 42–62. doi:10.1080/17437199.2013.766831
- Shelton, R. C., Jandorf, L., Thelemaque, L., King, S., & Erwin, D. O. (2012). Sociocultural determinants of breast and cervical cancer screening adherence: An examination of variation among

- immigrant Latinas by country of origin. *Journal of Health Care for the Poor and Underserved*, 23, 1768–1792. doi:10.1353/hpu.2012.0191
- Sieverding, M., Decker, S., & Zimmermann, F. (2010). Information about low participation in cancer screening demotivates other people. *Psychological Science*, 21, 941–943. doi:10.1177/0956797610373936
- Sieverding, M., Mattern, U., & Ciccarello, L. (2010). What role do social norms play in the context of men's cancer screening intention and behavior? Application of an extended theory of planned behavior. *Health Psychology*, 29, 72–81. doi:10.1037/a0016941
- Smith-McLallen, A., & Fishbein, M. (2008). Predictors of intentions to perform six cancer-related behaviours: Roles for injunctive and descriptive norms. *Psychology, Health & Medicine*, 13, 389–401. doi:10.1080/13548500701842933
- Southwell, B. G., Slater, J. S., Rothman, A. J., Friedenberg, L. M., Allison, T. R., & Nelson, C. L. (2010). The availability of community ties predicts likelihood of peer referral for mammography: Geographic constraints on viral marketing. *Social Science and Medicine*, 71, 1627–1635. doi:10.1016/j.socscimed.2010.08.809
- Suarez, L., Ramirez, A. G., Villarreal, R., Marti, J., McAlister, A., Talavera, G. A., ... Perez-Stable, E. J. (2000). Social networks and cancer screening in four U.S. Hispanic groups. *American Journal of Preventive Medicine*, 19, 47–52.
- Sweeny, K., Ghane, A., Legg, A. M., Huynh, H. P., & Andrews, S. E. (2014). Predictors of genetic testing decisions: A systematic review and critique of the literature. *Journal of Genetic Counseling*, 23, 263–288.
- Torke, A. M., Schwartz, P. H., Holtz, L. R., Montz, K., & Sachs, G. A. (2013). Older adults and forgoing cancer screening. *JAMA Internal Medicine*, 173, 526–531. doi:10.001/jamainternmed.2013.2903
- Wallace, D. A. C., Baltrus, P. T., Wallace, T. c., Blumenthal, D. S., & Rust, S. G. (2013). Black white disparities in receiving a physician recommendation for colorectal cancer screening and reasons for undergoing screening. *Journal of Health Care for the Poor and Underserved*, 24, 1115–1124. doi:10.1353/hpu.2013.0132
- Wardle, J., Robb, K., Vernon, S., & Waller, J. (2015). Screening for prevention and early diagnosis of cancer. *American Psychologist*, 70, 119–133. doi:10.1037/a0037357

Suggested Reading

- Fairfield, K. M., Gerstein, B. S., Levin, C. A., Stringfellow, V., Wierman, H. R., & McNaughton-Collins, M. (2015). Decisions about medication use and cancer screening across age groups in the United States. *Patient Education and Counseling*, 98, 338–343. doi:10.1016/pec.2014.11.012
- Kiviniemi, M. T., Bennett, A., Zaiter, M., & Marshall, J. R. (2011). Individual-level factors in colorectal cancer screening: A review of the literature on the relation of individual-level health behavior constructs and screening behavior. *Psycho-Oncology*, 20, 1023–2033. doi:10.1002/pon.1865
- Marquez, B., Elder, J. P., Arredondo, E. M., Madanat, H., Ji, M., & Ayala, G. X. (2014). Social network characteristics associated with health promoting behaviors among Latinos. *Health Psychology*, 33, 544–553. doi:10.1037/HEA0000092
- Schultz, P. W., Nolan, J. M., Cialdini, R. B., Goldstein, N. J., & Griskevicius, V. (2007). The constructive, destructive, and reconstructive power of social norms. *Psychological Science*, 28, 429–434. doi:10.1111/j.1467.9280.2007.01917.x
- Ye, J., Williams, S. D., & Xu, Z. (2009). The association between social networks and colorectal cancer screening in American males and females: Data from the 2005 Health Information National Trends Survey. *Cancer Causes and Control*, 20, 1227–1233. doi:10.1007/s10552-009-9335-x

Selective Exposure in the Domain of Health

Zachary Fetterman and William Hart

Psychology Department, University of Alabama, Tuscaloosa, AL, USA

Consider the following scenario. Carl was diagnosed with severe sleep apnea. Carl's doctor suggested a treatment called continuous positive airway pressure (CPAP) to manage his condition. Carl had heard anecdotally that this treatment was uncomfortable and ineffective, and he had a preferred treatment in mind prior to receiving his doctor's recommendation. His doctor suggested he read more about his options and provided Carl with some reading material on the benefits of CPAP and the possible shortcomings of his preferred non-recommended treatment. Carl never read this information. Instead, Carl sought out Internet sources that validated his preexisting preference. Carl continued to stubbornly believe that he knew the best treatment despite his doctor's advice. From Carl's perspective, he had done extensive research on the issue, and the research clearly confirmed his views on treatment. In the end, the interaction between Carl and his doctor ended without a resolution. Unfortunately, untreated sleep apnea is associated with increased mortality risk (Young et al., 2008). It is possible that Carl could have experienced a healthier and longer life if he had been more open and willing to expose himself to alternative views about available treatment options.

Carl's actions reflect a common human behavior termed *selective exposure*. Selective exposure refers to the tendency to selectively seek out viewpoint supportive information and neglect viewpoint inconsistent information (Festinger, 1957; Fischer & Greitemeyer, 2010; Hart et al., 2009). It ensures that people will know more about why they might be right than why they might be wrong. Reviews of selective exposure suggest that this effect can be large (Fischer & Greitemeyer, 2010; Frey, 1986; Hart et al., 2009) and takes place in various domains including health, consumer decision making, politics, morality and values, and business decision making (Knobloch-Westerwick, Johnson, & Westerwick, 2013). Selective exposure is similar to but distinguishable from two related phenomena. First, selective exposure refers to the relative preference for supportive over conflicting information, which distinguishes it from mere information avoidance. Second, the selective exposure of interest here is

driven by people's preferences for supportive over conflicting information, which distinguishes it from selective exposure driven by the availability of information in one's environment (a phenomenon called *de facto selective exposure*).

In this entry we will focus on selective exposure in the domain of health, but we will often refer to research without a clear health outcome that nonetheless sheds light on the mechanisms that guide selective exposure. Our assumption is that these mechanisms also operate in health-related selective exposure. Ultimately, understanding selective exposure and its manifestations in the context of health may allow scientists and practitioners to contribute to broad efforts to improve the health and well-being of both individuals and populations.

Selective Exposure and Health

Evidence suggests that people might selectively gather information that supports (vs. contradicts) their health choices. In one of the earliest studies relating selective exposure to health (Brock & Balloun, 1967), participants were asked to listen to recordings of health messages related to the smoking–cancer link; however, these messages were obscured by static. Participants were told that they could remove the static and more clearly hear the messages by repeatedly pressing a button. Smokers pressed the button more than nonsmokers to remove the static from a message discrediting the smoking–cancer link, and nonsmokers pressed the button more than smokers to remove static from a message validating the smoking–cancer link. Thus, people appear to have strategically clarified the message only when it would validate their own smoking-related behavior.

A more recent study assessed the relationship between participants' tendency to engage in various health behaviors (consumption of organic food, coffee, and vegetables as well as exercise habits) and the time they spent reading articles in support of or opposition to each of these various health behaviors (Knobloch-Westerwick et al., 2013). Across all studied health behaviors, the frequency of engaging in a behavior was positively associated with time spent reading information supporting (vs. challenging) that behavior. Other research in more naturalistic settings confirms the role of selective exposure in the health domain (e.g., Noguchi, Albarracín, Durantini, & Glasman, 2007). In one unfortunate example, individuals who are most at risk for contracting HIV appear to be the least willing to attend HIV intervention programs (Noguchi et al., 2007).

Although evidence suggests that selective exposure sometimes takes place in health decision making (Hart et al., 2009; Knobloch-Westerwick et al., 2013), the observed effects can be small. Presumably, health-related selective exposure effects might be dependent on the presence of personal and situational variables that bolster or weaken selective exposure. Next, we discuss the processes that seem to influence selective exposure and how these processes might be at work in health-related decision-making scenarios.

Mechanisms and Moderators

Feeling Good or Avoiding Feeling Bad

One long-standing assumption surrounding selective exposure is that it manifests, at least in part, as a means to make people feel good or avoid feeling bad. Indeed, early research conceptualized selective exposure as a way to cope with or avoid decisional remorse (i.e., dissonance;

e.g., Festinger 1957, 1964). By selectively seeking congenial information and avoiding uncongenial information, individuals can effectively reduce the unpleasantness of feeling foolish (e.g., “I made a stupid choice!”) and promote the pleasantness of feeling wise (e.g., “I made a smart choice!”). Research reveals that selective exposure tends to be amplified when invalidation might particularly sting and validation might particularly satisfy (Fischer & Greitemeyer, 2010; Frey, 1986; Hart et al., 2009). As a few examples, selective exposure is amplified when individuals (a) are highly committed to an attitude or behavior (Jonas & Frey, 2003); (b) are dealing with information relevant to enduring, personally defining values (McFarland & Warren, 1992); (c) are reminded of their mortality (Jonas, Greenberg, & Frey, 2003); (d) are forced to choose information that is difficult to refute (Frey, 1986); (e) do not feel self-affirmed (Klein & Harris, 2009) or are placed in a negative mood (Jonas, Graupmann, & Frey, 2006); and (f) care a great deal about cognitive consistency (Hart, Adams, Burton, Shreves, & Hamilton, 2012).

These findings provide insight into the factors that might influence selective exposure in health-related decision-making scenarios. As a few examples, health-related decision-making scenarios that evoke negative moods (Penner et al., 2010), prompt concerns about one’s own mortality (e.g., a health diagnosis that makes death salient; Bozo, Tunca, & Šimšek, 2009), or challenge a person’s autonomy to make their own choices (e.g., a pushy healthcare provider; Fitzsimons & Lehmann, 2004) should be particularly likely to inspire defensive attempts to validate one’s own health views. In these scenarios, addressing defensiveness is likely necessary to ensure evenhanded information gathering. Fortunately, research has found that defensiveness can be tempered, and selective exposure reduced, by validating a person’s autonomy and sense of self-worth (Howell & Shepperd, 2012).

High Confidence

An additional mechanism proposed to underlie selective exposure is confidence. Various studies converge on the idea that increased decisional certainty, or confidence in one’s attitudes, beliefs, and behaviors, is associated with an increased tendency to engage in selective exposure (Fischer, Fischer, Weisweiler, & Frey, 2010). For example, in one experiment (Fischer et al., 2010), some participants were led to feel reduced confidence in their decision because they made their decision under suboptimal, highly distracting conditions. Other participants were allowed to make an immediate decision before any distraction or were able to deliberate in a distraction-free environment. Participants in the distracted group, relative to participants in the other two non-distracted groups, showed less confidence in their decision and reduced selective exposure.

There are at least two reasons why high confidence might enhance selective exposure. First, it could be the case that highly confident individuals are more motivated to maintain their views than are less confident individuals (Frey, 1986). For highly confident individuals, confronting information that suggests the need for a change in attitudes, beliefs, or behaviors is likely to be perceived as counterproductive (e.g., “If it ain’t broke, don’t fix it!”: Frey, 1986). Second, it could be the case that highly confident individuals are prone to misperceive supportive information as higher in quality than unsupportive information (Fischer & Greitemeyer, 2010). Imagine feeling extremely certain that refined sugar consumption is linked with obesity. You are likely to perceive any information that denies this link as invalid (e.g., ignorant, biased). Conversely, you are likely to perceive information that supports the link as reliable and possibly worth a read.

The confidence mechanism contributing to selective exposure seems particularly important to consider in the context of health-related information. Research suggests that people tend to

be overconfident about their knowledge of various health topics (Weinstein & Lyon, 1999). In light of this overconfidence, it may be particularly important to address individuals' overconfidence and accordingly minimize selective exposure.

Cognitive Economy or Laziness

Selective exposure may also be motivated by efficiency. Information search is often conducted with a desire to conserve energy, and selecting supportive information is generally an effective way to preserve cognitive resources (Fischer, 2011). Disconfirming information is more difficult to process because it is harder to integrate into one's existing knowledge structures (Ditto & Lopez, 1992). Consistent with this idea, robbing people of energy increases their tendency to demonstrate selective exposure. In one study on this issue (Fischer, Greitemeyer, & Frey, 2008), participants either completed an easy task (watching a video of a politician speaking with words occasionally appearing on the bottom of the screen) or an energy-zapping version of the same task (watching the same video but with the instructions not to look at the words and to regularly redirect their attention from the words to the politician). All participants then indicated views on political and economic issues. As anticipated, participants who completed the energy-zapping task demonstrated increased selective exposure relative to those who did the easy task. Other research suggests that personal factors linked to "cognitive miserliness" predict enhanced selective exposure (Hart et al. 2009, Hart, Adams, Burton, Shreves, & Hamilton, 2012).

Selective exposure as a result of cognitive economy may be particularly prevalent in patient populations. Research shows that individuals with various health conditions report increased fatigue relative to controls (Stone, Richards, A'Hern, & Hardy, 2000). Consider this correlation in relation to our scenario involving Carl. Carl was suffering from sleep apnea and presumably often fatigued. This diminished energy should, in theory, make Carl even more motivated than a well-rested individual to preserve cognitive resources and thus engage in selective exposure.

A related barrier that potentially perpetuates selective exposure in the domain of health is the public's regular exposure to competing health information. Nutritional science, for example, often offers contradictory suggestions (e.g., high protein, low-carb diet vs. vegetarian diet vs. balanced diet based on moderation). In light of this lack of consensus, individuals may feel inclined to preserve their energy by avoiding exhaustive, potentially unproductive information search and instead indulge themselves in confirmatory messages.

Accuracy Motivation (Wanting to be Right)

Accuracy motivation refers to a desire to accurately appraise stimuli, reach correct conclusions, and discern truth independent of whether these appraisals and conclusions are consistent with existing thoughts, attitudes, and behaviors. Research shows that increases in accuracy motivation tend to reduce selective exposure because they can weaken the three mechanisms that promote this biased information gathering. For example, factors that enhance concerns for accuracy have been shown to encourage people to forsake immediate hedonic satisfaction (gained through selective exposure) for more long-term satisfaction that comes from making the most informed choice (Jonas & Frey, 2003), to question their preexisting confidence (Fischer, Greitemeyer, et al., 2008), and to inspire greater willingness to expend energy processing new information (Fischer, 2011).

Available research suggests strategies that may effectively induce accuracy concerns in health domains and reduce selective exposure. For example, encouraging people to confront the discrepancy between what they are doing and what they should be doing seems to inspire

more evenhanded gathering of health information. In one study (Knobloch-Westerwick et al., 2013), participants who perceived a larger discrepancy between their stated health behaviors and their health goals showed a reduced tendency to selectively prefer information that supported (vs. challenged) their current behavior. Additionally, existing research suggests that accuracy motivation may be ignited by encouraging individuals to consider higher-order goals (Fischer, Jonas, Frey, & Schulz-Hardt, 2005; Fischer, Schulz-Hardt, & Frey, 2008). For example, encouraging individuals to consider their desirable health-related goals (e.g., achieve a state of health that allows for increased experience of family, fun, and leisure) prior to information gathering may promote more evenhanded information gathering.

Conclusion

Selective exposure can have deleterious consequences for health decision making (Noguchi et al., 2007). Failure to fully consider contradictory evidence about one's treatment options, diet alternatives, views on exercise, and ideas about sleep could result in poor health literacy as well as suboptimal health choices and outcomes. Evidence suggests that selective exposure may occur in health-related decision making, and it seems likely that the extent of this bias is influenced by at least four mechanisms: (a) wanting to feel good or avoid feeling bad, (b) being highly confident, (c) desiring cognitive economy, and (d) desiring accuracy.

Author Biographies

Zachary Fetterman is a doctoral candidate at the University of Alabama, currently completing clinical internship at Wilford Hall Ambulatory Surgical Center. His interests include bias at individual and system levels.

William Hart is an assistant professor in social psychology at the University of Alabama. Will received his PhD from the University of Florida in 2009. He is interested in judgment and decision making and goal pursuit.

References

- Bozo, Ö., Tunca, A., & Şimşek, Y. (2009). The effect of death anxiety and age on health-promoting behaviors: A terror-management theory perspective. *The Journal of Psychology, 143*(4), 377–389. doi:10.3200/JRLP.143.4.377-389
- Brock, T. C., & Balloun, J. L. (1967). Behavioral receptivity to dissonant information. *Journal of Personality and Social Psychology, 6*(4), 413–428. doi:10.1037/h0021225
- Ditto, P. H., & Lopez, D. F. (1992). Motivated skepticism: use of differential decision criteria for preferred and nonpreferred conclusions. *Journal of Personality and Social Psychology, 63*(4), 568–584. doi:10.1037/0022-
- Festinger, L. (1957). *A theory of cognitive dissonance*. Stanford, CA: Stanford University Press.
- Festinger, L. (1964). *Conflict, decision, and dissonance*. Stanford, CA: Stanford University Press.
- Fischer, P. (2011). Selective exposure, decision uncertainty, and cognitive economy: A new theoretical perspective on confirmatory information search. *Social and Personality Psychology Compass, 5*(10), 751–762. doi:10.1111/j.1751-9004.2011.00386.x
- Fischer, P., Fischer, J., Weisweiler, S., & Frey, D. (2010). Selective exposure to information: How different modes of decision making affect subsequent confirmatory information processing. *British Journal of Social Psychology, 49*(4), 871–881.

- Fischer, P., & Greitemeyer, T. (2010). A new look at selective-exposure effects: An integrative model. *Current Directions in Psychological Science*, 19(6), 384–389. doi:10.1177/0963721410391246
- Fischer, P., Greitemeyer, T., & Frey, D. (2008). Self-regulation and selective exposure: The impact of depleted self-regulation resources on confirmatory information processing. *Journal of Personality and Social Psychology*, 94(3), 382–395. doi:10.1037/0022-3514.94.3.382
- Fischer, P., Jonas, E., Frey, D., & Schulz-Hardt, S. (2005). Selective exposure to information: The impact of information limits. *European Journal of Social Psychology*, 35, 469–492. doi:10.1002/ejsp.264
- Fischer, P., Schulz-Hardt, S., & Frey, D. (2008). Selective exposure and information quantity: How different information quantities moderate decision makers' preference for consistent and inconsistent information. *Journal of Personality and Social Psychology*, 94, 231–244. doi:10.1037/0022-3514.94.3.382
- Fitzsimons, G. J., & Lehmann, D. R. (2004). Reactance to recommendations: When unsolicited advice yields contrary responses. *Marketing Science*, 23(1), 82–94. doi:10.1287/mksc.1030.0033
- Frey, D. (1986). Recent research on selective exposure to information. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 19, pp. 41–80). New York: Academic Press.
- Hart, W., Adams, J. M., Burton, K. A., Shreves, W. B., & Hamilton, J. (2012). Shaping reality vs. hiding from reality: Reconsidering the effects of trait need for closure on information search. *Journal of Research in Personality*, 46, 489–496. doi:10.1016/j.jrp.2012.05.004
- Hart, W., Albarracín, D., Eagly, A. H., Brechan, I., Lindberg, M. J., & Merrill, L. (2009). Feeling validated versus being correct: A meta-analysis of selective exposure to information. *Psychological Bulletin*, 135(4), 555–588. doi:10.1037/a0015701
- Howell, J. L., & Shepperd, J. A. (2012). Reducing information avoidance through affirmation. *Psychological Science*, 23(2), 141–145. doi:10.1177/0956797611424164
- Jonas, E., & Frey, D. (2003). Information search and presentation in advisor-client interactions. *Organizational Behavior and Human Decision Processes*, 91, 154–168. doi:10.1016/S0749-5978(03)00059-1
- Jonas, E., Graupmann, V., & Frey, D. (2006). The influence of mood on the search for supporting versus conflicting information: Dissonance reduction as a means of mood regulation? *Personality and Social Psychology Bulletin*, 32, 3–15. doi:10.1177/0146167205276118
- Jonas, E., Greenberg, J., & Frey, D. (2003). Connecting terror management and dissonance theory: Evidence that mortality salience increases the preference for supporting information after decisions. *Personality and Social Psychology Bulletin*, 29(9), 1181–1189. doi:10.1177/0146167203254599
- Klein, W. M., & Harris, P. R. (2009). Self-affirmation enhances attentional bias toward threatening components of a persuasive message. *Psychological Science*, 20(12), 1463–1467. doi:10.1111/j.1467-9280.2009.02467.x
- Knobloch-Westerwick, S., Johnson, B. K., & Westerwick, A. (2013). To your health: Self-regulation of health behavior through selective exposure to online health messages. *Journal of Communication*, 63(5), 807–829. doi:10.1111/jcom.12055
- McFarland, S. G., & Warren, J. C., Jr. (1992). Religious orientations and selective exposure among fundamentalist Christians. *Journal for the Scientific Study of Religion*, 163–174. doi:10.2307/1387006
- Noguchi, K., Albarracín, D., Durantini, M. R., & Glasman, L. R. (2007). Who participates in which health promotion programs? A meta-analysis of motivations underlying enrollment and retention in HIV-prevention interventions. *Psychological Bulletin*, 133, 955–975. doi:10.1037/0033-2909.133.6.955
- Penner, L. A., Dovidio, J. F., West, T. V., Gaertner, S. L., Albrecht, T. L., Dailey, R. K., & Markova, T. (2010). Aversive racism and medical interactions with Black patients: A field study. *Journal of Experimental Social Psychology*, 46(2), 436–440. doi:10.1016/j.jesp.2009.11.004
- Stone, P., Richards, M., A'Hern, R., & Hardy, J. (2000). A study to investigate the prevalence, severity and correlates of fatigue among patients with cancer in comparison with a control group of volunteers without cancer. *Annals of Oncology*, 11(5), 561–567.

- Weinstein, N. D., & Lyon, J. E. (1999). Mindset, optimistic bias about personal risk and health-protective behaviour. *British Journal of Health Psychology*, 4(4), 289–300. doi:10.1348/135910799168641
- Young, T., Finn, L., Peppard, P. E., Szklo-Coxe, M., Austin, D., Nieto, F. J., ... Hla, K. M. (2008). Sleep disordered breathing and mortality: Eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep*, 31(8), 1071–1078.

Suggested Reading

- Fischer, P., & Greitemeyer, T. (2010). A new look at selective-exposure effects: An integrative model. *Current Directions in Psychological Science*, 19(6), 384–389. doi:10.1177/0963721410391246
- Hart, W., Albarracín, D., Eagly, A. H., Brechan, I., Lindberg, M. J., & Merrill, L. (2009). Feeling validated versus being correct: A meta-analysis of selective exposure to information. *Psychological Bulletin*, 135(4), 555–588. doi:10.1037/a0015701
- Knobloch-Westerwick, S., Johnson, B. K., & Westerwick, A. (2013). To your health: Self-regulation of health behavior through selective exposure to online health messages. *Journal of Communication*, 63(5), 807–829. doi:10.1111/jcom.12055

Self-Affirmation and Health

Peter R. Harris, Donna C. Jessop, and Philine S. Harris

School of Psychology, University of Sussex, Brighton & Hove, UK

Self-affirmation theory (Steele, 1988) posits that people are typically highly motivated to protect their “self-integrity,” which is their global sense of being morally and adaptively adequate. Steele described self-integrity as the “phenomenal experience” of such things as being “competent, good, coherent, unitary, stable, capable of free choice, [and] capable of controlling important outcomes ...” (1988, p. 262). The motive to protect self-integrity renders people vigilant to threats to self-integrity. Such threats are subjective, many, and various and include both major life events (e.g., divorce, redundancy, illness) and more mundane, day-to-day experiences (e.g., petty insults, disappointing feedback). They have been labeled “psychological threats” and defined as “the perception of environmental challenge to one’s self-integrity” (Cohen & Sherman, 2014, p. 335).

According to self-affirmation theory, experiencing or anticipating such psychological threats elicits the motivation to protect self-integrity. Protecting self-integrity can be achieved in different ways (Sherman & Cohen, 2006). People can reduce the threat by accommodation (i.e., changing their behavior or beliefs in appropriate ways), by assimilation (i.e., rendering the threat consistent with preexisting beliefs and knowledge, even if this involves distorting perceptions about the threat), or by self-affirmation. For example, from the perspective of self-affirmation theory, information about the health risks of smoking presents a self-integrity threat to smokers by challenging self-perceptions of adequacy; those who value being healthy are knowingly undermining their future health. A smoker might respond to such information by accommodation (e.g., quitting or at least trying to quit or cut down). However, behavior change is not always easy to achieve. Indeed, from the perspective of self-affirmation theory, change requires acknowledging that one’s behavior has been unwise, which presents a self-integrity threat. Consequently, for these and other reasons, it may be easier to assimilate the threat. Thus, smokers may instead reject the information or its personal relevance (e.g., by denigrating the evidence or the source of the message) or generate cognitions that enable them to keep smoking while reducing the self-integrity threat (e.g., by forming the intention to quit but postponing the date on which

they will begin). However, self-affirmation theory posits a third possibility: they may self-affirm.

Self-affirmation is “an act that manifests one’s adequacy and thus affirms one’s sense of global self-integrity” (Cohen & Sherman, 2014, p. 337). For example, when faced with information about the health risks of smoking (e.g., exposure to a warning on a cigarette pack), smokers may cope by reminding themselves that they are talented musicians or reliable friends or by bringing to mind a recent act of generosity that typifies important personal values. By reminding themselves that they are morally or adaptively adequate, despite smoking, an effective self-affirmation will offset the self-integrity threat entailed in the warning. Thus, self-affirmation has the potential to preserve self-integrity.

In everyday life self-affirmation can be realized in many and diverse ways. In experimental work using self-affirmation theory, however, self-affirmation is often operationalized as a value affirmation, “an activity that provides the opportunity to assert the importance of core values, often through writing exercises” (Cohen & Sherman, 2014, p. 337). For example, participants may be asked to write a brief essay about their most important personal value and occasions on which they manifest it (e.g., Harris & Napper, 2005).

Steele’s critical theoretical insight was that, in responding to psychological threats, people are concerned primarily with their general or global sense of adequacy, rather than with addressing the specific issue that triggered the threat (the focal threat). For instance, although smokers may indeed cope with the threat triggered by a health warning by quitting or by elaborating cognitions that reduce their dissonance without changing behavior, they may also do so by thoughts or deeds that are completely unrelated to smoking, such as through a value affirmation (e.g., by thinking about a recent act of generosity and the personal importance of that value). As a result, self-affirmation can ameliorate threats in a domain (e.g., health) that is different from the domain in which the self-affirmation occurs (e.g., generosity). That is, effective affirmations may involve thinking about the self or acting in a way that is unrelated to the focal threat.

Self-affirmations can be used prospectively, to ward off an expected psychological threat, or retrospectively, to ameliorate an experienced threat. To be effective, the self-affirmation must be at least as important to the self as is the focal threat.

It is important to note that self-affirmation does not eliminate the threat; instead, it serves to contain or contextualize it. That is, a threat that seemed potent seems less so once, through self-affirmation, individuals have reassured themselves that they are competent or morally worthy.

Most of the research on self-affirmation—especially in the context of health—has focused on a consequence of self-affirmation that Steele (1988) discussed only relatively briefly, namely, the possibility that self-affirmation may promote less defensive and more open-minded responding to threats. That is, the self-affirmed person should feel more secure and better able to confront a given threat because self-integrity has been bolstered by the self-affirmation. For example, smokers who have been reassured of their competence or moral worthiness through self-affirmation should experience less psychological threat when encountering a reminder of the foolhardiness of smoking and may be better able to face up to the need to quit. Thus, self-affirmation has the potential not only to preserve self-integrity but also to encourage adaptive change.

This element of the theory is of obvious interest to those working on problems such as defensive resistance to potentially useful but psychologically threatening information (e.g., critical feedback, uncongenial risk information), not least because the theory suggests how resistance may be reduced, by providing the opportunity to self-affirm.

Self-Affirmation and Health

Two broad strands of research have addressed self-affirmation and health. One strand applies self-affirmation theory to understanding and ameliorating defensive resistance to information about health risks and health status. The other strand, which has received less empirical attention, examines whether and how self-affirmation may ameliorate stress reactions or bolster well-being.

Self-Affirmation and Defensive Resistance to Health Information

Messages targeting unhealthy behavior and promoting healthier behavior are ubiquitous. They are an integral part of health promotion practice and come in many forms and through various media. However, it is not uncommon to find that members of the targeted audience engage in strategies to avoid, denigrate, or otherwise downplay the information and its implications for their behavior. As outlined above, the assumption underlying the extension of self-affirmation theory to research on message resistance is that such messages, at least in part, challenge self-integrity and therefore constitute a psychological threat. The benefits of this analysis in terms of self-affirmation theory are twofold. First, the analysis extends the account of the processes underlying message resistance to include psychological and physical threat. Second, it offers a practical solution, as self-affirming should reduce the psychological threat induced by the information and therefore lessen the need to engage in defensive resistance.

The research on self-affirmation and message resistance has been primarily experimental. The typical experiment involves the random assignment of participants to experimental condition (self-affirmation or a control activity), followed by the presentation of information targeting a specific health-promoting or health-damaging behavior and then the completion of an immediate set of outcome (dependent) measures assessing message uptake and readiness to change behavior. Additional elements may include some form of manipulation check, the assessment of potential moderators and mediators, and follow-up testing for effects on behavior subsequent to the experimental session. Space precludes exhaustive coverage of the many such studies here. Instead we attempt to illustrate the main findings, characteristics, and themes of this literature and to highlight some of the issues facing researchers (see Suggested Reading for more extensive coverage).

Overall, evidence suggests that self-affirmation can influence the cognitive, affective, and behavioral responses to health-related information, both immediately and at follow-up, and in the contexts of either health-damaging (alcohol consumption, cigarette smoking, unsafe sexual activity, consumption of mercury in oily fish, and doping in sport) or health-promoting (fruit and vegetable consumption, physical activity, dental flossing, sun protection, and screening for diseases) behaviors. Meta-analyses (see Suggested Reading) have reported small but reliable effects of self-affirmation manipulations on health-related outcomes comparable in magnitude to those obtained in meta-analyses of other health behavior change interventions. The health risk information used in these studies has been predominantly textual and has tended to emulate a typical health leaflet or brochure, but studies have also found positive effects of self-affirmation manipulations on other types of material (e.g., on-pack cigarette warning labels; Harris, Mayle, Mabbott, & Napper, 2007; Kessels, Harris, Ruiter, & Klein, 2016). Measures have been predominantly self-reported and have included measures of brain function (e.g. via fMRI; Falk et al., 2015); attention allocation (e.g. via eye tracking; Kessels et al., 2016); information avoidance (Howell & Shepperd, 2016); objective indices of behavior,

such as weight loss (Logel & Cohen, 2012) and activity recorded on pedometers (Falk et al., 2015); and measures indicating improved treatment adherence in hemodialysis patients (e.g. Wileman et al., 2016).

Effects of self-affirmation manipulations or interventions have been detected at follow-up months later (Falk et al., 2015; Harris et al., 2014; Harris & Napper, 2005; Logel & Cohen, 2012). However, relatively little theoretical attention has been given to the role that self-affirmation may play at later stages in the behavior change process (see Suggested Reading), albeit theoretical attention has been paid to explaining how such a relatively brief intervention may have enduring effects in other contexts (see Cohen & Sherman, 2014).

Although researchers have proposed numerous potential moderators of self-affirmation effects, the published meta-analyses of self-affirmation and health found limited evidence for moderation by the variables they tested (see Suggested Reading). There is a need for more tests of potential moderators in order to advance understanding of the boundary conditions surrounding the positive effects of self-affirmation on health-related outcomes. Theoretically it makes sense for self-affirmation effects to be moderated by variables that affect the experience of psychological threat, such that those who experience more threat should benefit most. For example, there is evidence that the impact of self-affirmation on the response to health-related information can be moderated by risk level, with those at higher risk of some negative health outcome as a result of their behavior being more responsive to the message after self-affirming than those at lower risk (e.g., Harris & Napper, 2005). However, not all studies that have tested for moderation by risk level have found it (see Schütz, Cooke, Schütz, & van Koningsbruggen, 2017).

Support for mediating variables has also been mixed. In all likelihood, self-affirmation is multiply mediated (Cohen & Sherman, 2014). In relation to health, it may be important to differentiate the mediators (and moderators) of message acceptance processes from the mediators (and moderators) of the processes involved in subsequent health behavior change (Harris & Epton, 2010).

As the literature matures, negative findings are also being published, notably in the area of binge drinking among students (e.g. Norman & Wrona-Clarke, 2015). It is also not uncommon to find positive effects of self-affirmation manipulations on immediate outcomes but not on behavior at follow-up. It is not clear what is reasonable to expect in terms of the longer-term, and especially the behavioral, impact of such a relatively brief intervention. However, in the educational context there have been several findings of significant long-term effects of self-affirmation interventions, suggesting that the effects can endure (Cohen & Sherman, 2014).

Self-Affirmation, Stress, and Well-Being

Many stressors in the modern world can be construed as threats to self-worth or self-integrity (Sherman & Cohen, 2006). For example, losing one's job or failing a test may threaten one's view of the self as valued or capable. Self-affirmation manipulations may help to remind people of the bigger picture in such situations, reassuring them that they have integrity and broadening their focus beyond the threat (Cohen & Sherman, 2014). Self-affirmation is thought to boost perceptions of the psychological resources available for coping, thereby allowing individuals to better gain perspective, with the result that potential stressors may have less of an influence on their mental and physical well-being (Cohen & Sherman, 2014).

There is evidence that self-affirmation can reduce adverse psychological and physiological responses to stressors, both experimental and naturalistic (Cohen & Sherman, 2014). For

example, participants who were self-affirmed before giving a speech and completing a mental arithmetic task in front of a hostile audience did not experience increases in cortisol (Creswell et al., 2005); similarly, self-affirmed university students taking midterm examinations displayed reduced epinephrine responses and reported less worry compared with their non-affirmed counterparts (Sherman, Bunyan, Creswell, & Jaremka, 2009).

Research attention has also been paid to whether self-affirmation might benefit well-being. Studies are few, but findings suggest that self-affirmation may have positive implications for both eudaimonic and hedonic well-being (see Howell, 2016). However, it should be noted that studies to date have tested for a direct impact of self-affirmation on well-being in the absence of an explicit threat. Although there are precedents for this in the broader literature on self-affirmation and health (e.g. Logel & Cohen, 2012, showed positive effects of self-affirmation on weight loss without explicitly introducing a threat), it is possible that the effects of self-affirmation on well-being may differ when an explicit threat is in place.

Practical Developments and Issues

Naturally, given the evidence that self-affirmation can reduce information resistance and avoidance, mitigate the response to stressors, and may even bolster well-being, researchers and practitioners have been keen to examine its role in interventions (see Schütz et al., 2017), including among various patient groups (e.g., Wileman et al., 2016), and its implications for public policy (Ehret & Sherman, 2014).

To date intervention research has produced some encouraging findings. However, caution should be exercised when employing self-affirmation as an intervention given that its mechanism of action is poorly understood, as are its limits and disadvantages. More generally, there is evidence that self-affirmation manipulations may produce distinctive and even detrimental effects on responses among those experiencing low or no threat (e.g., Briñol, Petty, Gallardo, & DeMarree, 2007). In mass communications, such individuals will inevitably form part of the audience.

Another potential problem for interventions is that being aware of the effects of self-affirmation can attenuate those effects; however, the effect of awareness seems to be eliminated when participants freely choose to self-affirm (Silverman, Logel, & Cohen, 2013).

Setting aside such concerns, the application of self-affirmation in many contexts outside the laboratory requires the development of reliable ways of self-affirming that are brief and easily implemented. Few such self-affirmation manipulations are currently available, and all require further testing.

One practical benefit of self-affirmation is that it can be used with existing health promotion materials. However, whether self-affirmation will boost persuasion is dependent on the materials themselves. Self-affirmation is hypothesized to work by promoting less defensiveness and therefore greater readiness to engage with information open-mindedly. It is not in itself a technique to promote persuasion. So, whether people are persuaded depends on the quality of the material, such as the strength of its arguments.

Individual Differences

Although the research to date has mainly used experimental manipulations of self-affirmation, there has been some research on health testing self-affirmation as an individual difference. Harris and colleagues (Harris et al., 2019) have developed a measure of individual differences

in self-affirmation, items from which have been associated with beneficial outcomes among cancer survivors in a nationally representative US sample (Taber, Klein, Ferrer, Kent, & Harris, 2016) and with reduced information avoidance tendencies in a sample recruited to an NIH genome sequencing study (ClinSeq®) (Taber et al., 2015). A measure developed by Pietersma and Dijkstra (2011) was related positively to perceptions of the negative consequences of smoking in samples of smokers.

Theoretical Developments and Issues

Crocker and colleagues (e.g. Crocker, Niiya, & Mischkowski, 2008) have argued that value affirmation reduces defensiveness by inducing positive other-directed feelings, such as love and connectedness, and thus promotes self-transcendence rather than self-integrity. In contrast, Lindsay and Creswell (2014) have proposed that self-affirmation boosts self-directed compassion rather than self-transcendence. Both the self-transcendence and self-directed compassion accounts have some empirical support but require further testing. Critcher and Dunning (2015) have claimed (with supportive evidence) that self-affirmation expands the working self-concept, producing a broader perspective that reduces the impact of the threat. Other work points to the effects of self-affirmation on neural reward mechanisms (e.g., Falk et al., 2015), executive function (Harris, Harris, & Miles, 2017), and goal pursuit (e.g., Vohs, Park, & Schmeichel, 2013). Critcher, Dunning, and Armor (2010) have shown that self-affirmation reduces defensiveness as long as a defensive response to threat has not been initiated, suggesting that self-affirmation after experiencing a threat may not always be effective.

Sherman (2013) has drawn on elements of the above theorizing and findings to develop a three-component model of the effects of self-affirmation in which self-affirmation (a) boosts resources, (b) broadens the perspective with which people view information and events in their lives, and (c) uncouples the threat from the self, thereby reducing its impact and promoting more open-minded appraisal.

Harris and Epton (2010) argue that there are two broad foci for theoretical development in relation to health risk information, explaining the effects of self-affirmation on message acceptance and on behavior change processes. Although it remains possible that there are features peculiar to health, or even to specific health threats, the application of self-affirmation theory to message acceptance is potentially a straightforward extension of the theory to a domain in which participants are exposed to information that constitutes a psychological threat, and the mechanisms by which self-affirmation promotes more open-minded threat appraisal should be common across domains.

The application of self-affirmation theory to behavior change processes, however, is less straightforward. For example, should the effects of self-affirmation on motivation to change and subsequent behavior change be fully mediated by changes in known predictors of intentions and behavior—induced by persuasion following exposure to the message—or are there other effects that self-affirmation induces in parallel, including direct effects on outcomes? Purchase on this question is in part hindered by features of study designs to date, such as the tendency to not include a no-information control condition, which undermines the ability to differentiate direct from indirect effects of the manipulation. More broadly, it is important to note that most of the research involving self-affirmation has tested the reflective path to behavior change (e.g., mediated by intentions), and little research attention has been paid to its impact on automatic processes.

Directions for Future Research

Research on self-affirmation and health, especially as applied to responses to health risk information, is burgeoning. However, there are limitations to the current literature. For example, there is a need to broaden the range of manipulations employed and populations sampled and to increase the sample sizes routinely used. There is also inconsistency from study to study concerning what is measured, how it is measured, and which variables are affected by the manipulation, limiting the ability to integrate findings. More broadly, theoretical development, clarification, and integration with other related ideas would also be welcome. For example, greater attention could be paid to using relevant theories of attitude change, persuasion, and behavior change, when choosing measures and when constructing persuasive materials for self-affirmation studies. Work also needs to be done to better connect self-affirmation theory with other theories of self and identity and to clarify and operationalize the central theoretical processes.

Nevertheless, as the research reviewed here indicates, self-affirmation can reduce information resistance and avoidance, foster health behavior change, and mitigate the response to stressors. It may even bolster well-being. There are many promising avenues, both theoretical and practical, waiting to be explored.

Author Biographies

Peter R. Harris is a social and health psychologist and professor of psychology at the University of Sussex, UK. His principal research interests lie in the cognitive, emotional, and behavioral response to risk, especially health risks. A founder member of Sussex University's Self-Affirmation Research Group, to date he has published over 30 papers and articles on self-affirmation, particularly as applied to health.

Donna C. Jessop is a senior lecturer at the University of Sussex. Her research interests broadly lie in the fields of social and health psychology, with a particular focus on the design of interventions to promote health protective behaviors and well-being. She has conducted research applying self-affirmation theory to a range of health outcomes, including health-related behaviors, stress responses, and well-being. Donna is a founder member of Sussex University's Self-Affirmation Research Group.

Philine S. Harris is a research fellow at the University of Southampton, UK, where she develops health interventions aimed at supporting patients' self-care. Her PhD research (conducted at the University of Sussex with Peter Harris) investigated the immediate cognitive and emotional effects of self-affirmation, in particular its effects on the use of executive functioning resources and on mood.

References

- Briñol, P., Petty, R. E., Gallardo, I., & DeMarree, K. G. (2007). The effect of self-affirmation in non-threatening persuasion domains: Timing affects the process. *Personality and Social Psychology Bulletin*, 33, 1533–1546. doi:10.1177/0146167207306282
- Cohen, G. L., & Sherman, D. K. (2014). The psychology of change: Self-affirmation and social psychological intervention. *Annual Review of Psychology*, 65, 333–371. doi:10.1146/annurev-psych-010213-115137

- Creswell, J. D., Welch, W. T., Taylor, S. E., Sherman, D. K., Gruenewald, T. L., & Mann, T. (2005). Affirmation of personal values buffers neuroendocrine and psychological stress responses. *Psychological Science*, 16, 846–851. doi:10.1111/j.1467-9280.2005.01624.x
- Critcher, C. R., & Dunning, D. (2015). Self-affirmations provide a broader perspective on self-threat. *Personality and Social Psychology Bulletin*, 41, 3–18. doi:10.1177/0146167214554956
- Critcher, C. R., Dunning, D., & Armor, D. A. (2010). When self-affirmations reduce defensiveness: Timing is key. *Personality and Social Psychology Bulletin*, 36, 947–959. doi:10.1177/0146167210369557
- Crocker, J., Niiya, Y., & Mischkowski, D. (2008). Why does writing about important values reduce defensiveness? Self-affirmation and the role of positive other-directed feelings. *Psychological Science*, 19, 740–747. doi:10.1111/j.1467-9280.2008.02150.x
- Ehret, P. J., & Sherman, D. K. (2014). Public policy and health: A self-affirmation perspective. *Policy Insights from the Behavioral and Brain Sciences*, 1, 222–230. doi:10.1177/2372732214549472
- Falk, E. B., O'Donnell, M. B., Cascio, C. N., Tinney, F., Kang, Y., Lieberman, M. D., ... Strecher, V. J. (2015). Self-affirmation alters the brain's response to health messages and subsequent behavior change. *Proceedings of the National Academy of Sciences*, 112, 1977–1982. doi:10.1073/pnas.1500247112
- Harris, P. R., Brearley, I., Sheeran, P., Barker, M., Klein, W. M., Creswell, J. D., ... Bond, R. (2014). Combining self-affirmation with implementation intentions to promote fruit and vegetable consumption. *Health Psychology*, 33, 729–736. doi:10.1037/hea0000065
- Harris, P. R., & Epton, T. (2010). The impact of self-affirmation on health-related cognition and health behaviour: Issues and prospects. *Social and Personality Psychology Compass*, 4, 439–454. doi:10.1111/j.1751-9004.2010.00270.x
- Harris, P. R., Griffin, D. W., Napper, L. E., Bond, R., Schütz, B., Stride, C., & Brearley, I. (2019). Individual differences in self-affirmation: Distinguishing self-affirmation from positive self-regard. *Self and Identity*, 18(6), 589–630.
- Harris, P. R., Mayle, K., Mabbott, L., & Napper, L. (2007). Self-affirmation reduces smokers' defensiveness to graphic on-pack cigarette warning labels. *Health Psychology*, 26, 437–446. doi:10.1037/0278-6133.26.4.437
- Harris, P. R., & Napper, L. (2005). Self-affirmation and the biased processing of threatening health-risk information. *Personality and Social Psychology Bulletin*, 31, 1250–1263. doi:10.1177/0146167205274694
- Harris, P. S., Harris, P. R., & Miles, E. (2017). Self-affirmation improves performance on tasks related to executive functioning. *Journal of Experimental Social Psychology*, 70, 281–285. doi:10.1016/j.jesp.2016.11.011
- Howell, A. J. (2016). Self-affirmation theory and the science of well-being. *Journal of Happiness Studies*. doi:10.1007/s10902-016-9713-5
- Howell, J. L., & Shepperd, J. A. (2016). Social exclusion, self-affirmation, and health information avoidance. *Journal of Experimental Social Psychology*. doi:10.1016/j.jesp.2016.05.005
- Kessels, L. T., Harris, P. R., Ruiter, R. A., & Klein, W. M. (2016). Attentional effects of self-affirmation in response to graphic antismoking images. *Health Psychology*, 35, 891–897. doi:10.1037/hea0000366
- Lindsay, E. K., & Creswell, J. D. (2014). Helping the self help others: Self-affirmation increases self-compassion and pro-social behaviors. *Frontiers in Psychology*, 5, 421. doi:10.3389/fpsyg.2014.00421
- Logel, C., & Cohen, G. L. (2012). The role of the self in physical health testing the effect of a values-affirmation intervention on weight loss. *Psychological Science*, 23, 53–55. doi:10.1177/0956797611421936
- Norman, P., & Wrona-Clarke, A. (2015). Combining self-affirmation and implementation intentions to reduce heavy episodic drinking in university students. *Psychology of Addictive Behaviors*, 30, 434–441. doi:10.1037/adb0000144

- Pietersma, S., & Dijkstra, A. (2011). Do behavioural health intentions engender health behaviour change? A study on the moderating role of self-affirmation on actual fruit intake versus vegetable intake. *British Journal of Health Psychology*, 16, 815–827. doi:10.1111/j.2044-8287.2011.02018.x
- Schüz, B., Cooke, R., Schüz, N., & van Koningsbruggen, G. M. (2017). Self-affirmation interventions to change health behaviors. In L. Little, E. Silence, & A. Joinson (Eds.), *Behaviour change research and theory: Psychological and technological perspectives* (pp. 87–114). San Diego, CA: Elsevier.
- Sherman, D. K. (2013). Self-affirmation: Understanding the effects. *Social and Personality Psychology Compass*, 7, 834–845. doi:10.1111/spc3.12072
- Sherman, D. K., Bunyan, D. P., Creswell, J. D., & Jaremka, L. M. (2009). Psychological vulnerability and stress: The effects of self-affirmation on sympathetic nervous system responses to naturalistic stressors. *Health Psychology*, 28, 554–562. doi:10.1037/a0014663
- Sherman, D. K., & Cohen, G. L. (2006). The psychology of self-defense: Self-affirmation theory. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 38, pp. 183–242). Amsterdam, The Netherlands: Elsevier. doi:10.1016/S0065-2601(06)38004-5
- Silverman, A., Logel, C., & Cohen, G. L. (2013). Self-affirmation as a deliberate coping strategy: The moderating role of choice. *Journal of Experimental Social Psychology*, 49, 93–98. doi:10.1016/j.jesp.2012.08.005
- Steele, C. M. (1988). The psychology of self-affirmation: Sustaining the integrity of the self. In L. Berkowitz (Ed.), *Advances in experimental social psychology*, 21 (pp. 261–302). New York, NY: Academic Press.
- Taber, J. M., Klein, W. M., Ferrer, R. A., Kent, E. E., & Harris, P. R. (2016). Optimism and spontaneous self-affirmation are associated with lower likelihood of cognitive impairment and greater positive affect among cancer survivors. *Annals of Behavioral Medicine*, 50, 198–209. doi:10.1007/s12160-015-9745-9
- Taber, J. M., Klein, W. M., Ferrer, R. A., Lewis, K. L., Harris, P. R., Shepperd, J. A., & Biesecker, L. G. (2015). Information avoidance tendencies, threat management resources, and interest in genetic sequencing feedback. *Annals of Behavioral Medicine*, 49, 616–621. doi:10.1007/s12160-014-9679-7
- Vohs, K. D., Park, J. K., & Schmeichel, B. J. (2013). Self-affirmation can enable goal disengagement. *Journal of Personality and Social Psychology*, 104, 14–27. doi:10.1037/a0030478
- Wileman, V., Chilcot, J., Armitage, C. J., Farrington, K., Wellsted, D. M., Norton, S., ... Almond, M. (2016). Evidence of improved fluid management in patients receiving haemodialysis following a self-affirmation theory-based intervention: A randomised controlled trial. *Psychology & Health*, 31, 100–114. doi:10.1080/08870446.2015.1073729

Suggested Reading

- Epton, T., Harris, P. R., Kane, R., van Koningsbruggen, G. M., & Sheeran, P. (2015). The impact of self-affirmation on health-behavior change: A meta-analysis. *Health Psychology*, 34, 187–196. doi:10.1037/hea0000116
- Harris, P. R. (2011). Self-affirmation and the self-regulation of health behavior change. *Self and Identity*, 10, 304–314. doi:10.1080/15298868.2010.517963
- Sweeney, A. M., & Moyer, A. (2015). Self-affirmation and responses to health messages: A meta-analysis on intentions and behavior. *Health Psychology*, 34, 149–159. doi:10.1037/hea0000110
- van Koningsbruggen, G. M., Miles, E., & Harris, P. R. (2018). Self-affirmation and self-control: Counteracting defensive processing of health information and facilitating health behavior change. In D. Ridder, M. Adriaanse, & K. Fujita (Eds.), *Handbook of self-control in health and wellbeing* (pp. 495–507). Abingdon-on-Thames, UK: Routledge/Taylor & Francis Group.
- van't Riet, J., & Ruiter, R. A. (2013). Defensive reactions to health-promoting information: An overview and implications for future research. *Health Psychology Review*, 7(sup1), S104–S136. doi:10.1080/17437199.2011.606782

Self-Awareness and Health

Melissa Spina and Jamie Arndt

Psychology Department, University of Missouri System, Columbia, MO, USA

Introduction

In the early 1970s, Shelley Duval and Robert Wicklund (1972; see Suggested Reading) formally introduced the modern psychological world to the concept of self-awareness. With their presentation of objective self-awareness theory, they explained how people can regard the self as the agent of experience or as the object of attention, and how the latter leads people to reduce or avoid discrepancies between their current state and their attitudes, standards, and goals. This process is now widely recognized as a pivotal aspect of self-regulation. Thus, although not always credited as such, the theory is the conceptual parent of many self-regulatory models. This and other contributions mark the concept of self-awareness as having a profound influence on understanding of human social behavior that notably extends into the field of health psychology. This entry offers a brief overview of conceptualizations of self-awareness and their impact on understanding health and health risk behavior.

Objective Self-Awareness Theory and Self-Regulation

According to objective self-awareness theory (Duval & Wicklund, 1972), an individual's conscious attention can be either focused outward on the environment where the self is the subjective agent of experience, or it can be focused inward with the self regarded as the object of attention. Holding the self as an object of attention initiates self-evaluative processes that help people navigate their social environments with desired behavior. The process of self-evaluation is such that when self-aware, people are motivated to match their behavior and attitudes with the standards they maintain for themselves. When an individual observes that she/he is meeting these standards, the individual experiences positive affect and a general tendency to continue this self-focused state. However, when a discrepancy is detected, this self-reflective process has the potential to arouse negative affect. These negative feelings then motivate

efforts to reduce the discrepancy between the individual's current state and the standard the individual holds for the self (e.g., changing the standard or changing behavior to meet said standard) or to avoid the self-focused state (Gibbons & Wicklund, 1976).

To illustrate how these ideas might work, consider a person who wants to maintain a healthy diet and is approaching the drive-through window at a fast-food restaurant. Will the person order the mega-burger or the grilled chicken? The theory suggests that people are more likely to act in accordance with their value of maintaining a healthy diet by ordering the chicken sandwich if they are self-aware. Further, if they do so, they will experience positive feelings as a result of matching their behavior of ordering grilled chicken with their goal of eating well. But if they order the mega-burger and are then made self-aware, the awareness of this discrepancy will increase negative affect. Faced with this situation, people may adjust their standard (e.g., "it is not that important to eat well"), their behavior (e.g., "I will exchange the burger for the chicken"), or try to avoid being self-aware and thus cognizant of the discrepancy.

Over the years, research has supported the core hypotheses of self-awareness induced discrepancy reduction and avoidance of self-focused attention in a variety of ways (see Silvia & Duval, 2001; see Suggested Reading). Research has also shed light on the conditions that motivate people to reduce discrepancies or avoid self-focused attention. For example, people will try to reduce a discrepancy to the extent that their rate of progress in reducing said discrepancy is sufficient relative to the degree of the problem (e.g., if our health conscious friend thinks eating well is important and feels like she/he is making progress toward maintaining a healthy diet); otherwise they will avoid self-focused attention. The original theory has also inspired a number of extensions, refinements, and alternatives. The most generative has been Carver and Scheier's (1981) self-regulatory theory. From this perspective, if people perceive their ability to reduce the discrepancy as high (e.g., our friend thinks she/he can buy, cook, and eat healthy food), they engage in discrepancy reduction behavior. However, if people perceive a low likelihood of reducing the discrepancy (e.g., our friend does not think she/he will be able to actually eat healthy food), they experience negative affect which then propels them to avoid self-focused attention.

Carver and Scheier's (1981) theory also distinguished between focusing on "private" or "public" aspects of the self and, importantly, more explicitly considered the self-evaluative process to exist within a hierarchy of goal-directed self-regulatory systems. For example, the goal of eating well may operate at an intermediate level, serving both the broader more abstract goal of being a healthy person and prescribing the more specific goal of eating more fruits and vegetables. This conceptualization led to further insights into the dynamics of self-regulation. This includes findings indicating that when people do not meet the standards of a certain representation (e.g., "I want to eat well"), they might either shift down to a lower level representation of the goal (e.g., "In the future I will eat more vegetables") on the "behavioral representation ladder" or might disengage and avoid self-focused attention (Carver & Scheier, 1981). While other models of self-awareness have been proposed over the years (see Silvia & Gendolla, 2001), the conceptualization of self-awareness as initiating a self-regulatory process by motivating either discrepancy reducing behavior or self-focused avoidance is the most well-supported and widely accepted view.

Across the literature, researchers have often used the terms self-awareness and self-focus interchangeably. The first experimental approach to inducing such a state was to present participants with a (unexpected) mirror in the research environment, thereby literally rendering the self the object of attention (see Duval & Wicklund, 1972). Over the years, a number of other methods have been used (e.g., listening to a tape -recording of one's voice; see Silvia & Duval, 2001). Research has also developed measures of self-consciousness, often used to describe a

dispositional state of self-awareness to which certain individuals might be more or less disposed (Fenigstein, Scheier, & Buss, 1975). Fenigstein and colleagues also added the terms private and public self-consciousness to the lexicon. Private self-consciousness connotes an inclination to be highly aware of one's personal values and behavior, whereas public self-consciousness describes a tendency to be concerned with the opinions of others and thus to seek approval and avoid rejection from others. These concepts have implications for explaining *when* an individual might be more inclined to experience discrepancies between their own behavior and their self-standards and how concerned one might be about others' perceptions of the discrepancy.

Research on Self-Awareness and Health

Self-Awareness and Perceptions of Physical Symptoms

The original objective self-awareness theory makes no claims about the effect of self-focused attention on how accurately people perceive themselves. Yet such an effect has been implicated to varying degrees by different theories of self-focused attention (see Silvia & Gendolla, 2001). Of relevance to health, this idea inspired research on the effects of self-awareness on people's perceptions of their physical experiences and symptoms. For example, in a series of studies, Pennebaker and Lightner (1980) examined the impact of competing sources of attentional cues on physical performance and perceptions. This research discovered that individuals who engaged in physical exercise in environments containing little external distraction were more aware of their internal states (e.g., enhanced volume of breathing) than were individuals exercising in more stimulating environments (e.g., with distracting sounds). Lacking external distraction also led to a greater awareness of the physical symptoms of exercising (e.g., fatigue). Additional studies found that individuals who ran cross country as opposed to on a track typically ran faster. Presumably this occurred because less external stimuli were present on the track, and thus participants were more focused on the performance impairing awareness of their own physical experiences.

This work has implications for understanding psychological factors that can influence both the engagement in health-relevant behavior and for self-perceptions of health-relevant symptoms. With regard to the former, one possibility is that under certain conditions self-awareness might interfere with people's motivations to engage in a healthy activity (e.g., exercising). If the activity in question has the potential to bring about negative physical experiences (e.g., soreness and fatigue), especially when there is limited experiential stimulation and thus greater internal awareness, people may be less motivated to engage in the activity even if the activity is beneficial to physical well-being.

The effect of self-awareness on perceptions of physical states may also be helpful for understanding symptom reporting. Over the years research has suggested that interoception, or the perception of bodily cues, and the source to which these cues are attributed are important factors in hypochondriasis (e.g., Mechanic, 1972). The general idea is that heightened levels of interoception, as a facet of elevated self-awareness, lead people to amplify and misinterpret bodily sensations as reflecting symptoms of physical disease. This amplification and misinterpretation may be especially likely when heightened self-awareness is combined with negative feelings. In support of this joint impact hypothesis, Gendolla, Abele, Andrei, Spurk, and Richter (2005) found that elevated levels of negative affect coupled with high self-focused attention increased reporting of greater intensity somatic symptoms. Thus, overreporting of physical ailments as a result of enhanced self-awareness and negative mood could have

implications for inaccurate symptomatic self-report, which in turn might contribute to inaccurate diagnoses and other health problems. For example, more recent research has focused on the role of interoceptive sensitivity in anxiety disorders (Domschke, Stevens, Pfleiderer, & Gerlach, 2010) and medication nonadherence (Alcántara et al., 2014).

Risky Health Behavior as Escape From Self-Awareness

Recall that different perspectives converge to posit that self-awareness initiates a self-regulatory process of comparing one's behavior with one's self-standards. When these standards are not being met, the individual is motivated to reduce this discrepancy between their behavior and self-standards or to avoid self-focused attention. But what happens when the need to avoid self-focused attention becomes chronic and is difficult to accomplish? This question has given rise to the general hypothesis that in these instances people might engage in maladaptive health behaviors that facilitate the avoidance of self-focused attention. This hypothesis was initially and perhaps most extensively examined in the context of alcohol consumption and binge eating, though conceptually similar analyses have also been directed to such domains as risky sex and drug use (McKirnan, Ostrow, & Hope, 1996) and self-harm (Chapman, Gratz, & Brown, 2006).

Alcohol Consumption

Excessive alcohol consumption as a mechanism to escape self-awareness has been proposed in several models. Hull (1981) suggested that alcohol use helps to disrupt the cognitive processes that contribute to stress when people are self-aware and confronted with an unfavorable self-evaluation. A number of studies support this hypothesis. For example, Hull, Levenson, Young, and Sher (1983) demonstrated in a series of experiments that alcohol reduced self-awareness and that this effect was especially strong in individuals high in private self-consciousness. Additional studies showed that individuals high in private self-consciousness consumed more alcohol when given failure feedback than individuals high in private self-consciousness who were given success feedback, whereas people low in private self-consciousness did not change their level of alcohol consumption as a function of the feedback given. Such studies suggest that for highly self-conscious individuals, alcohol use can be motivated by a desire to reduce self-focused attention in situations that reflect negatively on the self.

The attention-allocation model (an extension of alcohol myopia theory) offers a different perspective and argues that alcohol leads to a narrowing of attention to informational cues (e.g., "myopia") that enable the individual to avoid anxiety and depressive affect (Steele & Josephs, 1990). In accord with these hypotheses, individuals who are intoxicated and experience an anxiety-inducing situation are less likely to report anxiety when given a distracting cognitive task than intoxicated individuals who were not given the task (Steele & Josephs, 1988). This implies that for people who were performing a distracting task, alcohol facilitated a reduction in anxiety by narrowing the focus of their attention to the task at hand rather than on the potential for anxiety. However, individuals who were intoxicated but did not engage in a distracting task reported more anxiety than people who had consumed a placebo beverage, suggesting that without a distracting task, alcohol enhanced their focus on the anxiety experienced. Thus, the attention-allocation model suggests that using alcohol as a means to escape self-focused attention is primarily effective to the extent it enables one to narrow focus onto some other external cue unrelated to the self.

Eating Disorders and Appearance Concerns

A number of models have applied the general principle of escaping self-awareness to understanding eating disorders. Heatherton and Baumeister (1991) begin by noting that individuals who overeat seem to suffer from high expectations for the self as well as sensitivity to the approval of others (i.e., private and public self-consciousness). For individuals who maintain high standards for their physical appearance, failure to meet the standards they set for themselves can lead to the development of heightened levels of self-awareness and a chronic state of negative affect (i.e., depression and anxiety). The escape model proposes that to escape these negative emotions related to the discrepancy between self-standards and the reality of one's physical appearance, people might (ironically) overeat in an attempt to narrow their attentional focus on the immediate environment and avoid thoughts about their own self-values (Heatherton & Baumeister, 1991; see also Bardone-Cone, Abramson, Vohs, Heatherton, & Joiner, 2006).

Further, individuals with eating disorders are suggested to be plagued by concern for and preoccupation with others' evaluations of themselves. As a result, they construct a socially appealing "false self" (Striegel-Moore, Silberstein, & Rodin, 1993). Indeed, among individuals with bulimia nervosa, reported body dissatisfaction positively relates to their social anxiety, perceived fraudulence (or lack of true self), and public self-consciousness (but not private; Striegel-Moore et al., 1993). Thus, for those who are consumed by thoughts of others' perceptions of themselves, maintenance of a socially desirable "false self" to attain the approval of others could be an indication of a risk factor for an eating disorder (e.g., bulimia nervosa, anorexia).

The aforementioned research suggests that when individuals have difficulty with meeting self-standards (e.g., Heatherton & Baumeister, 1991) or are preoccupied with others' approval (e.g., Striegel-Moore et al., 1993), they can engage in a variety of risky eating behaviors. However, it is important to recognize that the self-regulatory processes instigated by self-awareness are not inherently harmful to health. Recall the example of the person interested in maintaining a healthy diet. The critical issue is whether the behavioral standard is appropriate and facilitates (or undermines) health enhancement or maintenance. Consider, for example, associations between self-consciousness, concern for oral hygiene, and oral well-being (Klages, Bruckner, & Zentner, 2004). If high self-consciousness motivates dental care habits that contribute to oral hygiene, then the self-awareness induced regulatory processes can be appreciated as having a productive effect on health.

Self-Regulation and Goal-Directed Health Behavior

Recognizing that people may seek to escape aversive self-awareness through health risk behavior is one way that understanding self-awareness processes can contribute to understanding health behavior. But more broadly, the maintenance and change of health behavior often, if not inevitably, requires self-regulatory steps of goal setting, goal striving, behavioral monitoring, and adjustment (Mann, de Ridder, & Fujita, 2013; see Suggested Reading). All of these steps either directly or indirectly depend on a capacity for self-focused attention. Thus, self-awareness can be noted as playing a key role in various aspects of health behavior maintenance and change.

One method of meeting one's goals, for example, is through effortful inhibition, a process of self-regulation whereby the individual attempts to elicit self-control to avoid temptation (e.g., avoiding unhealthy food when it is available). Being successful in this endeavor depends on one's capacity to consciously fight off temptation, to remain self-aware of one's current state relative to the goal one is trying to reach. Thus, when people experience a cognitive

impairment (e.g., diminished cognitive resources), it is more likely they will act on impulsive processes that derail the pursuit of their goals. Indeed, research has shown that impairments in working memory and executive functioning relate to reduced self-regulation capacity. For instance, people are more likely to overeat when they are experiencing decrements in working memory capacity (e.g., Hofmann, Gschwendner, Friese, Wiers, & Schmitt, 2008) and a high cognitive burden (e.g., divided attention on multiple tasks; Ward & Mann, 2000). In sum, although classic self-awareness ideas are not always explicitly used in self-regulatory research on health goal maintenance, the basic concepts and processes are foundational to theoretical efforts to understand and predict health behavior.

Conclusion

Self-awareness processes arise in people's everyday involvement with their social environments, and these processes also are pervasively implicated within behavioral health domains. This entry has highlighted a few illustrative connections. These include the involvement of self-awareness processes in people's perceptions of physical symptoms, with implications for overreporting of negative bodily sensations. Self-regulation can also motivate an escape from self-focused attention through risky health behaviors (e.g., binge drinking, overeating) when people fail to meet self-relevant standards or are overly vigilant about the evaluations others hold for them. Lastly, self-awareness plays a general role in the self-regulatory maintenance of health goals.

Author Biographies

Melissa Spina received her BA in psychology from California State University, Fresno, in 2013 and began pursuing her PhD at the University of Missouri, Columbia, in 2013. Her research examines motivational theories generally, with an emphasis in terror management theory, and applies these theories to such areas as health decision making, prosociality, and environmentally friendly behaviors.

Jamie Arndt received his BA from Skidmore College and his PhD from the University of Arizona in 1999. He is currently a professor in the Department of Psychological Sciences at the University of Missouri. His work focuses on the self, existential motivation, psychological defense, and health decision making, among other topics, with an increasing interest in moving basic social psychological ideas to translational health domains.

References

- Alcántara, C., Edmondson, D., Moise, N., Oyola, D., Hiti, D., & Kronish, I. M. (2014). Anxiety sensitivity and medication nonadherence in patients with uncontrolled hypertension. *Journal of Psychosomatic Research*, 77(4), 283–286. <http://dx.doi.org/10.1016/j.jpsychores.2014.07.009>
- Bardone-Cone, A. M., Abramson, L. Y., Vohs, K. D., Heatherton, T. F., & Joiner, T. E. (2006). Predicting bulimic symptoms: An interactive model of self-efficacy, perfectionism, and perceived weight status. *Behaviour Research and Therapy*, 44(1), 27–42. <http://dx.doi.org/10.1016/j.brat.2004.09.009>
- Carver, C. S., & Scheier, M. F. (1981). *Attention and self-regulation*. New York, NY: Springer-Verlag. <http://dx.doi.org/10.1007/978-1-4612-5887-2>

- Chapman, A. L., Gratz, K. L., & Brown, M. Z. (2006). Solving the puzzle of deliberate self-harm: The experiential avoidance model. *Behaviour Research and Therapy*, 44(3), 371–394. <http://dx.doi.org/10.1016/j.brat.2005.03.005>
- Domschke, K., Stevens, S., Pfleiderer, B., & Gerlach, A. L. (2010). Interoceptive sensitivity in anxiety and anxiety disorders: An overview and integration of neurobiological findings. *Clinical Psychology Review*, 30(1), 1–11. <http://dx.doi.org/10.1016/j.cpr.2009.08.008>
- Duval, S., & Wicklund, R. A. (1972). *A theory of objective self-awareness*. Oxford, England: Academic Press.
- Fenigstein, A., Scheier, M. F., & Buss, A. H. (1975). Public and private self-consciousness: Assessment and theory. *Journal of Consulting and Clinical Psychology*, 43(4), 522–527. <http://dx.doi.org/10.1037/h0076760>
- Gendolla, G. H., Abele, A. E., Andrei, A., Spurk, D., & Richter, M. (2005). Negative mood, self-focused attention, and the experience of physical symptoms: The joint impact hypothesis. *Emotion*, 5(2), 131–144. <http://dx.doi.org/10.1037/1528-3542.5.2.131>
- Gibbons, F. X., & Wicklund, R. A. (1976). Selective exposure to self. *Journal of Research in Personality*, 10(1), 98–106. [http://dx.doi.org/10.1016/0092-6566\(76\)90087-8](http://dx.doi.org/10.1016/0092-6566(76)90087-8)
- Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin*, 110(1), 86–108. <http://dx.doi.org/10.1037/0033-2909.110.1.86>
- Hofmann, W., Gschwendner, T., Friese, M., Wiers, R. W., & Schmitt, M. (2008). Working memory capacity and self-regulatory behavior: Toward an individual differences perspective on behavior determination by automatic versus controlled processes. *Journal of Personality and Social Psychology*, 95(4), 962–977. <http://dx.doi.org/10.1037/a0012705>
- Hull, J. G. (1981). A self-awareness model of the causes and effects of alcohol consumption. *Journal of Abnormal Psychology*, 90(6), 586–600. <http://dx.doi.org/10.1037/0021-843X.90.6.586>
- Hull, J. G., Levenson, R. W., Young, R. D., & Sher, K. J. (1983). Self-awareness-reducing effects of alcohol consumption. *Journal of Personality and Social Psychology*, 44(3), 461–473. <http://dx.doi.org/10.1037/0022-3514.44.3.461>
- Klages, U., Bruckner, A., & Zentner, A. (2004). Dental aesthetics, self-awareness, and oral health-related quality of life in young adults. *The European Journal of Orthodontics*, 26(5), 507–514. <http://dx.doi.org/10.1093/ejo/26.5.507>
- Mann, T., de Ridder, D., & Fujita, K. (2013). Self-regulation of health behavior: Social psychological approaches to goal setting and goal striving. *Health Psychology*, 32(5), 487–498. <http://dx.doi.org/10.1037/a0028533>
- McKirnan, D. J., Ostrow, D. G., & Hope, B. (1996). Sex, drugs and escape: A psychological model of HIV-risk sexual behaviours. *AIDS Care*, 8(6), 655–670. <http://dx.doi.org/10.1080/09540129650125371>
- Mechanic, D. (1972). Social psychological factors affecting the presentation of bodily complaints. *New England Journal of Medicine*, 286(2), 1132–1139. <http://dx.doi.org/10.1056/NEJM197205252862105>
- Pennebaker, J. W., & Lightner, J. M. (1980). Competition of internal and external information in an exercise setting. *Journal of Personality and Social Psychology*, 39(1), 165–174. <http://dx.doi.org/10.1037/0022-3514.39.1.165>
- Silvia, P. J., & Duval, T. S. (2001). Objective self-awareness theory: Recent progress and enduring problems. *Personality and Social Psychology Review*, 5(3), 230–241. http://dx.doi.org/10.1207/S15327957PSPR0503_4
- Silvia, P. J., & Gendolla, G. H. (2001). On introspection and self-perception: Does self-focused attention enable accurate self-knowledge? *Review of General Psychology*, 5(3), 241–269. <http://dx.doi.org/10.1037/1089-2680.5.3.241>
- Steele, C., & Josephs, R. (1988). Drinking your troubles away II: An attention-allocation model of alcohol's effect on psychological stress. *Journal of Abnormal Psychology*, 97(2), 196–205. <http://dx.doi.org/10.1037/0021-843X.97.2.196>

- Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia: Its prized and dangerous effects. *American Psychologist*, 45(8), 921–933. <http://dx.doi.org/10.1037/0003-066X.45.8.921>
- Striegel-Moore, R. H., Silberstein, L. R., & Rodin, J. (1993). The social self in bulimia nervosa: Public self-consciousness, social anxiety, and perceived fraudulence. *Journal of Abnormal Psychology*, 102(2), 297–303. <http://dx.doi.org/10.1037/0021-843X.102.2.297>
- Ward, A., & Mann, T. (2000). Don't mind if I do: Disinhibited eating under cognitive load. *Journal of Personality and Social Psychology*, 78(4), 753–763. <http://dx.doi.org/10.1037/0022-3514.78.4.753>

Suggested Reading

- Carver, C. S., & Scheier, M. F. (1981). *Attention and self-regulation*. New York, NY: Springer-Verlag. <http://dx.doi.org/10.1007/978-1-4612-5887-2>
- Duval, S., & Wicklund, R. A. (1972). *A theory of objective self-awareness*. Oxford, England: Academic Press.
- Mann, T., de Ridder, D., & Fujita, K. (2013). Self-regulation of health behavior: Social psychological approaches to goal setting and goal striving. *Health Psychology*, 32(5), 487–498. <http://dx.doi.org/10.1037/a0028533>
- Silvia, P. J., & Duval, T. S. (2001). Objective self-awareness theory: Recent progress and enduring problems. *Personality and Social Psychology Review*, 5(3), 230–241. http://dx.doi.org/10.1207/S15327957PSPR0503_4

Self-Efficacy and Health

Lisa Marie Warner¹ and Ralf Schwarzer^{1,2}

¹*Health Psychology, Freie Universität Berlin, Berlin, Germany*

²*Health Psychology, Australian Catholic University, Melbourne, Victoria, Australia*

Perceived self-efficacy has been described as “the conviction that one can successfully execute the behavior required to produce the outcomes” (Bandura, 1977, p. 193) or the “can-do cognition” (Schwarzer & Warner, 2013, p. 139). This optimistic belief in being capable of succeeding at novel or difficult tasks reflects a sense of control over unforeseen situations as well as challenging environmental demands by means of one’s own behavior (Bandura, 1997). Engaging in a healthy lifestyle is a challenging task for most people. Therefore, self-efficacy is a construct of great importance to health psychology.

Individuals with high self-efficacy tend to see challenges and opportunities instead of problems and obstacles, choose more ambitious goals, and motivate themselves to attain these goals. They think in a more positive manner and are less stressed prior to challenging situations. If they are confronted with obstacles, people with high self-efficacy show more perseverance and maintain their efforts over longer periods of time. In contrast, individuals low in self-efficacy doubt their capabilities to reach their goals. They experience more self-doubts and anxiety when confronted with situations they perceive as challenging and avoid such situations. Therefore, they cope less effectively and are more vulnerable to stress and depression. Self-efficacy beliefs hence affect how people feel, think, and act (Bandura, 1997).

Concepts Distinct From Self-Efficacy Beliefs

Self-efficacy beliefs do not necessarily reflect *ability*—they are an individual’s subjective impression of being able to succeed. Actual abilities do not need to be high to build self-efficacy, as self-efficacy can also generalize if related tasks have been mastered successfully (Bandura, 1977).

Self-efficacy beliefs are distinct from *optimism*. Optimism is the belief that one’s future will be prosperous and favorable. The causes for this positive outlook into the future need not lie within the person. They can lie, for example, in favorable circumstances, help by others, or

pure chance. Self-efficacious persons, however, believe that they can shape their own future and attain desired outcomes by means of their own actions and decisions. Although the correlation between self-efficacy and optimism is moderate to high (about .60), optimism can be considered a superordinate construct under which optimistic self-beliefs, such as self-efficacy, can be subsumed (Alarcon, Bowling, & Khazon, 2013).

Whereas *outcome expectancies* refer to the perception of consequences of a behavior (e.g., better health, lower weight), self-efficacy refers to personal *control* over the execution of a behavior. Both outcome expectancies and self-efficacy, however, predict health behaviors well and play a major role in Bandura's (1997) social cognitive theory as clearly distinct constructs.

The *self-concept* is regarded as being multidimensional, and it subsumes hierarchically organized cognitions people hold about themselves at different levels of concreteness. The self-concept is also associated with affective components such as self-regard or self-esteem. It is rather normative (developed in comparison to others) and stable, whereas self-efficacy has been conceptualized as situation and task specific and malleable to new experiences. Therefore, self-efficacy is a cognition that is more proximal to behavior.

Whereas *autonomy* refers to the experience of choice and the ability to pursue activities that are intrinsically motivated, self-efficacy refers to the belief in being able to pursue those actions that are necessary to successfully accomplish the chosen goals. Some studies find a strong association between autonomy and self-efficacy in specific domains (Vieira & Grantham, 2011), whereas others show that they discriminate well (Warner, Ziegelmann, et al., 2011).

Locus of control incorporates external causes for an outcome, such as luck or task difficulty, as well as internal causes, such as ability or effort. Despite the close connection (about .56) between having an internal locus of control and self-efficacy, the two constructs are clearly distinct (Judge, Erez, Bono, & Thoresen, 2002). Individuals who think that the cause for an outcome is inherent within themselves do not necessarily believe that they can easily change this internal factor. The difference hence lies in the amenability of the internal cause. Self-efficacy is operative, which means that it pertains to one's future behavior, whereas internal control may refer to one's personal characteristics in the past (e.g., I caused the accident because I am a poor driver).

Whereas some researchers use the terms *perceived behavioral control* and self-efficacy interchangeably, others prefer to distinguish them. To understand their conceptual difference, a closer look into typical scale items helps. Perceived behavioral control items usually include a statement about the ease or difficulty of a task, such as "It is easy/difficult for me to..." Self-efficacy items mostly incorporate the "can-do" component, like "I am sure that I can do..."

In contrast to related constructs, self-efficacy hence always implies an internal attribution (one's own capability is the cause), refers to future actions (prospective), and is proximal to the behavior to be executed (operative).

Self-Manifesting Mechanism of Self-Efficacy

Through the processes of setting more ambitious goals, acting more determined to attain those goals, and putting more effort into overcoming obstacles, individuals with high levels of self-efficacy create more opportunities for themselves to experience mastery (Bandura, 1997). Mastery experiences, in turn, increase self-efficacy beliefs, maintaining a self-manifesting mechanism of both. As individuals with low self-efficacy believe less in being able

to achieve what they want, they give up more easily and invest less effort—or as Bandura (1995, p. 4) describes it, “Disbelief in one’s capabilities creates its own behavioral validation.” Also, less self-efficacious people tend to take more responsibility for their failure than for their success. The issue of attribution—the perceived reasons for success or failure—is very important to consider. Failure only decreases self-efficacy if attributed to internal causes such as low capabilities. If failure is attributed externally, for instance, to low effort or overwhelming obstacles, it does not affect self-efficacy. Likewise, success only increases self-efficacy if attributed internally. If attributed to chance or help from others, successes do not increase self-efficacy.

Measurement of Self-Efficacy

Dimensionality of Self-Efficacy Measures

Items to assess self-efficacy can differ on three dimensions: level, strength, and generality. The *level* dimension describes the difficulty of a task. For tasks that can easily be performed, most people will have similarly high self-efficacy beliefs, for example, “I am certain that I can eat five portions of vegetables.” To assess variation in levels of self-efficacy, items need to include tasks with varying difficulty, such as “I am certain that I can eat five portions of vegetables every day,” or additional contextual challenges, such as “I am certain that I can eat five portions of vegetables every day, even on holidays.” For most health behaviors it is the self-regulatory challenge that determines the level of self-efficacy that is needed on a regularly basis and in the face of difficulties and temptations. These difficulties or barriers should previously be identified for each target group.

The *strength* dimension refers to how robust the self-efficacy belief is against failures. If failures can easily diminish a self-efficacy belief, the belief was low on the strength dimension. Thus, the strength dimension is of special importance for the maintenance of effort and recovery after setbacks.

The *generality* dimension of self-efficacy spans the narrowness or broadness of a capability belief. If individuals have efficacy beliefs for very specific actions in specific situations but not for similar tasks in comparable situations, their generality dimension is low. Self-efficacy was originally developed as a task- or domain-specific construct (Bandura, 1997). Bandura assumed that individuals hold very different self-efficacy beliefs for different domains and situations on a more or less general level, like being good at math or at academic tasks in general. This view implies that items to assess self-efficacy should be formulated at a level of specificity that corresponds with the specificity of the outcome behavior. Specifically assessed self-efficacy beliefs were also found to predict specific outcomes best (Bandura, 1997). Some researchers, however, argue that experiencing failure and success can generalize to a global perception of one’s ability to deal with various challenges. Hence, the concept of *general self-efficacy* was developed (Schwarzer & Jerusalem, 1995a; Sherer et al., 1982). It represents a global confidence in one’s abilities to cope with a wide range of demanding or novel situations.

Self-efficacy should preferably be measured with a psychometric scale consisting of several items. To construct self-efficacy items, one can apply this scheme: “I am confident that I can perform action XYZ, even if the XYZ barrier occurs.” Sometimes the barrier does not have to be mentioned explicitly, for example, in domains where the execution of a task is quite challenging for an individual without additional barriers, like “I am confident that I can finish a

marathon.” Detailed guidelines on the construction of self-efficacy measures can be found in a comprehensive book chapter by Bandura (2006).

Measurement of General Self-Efficacy

Examples of items to measure general self-efficacy would be “I am confident that I could deal efficiently with unexpected events,” and “If I am in trouble, I can usually think of a solution” (Schwarzer & Jerusalem, 1995a). Assessing a global measure of self-efficacy can be useful in some research contexts, for example, if multiple health behaviors, multiple chronic diseases, or adjustment to traumatizing experiences are being studied.

Measurement of Domain-Specific Self-Efficacy

There are measures for more or less specific self-efficacy beliefs depending on the focus of research. To investigate a healthy diet, self-efficacy items can, for example, include particular foods or refer to the self-regulatory effort to resist temptations. For safer sexual behavior, different barriers are named within self-efficacy items, such as the ability to use condoms correctly or suggest condoms to a new partner. Other examples of specific self-efficacy scales are diabetes self-care self-efficacy, breast feeding self-efficacy, or work self-efficacy.

Self-efficacy scales, however, are not without criticism. Williams and Rhodes (2014) recently reactivated a debate about the confoundedness of self-efficacy measures with outcome expectancies and motivation and suggest including “if I wanted to” at the end of each self-efficacy items to control for some of the shared variance.

Measurement of Phase-Specific Self-Efficacy

As the challenge for most health behavior is not the performance of the specific behavior but the initiation and maintenance of that behavior over longer periods of time, Scholz, Sniehotta, and Schwarzer (2005) have developed phase-specific self-efficacy beliefs. *Motivational self-efficacy* (task or pre-action self-efficacy) refers to the goal-setting phase and can, for example, be assessed with the stem “I am certain...” followed by items such as “...that I can be physically active on a regular basis, even if it is difficult” (Lippke, Fleig, Pomp, & Schwarzer, 2010). *Volitional self-efficacy* refers to the goal-pursuit phase. It can be subdivided into *maintenance self-efficacy* (e.g., “I am capable of strenuous physical exercise on a regular basis even if I need several attempts until I will be successful”) (Lippke et al., 2010) and *recovery self-efficacy* (e.g., “I am confident that I can resume a physically active lifestyle, even if I have relapsed several times”).

Measurement of Sources of Self-Efficacy

There has not been much research on the systematic assessment of the sources of self-efficacy for specific health behaviors. According to Bandura (1997), mastery experience is the best source, followed by vicarious experience (role modeling), verbal persuasion, and physiological feedback. To assess the sources of self-efficacy for physical activity, Warner et al. (2014) validated six scales with items such as “Even if it turned out challenging at times, I have managed to remain active” for mastery experiences; “I feel motivated to be active if I see people my age being active” for vicarious experiences; “Others encourage me

to be physically active” for verbal persuasion by others; “I motivate myself to be physically active on a regular basis” for self-persuasion; “Just before I start physical activities, I feel tired” for negative affective states; and “Just before I start physical activities, I feel energetic” for positive affective states.

Self-Efficacy in Health Psychology: Some Research Findings

Self-Efficacy in Health Behavior Change Theories

The concept of self-efficacy was originally developed by Bandura as the key construct within his social cognitive theory (Bandura, 1977, 1997). Because of the predictive power of self-efficacy beliefs for various health behaviors, other health behavior change theories like the protection motivation theory, the revised health belief model, and the transtheoretical model included self-efficacy beliefs as well. The related construct of perceived behavioral control was added to the theory of reasoned action, which was then renamed the theory of planned behavior. In the meantime, specific self-efficacy beliefs were found to be highly predictive of a wide range of health behaviors.

There are also several hypotheses addressing how self-efficacy beliefs interact with other resources such as social support. Luszczynska and Cieslak (2009), for example, found support for the *enabling hypothesis* (assuming that social support elicits self-efficacy), as self-efficacy mediated the association between social support from families and fruit and vegetable consumption in patients after a myocardial infarction. Evidence for the *cultivation hypothesis* (more self-efficacious individuals mobilize more support) was found among patients after prostatectomy, who received more social support to perform pelvic floor exercises if they showed more self-efficacy previously (Hohl et al., 2015). For the adaptation of survivors of an earthquake, higher levels of family support were found to compensate for lower levels of self-efficacy (*compensation hypothesis*; Warner, Gutiérrez-Doña, Angulo, & Schwarzer, 2015). However, for individuals with high self-efficacy beliefs, additional support sometimes also shows an *interference effect*, for example, for autonomy beliefs of older adults (*interference hypothesis*; Warner, Ziegelmann, et al., 2011).

General Self-Efficacy

A good example of the use of general self-efficacy is a study on East Germans who migrated to the West after the Berlin Wall had come down. Over a 2-year period, general self-efficacy turned out to be the best single predictor of overall adjustment, as assessed by a number of outcomes such as employment status, social integration, physical health, and subjective well-being (Schwarzer & Jerusalem, 1995b). A measure of general self-efficacy proved to be valuable in this study because an assessment of all corresponding domain-specific self-efficacy measures for every coping outcome would not have been possible under the circumstances of this particular study. Measures of general self-efficacy have also been found to relate to a number of mental and physical health outcomes (e.g., depression, stress, physical activity).

Phase-Specific Self-Efficacy

In an intervention to increase breast self-examination, task self-efficacy was associated with intention, whereas maintenance self-efficacy related to planning and behavior (Luszczynska, 2004). There is also evidence that interventions to increase physical activity affect different

phase-specific self-efficacy beliefs depending on whether they focus on the adoption or maintenance of physical activity (Higgins, Middleton, Winner, & Janelle, 2014).

Sources of Self-Efficacy

A study on the sources of self-efficacy for physical activity in older adults concluded that mastery experiences, vicarious experiences, and subjective perceptions of health were associated with self-efficacy and physical activity over time (Warner, Schütz, Knittle, Ziegelmann, & Wurm, 2011). A longitudinal study with validated scales showed that mastery experience, self-persuasion, and negative affective states were the most prominent predictors of self-efficacy for physical activity in community-dwelling older adults (Warner et al., 2014).

How Can Self-Efficacy Be Increased?

From a theoretical perspective, self-efficacy is built upon four sources: mastery experiences, vicarious experiences, verbal persuasion, and somatic and affective states (Bandura, 1997). Bandura states that *mastery experiences* are “the most effective source of efficacy information because they provide the most authentic evidence of whether one can master whatever it takes to succeed” (1997, p. 80). Intervention techniques have proven successful for enhancing mastery experiences. However, meta-analyses have come to the conclusion that graded mastery could also lower self-efficacy (e.g., Ashford, Edmunds, & French, 2010).

To make the most of newly gained mastery experiences, it is also essential to target a positive attributional style. Interventions can prepare for temporal setbacks and lapses to help individuals avoid what Marlatt and Gordon (1985) called the abstinence violation effect. This effect describes that people tend to see lapses as proof for incapability, which makes them drop all effort so they experience a full relapse. When people learn to attribute lapses to external causes, such as the end of a stressful day or highly tempting situations, self-efficacy can be maintained and trained for future risk situations.

Although not always named among the sources of self-efficacy, Bandura also identifies *mental imagery* as another possible origin of self-efficacy beliefs (Bandura, 1977) and proves its use to treat phobias. Health psychological research shows that imagining the outcomes of a behavior (*approach imagery*) as well as the steps that need to be fulfilled to attain a goal (*process imagery*) can also be effective to increase physical activity among sedentary adults (Chan & Cameron, 2012).

Vicarious experience, like observing others perform a specific behavior with a specific outcome—be it live or symbolic such as in a video or testimonial—increases the belief in being able to master similar tasks and produce comparable results (Bandura, 1977). Vicarious experiences provide the observer with strategies and techniques needed to attain desired goals or to overcome certain stressors. Usually, it is assumed that role models that resemble the observer in attributes such as background, age, gender, or level of expertise show better effects on self-efficacy than dissimilar models (Bandura, 1997).

Verbal or social persuasion happens if someone expresses faith in the capabilities of another (Bandura, 1997). Verbal persuasion from credible sources such as health professionals is most effective (Perloff, 1993). If, however, someone already has a strong belief in *not* being capable to achieve their goal, for example, derived from negative experience in the past, it is easier to lower their self-efficacy than to increase it by attempts of persuasion. Therefore, this source of self-efficacy needs to be addressed with care and experience to avoid reactance.

Previous to challenging tasks, most people perceive *somatic* and *affective states* such as a fast beating heart or sweaty hands. As the natural tendency is to interpret such physiological symptoms as signs of unpreparedness or anticipation of poor performance, people are more likely to feel competent if they do not experience highly aversive arousal (Bandura, 1997). The optimal level of arousal should, however, lay somewhere in the middle, as too high and too low arousal may impede performance. A meta-analysis by Prestwich et al. (2014) shows how stress management strategies can be used to prompt self-efficacy for a healthier diet.

Several meta-analyses have summarized the behavior change techniques that are most effective to increase self-efficacy in physical activity interventions (e.g., Ashford et al., 2010; Olander et al., 2013). However, interventionists in this field should keep in mind that there can be “too much of a good thing.” First, evidence shows that very high levels of self-efficacy can be detrimental to intervention success, especially in the smoking context (e.g., Staring & Breteler, 2004). Self-efficacy beliefs should hence be realistic to generate motivation and health behavior, as overconfidence could elicit frustration and disappointment along the health behavior change process. To conclude in Bandura’s (2004, p. 162) words, “As you venture forth to promote your own health and that of others, may the efficacy force be with you.”

Author Biographies

Lisa Marie Warner is a guest professor at the Division of Health Psychology at the Freie Universität Berlin, Germany. Her research at the interface between health psychology and gerontology focuses on personal and social resources for older adults’ physical activity and social participation, such as their social support and self-efficacy.

Ralf Schwarzer is a professor at the SWPS University of Social Sciences and Humanities in Wroclaw, Poland, and emeritus professor at Freie Universität Berlin, Germany. His research focuses on various aspects of psychology, including stress and anxiety, coping, social support, health behavior change, and self-efficacy. He is the editor of the journal *Applied Psychology: Health and Well-being*. Visit <http://my.psyc.de> for more information.

References

- Alarcon, G. M., Bowling, N. A., & Khazon, S. (2013). Great expectations: A meta-analytic examination of optimism and hope. *Personality and Individual Differences*, 54(7), 821–827. doi:10.1016/j.paid.2012.12.004
- Ashford, S., Edmunds, J., & French, D. P. (2010). What is the best way to change self-efficacy to promote lifestyle and recreational physical activity? A systematic review with meta-analysis. *British Journal of Health Psychology*, 15(2), 265–288. doi:10.1348/135910709X461752
- Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84(2), 191–215. doi:10.1037/0033-295x.84.2.191
- Bandura, A. (1995). Exercise of personal and collective efficacy in changing societies. In A. Bandura (Ed.), *Self-efficacy in changing societies*. Cambridge: Cambridge University Press. doi:10.1017/cbo9780511527692.003
- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York, NY: Freeman. doi:10.5860/choice.35-1826
- Bandura, A. (2004). Health promotion by social cognitive means. *Health Education & Behavior*, 31(2), 143–164. doi:10.1177/1090198104263660

- Bandura, A. (2006). Guide for constructing self-efficacy scales. In F. Pajares & T. Urdan (Eds.), *Self-efficacy beliefs of adolescents* (Vol. 5, pp. 307–337). Greenwich, CT: Information Age Publishing.
- Chan, C., & Cameron, L. (2012). Promoting physical activity with goal-oriented mental imagery: A randomized controlled trial. *Journal of Behavioral Medicine*, 35(3), 347–363. doi:10.1007/s10865-011-9360-6
- Higgins, T. J., Middleton, K. R., Winner, L., & Janelle, C. M. (2014). Physical activity interventions differentially affect exercise task and barrier self-efficacy: A meta-analysis. *Health Psychology*, 33(8), 891–903. doi:10.1037/a0033864
- Hohl, D., Knoll, N., Wiedemann, A., Keller, J., Scholz, U., Schrader, M., & Burkert, S. (2015). Enabling or cultivating? The role of prostate cancer patients' received partner support and self-efficacy in the maintenance of pelvic floor exercise following tumor surgery. *Annals of Behavioral Medicine*, 1–12. doi:10.1007/s12160-015-9748-6
- Judge, T. A., Erez, A., Bono, J. E., & Thoresen, C. J. (2002). Are measures of self-esteem, neuroticism, locus of control, and generalized self-efficacy indicators of a common core construct? *Journal of Personality and Social Psychology*, 83(3), 693–710. doi:10.1037/0022-3514.83.3.693
- Lippke, S., Fleig, L., Pomp, S., & Schwarzer, R. (2010). Validity of a stage algorithm for physical activity in participants recruited from orthopedic and cardiac rehabilitation clinics. *Rehabilitation Psychology*, 55(4), 398–408. doi:10.1037/a0021563
- Luszczynska, A. (2004). Change in breast self-examination behavior: Effects of intervention on enhancing self-efficacy. *International Journal of Behavioral Medicine*, 11(2), 95–103. doi:10.1207/s15327558ijbm1102_5
- Luszczynska, A., & Cieslak, R. (2009). Mediated effects of social support for healthy nutrition: Fruit and vegetable intake across 8 months after myocardial infarction. *Behavioral Medicine*, 35(1), 30–38. doi:10.3200/bmed.35.1.30-38
- Marlatt, G. A., & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York, NY: Guilford Press.
- Olander, E. K., Fletcher, H., Williams, S., Atkinson, L., Turner, A., & French, D. P. (2013). What are the most effective techniques in changing obese individuals' physical activity self-efficacy and behaviour: A systematic review and meta-analysis. *International Journal of Behavioral Nutrition and Physical Activity*, 10(1), 29–29. doi:10.1186/1479-5868-10-29
- Perloff, R. M. (1993). *The dynamics of persuasion*. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Prestwich, A., Kellar, I., Parker, R., MacRae, S., Learmonth, M., Sykes, B., ... Castle, H. (2014). How can self-efficacy be increased? Meta-analysis of dietary interventions. *Health Psychology Review*, 8(3), 270–285. doi:10.1080/17437199.2013.813729
- Scholz, U., Snichotta, F. F., & Schwarzer, R. (2005). Predicting physical exercise in cardiac rehabilitation: The role of phase-specific self-efficacy beliefs. *Journal of Sport & Exercise Psychology*, 27(2), 135–151.
- Schwarzer, R., & Jerusalem, M. (1995a). Generalized self-efficacy scale. In J. Weinman, S. Wright, & M. Johnston (Eds.), *Measures in health psychology: A user's portfolio. Causal and control beliefs* (pp. 35–37). Windsor: NFER-Nelson.
- Schwarzer, R., & Jerusalem, M. (1995b). Optimistic self beliefs as a resource factor in coping with stress. In S. E. Hobfoll & M. W. deVries (Eds.), *Extreme stress and communities: Impact and intervention* (pp. 159–177). Dordrecht: Kluwer. doi:10.1007/978-94-015-8486-9_7
- Schwarzer, R., & Warner, L. M. (2013). Perceived self-efficacy and its relationship to resilience. In S. Prince-Embury & D. H. Saklofske (Eds.), *Resilience in children, adolescents, and adults: Translating research into practice* (pp. 139–150). New York, NY: Springer Science + Business Media. doi:10.1007/978-1-4614-4939-3_10
- Sherer, M., Maddux, J. E., Mercandante, B., Prentice-Dunn, S., Jacobs, B., & Rogers, R. W. (1982). The self-efficacy scale: Construction and validation. *Psychological Reports*, 51, 663–671. doi:10.2466/pr0.1982.51.2.663
- Staring, A. B. P., & Breteler, M. H. M. (2004). Decline in smoking cessation rate associated with high self-efficacy scores. *Preventive Medicine*, 39(5), 863–868. doi:10.1016/j.ypmed.2004.03.025

- Vieira, E. T., Jr., & Grantham, S. (2011). University students setting goals in the context of autonomy, self-efficacy and important goal-related task engagement. *Educational Psychology*, 31(2), 141–156. doi:10.1080/01443410.2010.536508
- Warner, L. M., Gutiérrez-Doña, B., Angulo, M. V., & Schwarzer, R. (2015). Resource loss, self-efficacy, and family support predict posttraumatic stress symptoms: A 3-year study of earthquake survivors. *Anxiety, Stress & Coping*, 28(3), 239–253. doi:10.1080/10615806.2014.955018
- Warner, L. M., Schüz, B., Knittle, K., Ziegelmann, J. P., & Wurm, S. (2011). Sources of perceived self-efficacy as predictors of physical activity in older adults. *Applied Psychology: Health and Well-Being*, 3(2), 172–192. doi:10.1111/j.1758-0854.2011.01050.x
- Warner, L. M., Schüz, B., Wolff, J. K., Parschau, L., Wurm, S., & Schwarzer, R. (2014). Sources of self-efficacy for physical activity. *Health Psychology*, 33(11), 1298–1308. doi:10.1037/hea0000085
- Warner, L. M., Ziegelmann, J. P., Schüz, B., Wurm, S., Tesch-Römer, C., & Schwarzer, R. (2011). Maintaining autonomy despite multimorbidity: Self-efficacy and the two faces of social support. *European Journal of Aging*, 8(1), 3–12. doi:10.1007/s10433-011-0176-6
- Williams, D. M., & Rhodes, R. E. (2014). The confounded self-efficacy construct: Conceptual analysis and recommendations for future research. *Health Psychology Review*, 1–16. doi:10.1080/17437199.2014.941998

Suggested Reading

- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York, NY: Freeman. doi:10.5860/choice.35-1826
- Benight, C. C., & Bandura, A. (2004). Social cognitive theory of posttraumatic recovery: The role of perceived self-efficacy. *Behaviour Research and Therapy*, 42(10), 1129–1148. doi:10.1016/j.brat.2003.08.008
- Luszczynska, A., & Schwarzer, R. (2015). Social-cognitive theory. In M. Conner & P. Norman (Eds.), *Predicting health behaviours* (3rd ed., pp. 225–251). Maidenhead: McGraw Hill Open University Press.
- Schwarzer, R., & Knoll, N. (2007). Functional roles of social support within the stress and coping process: A theoretical and empirical overview. *International Journal of Psychology*, 42(4), 243–252. doi:10.1080/00207590701396641
- Williams, S. L., & French, D. P. (2011). What are the most effective intervention techniques for changing physical activity self-efficacy and physical activity behaviour-and are they the same? *Health Education Research*, 26(2), 308–322. doi:10.1093/her/cyr005

Self-Esteem and Health

Danu Anthony Stinson and Alexandra N. Fisher

Psychology Department, University of Victoria, Victoria, BC, Canada

Self-esteem has been the subject of intense empirical and theoretical study by psychological scientists for over 50 years, and in the latter decades of the twentieth century, this psychological phenomenon also succeeded in capturing the imagination of popular culture. The notion that one's feelings of worth as a person matter and have important consequences for emotion, cognition, behavior, and experiences resonated with the general public. People embraced insights from psychological science concerning the origins and nature of self-esteem, and individuals and governmental organizations alike sought methods for improving self-esteem and reaping the many benefits associated with positive self-regard, including career and academic success, and increased life satisfaction. As a result of this academic and public attention, if a student of psychology tells her friends or family that she is studying self-esteem, her disclosure will often be met with a nod of understanding or a personal anecdote concerning the listener's own experiences.

Yet despite the widespread popularity of self-esteem and the general acknowledgment of its importance, most students of psychology and the general public remain unaware that self-esteem also plays an important role in shaping people's health. Many people who hear of this connection may wonder, "How can *feelings* influence something tangible and *physical* like health?" This question arises from the lingering assumption that the mind and body are distinct entities, an assumption that arose during the dawn of modern Western medicine and continues to dominate Western conceptions of health. But this assumption is erroneous. In fact, the mind and the body are intimately connected, and this connection is abundantly evident in the pervasive links between self-esteem and the various dimensions of well-being that characterize human health.

What Is Self-Esteem?

Before it is possible to understand how and why self-esteem predicts such a seemingly unrelated experience as health, it is essential to first understand how this psychological

characteristic develops and how it relates to other aspects of *the self*: the collection of traits, qualities, schema, roles, beliefs, and attitudes that form the core of one's identity.

The self resides at the very center of one's psychological universe, allowing people to make sense of their past experiences, guide their present behavior, and predict their future experiences. Indeed, when the philosopher René Descartes famously opined, "I think, therefore I am," he was relying on his sense of self to prove his very existence.

As one might expect, a psychological system so important takes time to develop. The first sense of self that coheres during infancy is the sense of being an entity distinct from others, who persists over time and across places. Around the age of 3, as children mature and interact with their social and physical worlds, they gradually develop beliefs about their specific traits and abilities ("I am athletic"), learn the social roles that they are expected to fulfill ("I am a girl"), and develop theories, or scripts, to explain and predict their actions ("If I ask Marjan to play with me, she will say yes"). As children approach puberty, they begin to evaluate their worth and value, and it is this global evaluative component of the self that psychologists call *self-esteem*.

Self-esteem develops in part through a reflected appraisal process, whereby children observe how other people treat them, and from that treatment infer their worth. Being treated with responsive kindness, warmth, and positivity by social partners, especially family, communicates that the self is worthy and valuable. In contrast, being ignored, invalidated, overlooked, or abused by unresponsive or unavailable social partners, especially family, communicates that the self is not worthy of care or deserving of loving kindness. Over time and through repeated exposure, children internalize these social messages, and thus self-esteem is born.

Of course, people are not passive *tabula rasa* onto which their social experiences write. Babies are born with a genetic heritage that shapes the ways in which they interact with the world, and these same forces influence the development of self-esteem. Of particular relevance to the discussion at hand, some people are temperamentally inclined to focus their attention and energy toward seeking rewarding experiences and to experience high levels of positive affect, whereas others are temperamentally inclined to focus their attention and energy toward avoiding punishing experiences and to experience high levels of negative affect. In turn, these higher-order temperamental factors are also related to self-esteem. Reward-focused people who are relatively insensitive to stressors and recover quickly from negative moods will tend to develop higher self-esteem. In contrast, people who are temperamentally attuned to punishments like criticism, who react strongly to stress and take a long time to recover, and who experience frequent negative moods tend to develop lower self-esteem.

This association between temperament and self-esteem probably exists because temperament influences the ways in which people experience the world, which in turn informs self-evaluations. For example, if someone is very sensitive to rewards, she will be more likely to notice and internalize praise and thus develop higher self-esteem. Conversely, if someone is very sensitive to criticism, he will feel the sting of social rebuff more deeply and thus will be more likely to develop lower self-esteem. Thus, temperament is a lens through which self-esteem-relevant experiences and messages must first pass before they are internalized, resulting in self-esteem that is highly subjective in nature. Two people with different temperaments can have the same experiences and from those experiences infer different conclusions about their social worth and thus develop different levels of self-esteem. As this example illustrates, self-esteem develops from a true symbiotic interaction between nature and nurture.

Once self-esteem crystallizes around the age of 12, it remains remarkably stable across the lifespan. Perhaps due to its high degree of stability, people often rely on their self-esteem to regulate their interactions with their material and social worlds. The lens of self-esteem helps

people to make sense of their past experiences, to determine a present course of action, and to predict future outcomes. In each of these domains, lower self-esteem individuals tend to adopt a more pessimistic and cautious outlook than higher self-esteem individuals, who can be quite optimistic and blithe in many circumstances. For example, a lower self-esteem person may attribute past failures to personal shortcomings and thus avoid situations that call on the skills he believes he does not possess because he anticipates failure. In contrast, a higher self-esteem person is more likely to dismiss past failures as resulting from external factors and thus pursue opportunities that offer rewards he feels equipped to effectively claim. These differing orientations are evident in school and workplace settings and in social contexts ranging from interactions with strangers to interactions with lovers, and they result in differing outcomes for people with lower and higher self-esteem. In many life domains, higher self-esteem individuals tend to experience more positive outcomes than their lower self-esteem counterparts, and this difference extends to the domain of health.

How Is Self-Esteem Related to Health?

Following the World Health Organization, we consider health to be a multifaceted construct comprising psychological, physical, and social well-being. Self-esteem predicts outcomes in each of these varied domains.

Psychological Well-Being

Self-esteem is negatively associated with a range of mental health concerns, including depression, anxiety, stress, disordered eating, negative body image, and suicidal ideation. Although it is difficult to determine whether self-esteem is a cause or consequence of these mental health conditions, longitudinal studies that control for third variables and rule out reverse causation can help to tease apart these possibilities. For example, one such study observed that adolescents with low self-esteem and individuals whose self-esteem declined during adolescence were more likely to experience depression fully two decades later (Steiger, Allemand, Robins, & Fend, 2014). Another study that examined self-esteem and mental health outcomes at multiple points in time found that lower self-esteem predicts depression, which, in turn, predicts heightened rates of stressful life events (Orth, Robins, & Meier, 2009). The consensus among psychological scientists is that lower self-esteem is an independent risk factor for the development of numerous mental health conditions.

Physical Well-Being

Self-esteem is also related to a broad range of physical health conditions. For example, lower self-esteem university students report that they experience worse physical health, visit the doctor more often, and miss more days of school due to illness than their higher self-esteem counterparts (Stinson et al., 2008). Moreover, self-esteem is positively associated with people's ability to recover from physical illness and cope with chronic health conditions, including heart disease, HIV/AIDS, cystic fibrosis, and autoimmune disorders. For example, compared with higher self-esteem individuals, lower self-esteem individuals with asthma or rheumatoid arthritis experience more negative affect, less positive affect, greater stress and symptom severity, and more symptom interference and activity restrictions in daily life (Juth, Smyth, & Santuzzi, 2008). Lower self-esteem is also a risk factor for mortality among the elderly. These and other

links between self-esteem and physical health offer a stark reminder of the extensive mind–body connections that characterize the human experience.

Social Well-Being

Self-esteem is also strongly tied to social well-being. Lower self-esteem individuals report that they receive less social support, experience more interpersonal stress, and suffer from loneliness and social isolation to a greater extent than their higher self-esteem counterparts. Such perceptions may accurately reflect important developmental experiences of acceptance and rejection from family and peers. But with the changing social context of adulthood, appraisals of social well-being can diverge from objective reality. Lower self-esteem individuals often underestimate their social partners' actual regard, whereas higher self-esteem individuals' perceptions are often more accurate, or perhaps even overly optimistic. Such individual differences are thought to be motivated by concerns about rejection. Lower self-esteem people are extremely risk averse and would rather err on the side of caution and overlook some acceptance cues than risk rejection by perceiving acceptance that is not actually present. In contrast, higher self-esteem people are blithe in the face of rejection and will boldly perceive acceptance from even the most neutral social companions. These differing social orientations can have self-fulfilling consequences. Lower self-esteem individuals' social insecurities and resultant cool, inhibited social behavior can be off-putting to social partners, whereas higher self-esteem individuals' confident and warm social behavior is attractive to others. Thus, self-esteem and social well-being are linked throughout the lifespan by a recursive process whereby early social experiences help to forge self-esteem, which later guides social experiences that further reinforce self-esteem.

Why Is Self-Esteem Related to Health?

Two dominant models have emerged to explain why self-esteem and health are so intricately woven together.

A Resource Model of Self-Esteem and Health

Self-esteem and health may be linked because higher self-esteem is a psychological resource on which people can rely in times of adversity. In this model, the same temperamental factors and developmental experiences that give rise to higher or lower self-esteem also give rise to characteristic psychological orientations that benefit, or undermine, health. Specifically, the affective, cognitive, and motivational styles that characterize higher self-esteem may allow such individuals to cope with and recover from stressors more effortlessly and quickly than their lower self-esteem counterparts. For example, higher self-esteem individuals effectively employ strategies to improve negative moods and prolong positive moods, whereas lower self-esteem individuals tend to allow negative moods to linger and will actively dampen positive moods (Wood, Heimpel, & Michela, 2003). Higher self-esteem individuals are also motivationally inclined to overlook criticism, failure, and other negative outcomes in favor of rewarding goal pursuit, whereas lower self-esteem individuals are decidedly attentive and sensitive to negative stimuli.

In turn, these characteristic psychological orientations are thought to influence health. Thus, people with lower self-esteem exhibit a stronger stress–illness association than higher self-esteem individuals, such that they are more likely to experience psychological and somatic problems

both on and following stressful days (DeLongis, Folkman, & Lazarus, 1988). Lower self-esteem individuals are also more susceptible to illness and mood disturbance when stress levels increase, even if overall stress is low. In light of these and other findings, many psychological scientists consider lower self-esteem to be a causal risk factor for poor health outcomes.

A Self-and-Social-Bonds Model of Health

A second explanatory model posits that self-esteem predicts health outcomes because self-esteem regulates responses to the threat of rejection in a manner that can undermine lower self-esteem individuals' health (Stinson et al., 2008).

A growing body of evidence suggests that self-esteem plays an important role in a broader psychobiological regulatory system that services the fundamental human *need to belong*, which is a drive to connect with and maintain relationships with benevolent social partners, and conversely, avoid the social pain of rejection. In particular, people rely on their self-esteem to make sense of their past social experiences, to regulate their responses to in-the-moment social cues concerning acceptance and rejection, and to predict their future social experiences. As we have already discussed, lower self-esteem individuals readily perceive rejection from others and react strongly to avoid the rejection they often anticipate, whereas higher self-esteem individuals eagerly anticipate acceptance from others and pursue rewarding opportunities for social connection with little concern about rejection. In turn, these differing psychological orientations influence physiological responses to the threat of rejection.

For everyone, the threat of rejection activates a neuroendocrine system called the hypothalamic–pituitary–adrenal axis (HPA), which triggers a cascade of hormones that characterize the physiological stress response. However, because lower self-esteem individuals are more sensitive to rejection threats than higher self-esteem individuals, they are more likely to exhibit this stress response in their daily lives. For example, lower self-esteem individuals experience a heightened physiological stress response to the threat of rejection, as indicated by increased salivary cortisol levels immediately following rejection and during the early recovery phase (Ford & Collins, 2010). Unfortunately, chronic activation of the HPA is physiologically costly and can undermine health, which may explain why lower self-esteem individuals experience worse health outcomes than their higher self-esteem counterparts. Consistent with this account, on the day following an experience of interpersonal rejection, lower self-esteem individuals report heightened stress, poorer general health, increased physical symptoms of illness, and worse sleep quality relative to both higher self-esteem individuals' experiences on similar days and their own experiences following days when they did not experience rejection (Ford & Collins, 2013). Thus, poor outcomes in the domain of social well-being may be an important mechanism to explain why individuals with lower self-esteem also experience poor outcomes in psychological and physical health domains.

Conclusions

It is tempting to conclude from the preceding discussion that improving self-esteem could result in a miracle panacea. Unfortunately, attempting to improve self-esteem is more easily said than done. The stability of important self-views like self-esteem is so essential to people's daily functioning that people will vociferously defend their existing self-views against the forces of change, even when such defense means maintaining negative evaluations about the self. As a result, attempts to directly improve self-esteem through positive self-statements or other

forms of positive feedback often backfire for lower self-esteem individuals, resulting in undesirable decrements to well-being.

Thus, we suggest that rather than targeting self-esteem directly, health interventions should instead attempt to improve the quality of people's social bonds. Such interventions could directly improve health by improving people's social well-being. In turn, because social well-being has been causally implicated in both physical and psychological well-being, such interventions may have spillover benefits for other health domains. Moreover, social well-being interventions could result in persistent benefits if repeated positive social experiences are internalized over time, resulting in increased self-esteem. Psychological scientists have already identified a number of effective social well-being interventions, including belongingness interventions for stigmatized or marginalized groups. Close relationships researchers have also identified simple interventions that specifically target the social doubts that are characteristic of lower self-esteem. Currently, psychological scientists are working to determine whether this and other promising social well-being interventions can serve as "psychological immunizations," yielding long-term increments in self-esteem that also benefit well-being and health.

Author Biographies

Danu Anthony Stinson is an associate professor in the Psychology Department at the University of Victoria, Canada. Most of her research to date has examined how and why self-esteem influences health and well-being. More recently, she has begun to investigate and seek ways to alleviate social psychological barriers to health and well-being that affect negatively stereotyped groups, including higher body-weight people and women in high-status social positions.

Alexandra N. Fisher is completing her master of science degree in social psychology at the University of Victoria, Canada, under the supervision of Danu Anthony Stinson. She studies the social and interpersonal experiences of female breadwinners and other successful women. Specifically, her research examines the stereotypes and impressions that are often formed and applied to female breadwinners and how these impressions may influence close relationship initiation and maintenance processes.

References

- DeLongis, A., Folkman, S., & Lazarus, R. S. (1988). The impact of daily stress on health and mood: Psychological and social resources as mediators. *Journal of Personality and Social Psychology*, 54, 486–495. doi:10.1037/0022-3514.54.3.486
- Ford, M. B., & Collins, N. L. (2010). Self-esteem moderates neuroendocrine and psychological responses to interpersonal rejection. *Journal of Personality and Social Psychology*, 98(3), 405–419. doi:10.1037/a0017345
- Ford, M. B., & Collins, N. L. (2013). Self-esteem moderates the effects of daily rejection on health and well-being. *Self and Identity*, 12(1), 16–38. doi:10.1080/15298868.2011.625647
- Juth, V., Smyth, J. M., & Santuzzi, A. M. (2008). How do you feel? Self-esteem predicts affect, stress, social interaction, and symptom severity during daily life in patients with chronic illness. *Journal of Health Psychology*, 13(7), 884–894. doi:10.1177/1359105308095062
- Orth, U., Robins, R. W., & Meier, L. L. (2009). Disentangling the effects of low self-esteem and stressful events on depression: Findings from three longitudinal studies. *Journal of Personality and Social Psychology*, 97(2), 307–321. doi:10.1037/a0015645

- Steiger, A. E., Allemand, M., Robins, R. W., & Fend, H. A. (2014). Low and decreasing self-esteem during adolescence predict adult depression two decades later. *Journal of Personality and Social Psychology, 106*(2), 325–338. doi:10.1037/a0035133
- Stinson, D. A., Logel, C., Zanna, M. P., Holmes, J. G., Cameron, J. J., Wood, J. V., & Spencer, S. J. (2008). The cost of lower self-esteem: Testing a self-and-social-bonds model of health. *Journal of Personality and Social Psychology, 94*, 412–428. doi:10.1037/0022-3514.94.3.412
- Wood, J. V., Heimpel, S. A., & Michela, J. L. (2003). Savoring versus dampening: Self-esteem differences in regulating positive affect. *Journal of Personality and Social Psychology, 85*, 566–580. doi:10.1037/0022-3514.85.3.566

Suggested Reading

- Cohen, S., & Janicki-Deverts, D. (2009). Can we improve our physical health by altering our social networks? *Perspectives on Psychological Science, 4*, 375–378. doi:10.1111/j.1745-6924.2009.01141.x
- Martens, A., Greenberg, J., & Allen, J. B. (2008). Self-esteem and autonomic physiology: Parallels between self-esteem and cardiac vagal tone as buffers of threat. *Personality and Social Psychology Review, 12*, 370–389. doi:10.1177/1088868308323224
- Walton, G. M., Cohen, G. L., Cwir, D., & Spencer, S. J. (2012). Mere belonging: The power of social connections. *Journal of Personality and Social Psychology, 102*, 513–532. doi:10.1037/a0025731
- Wood, J. V., Anthony, D. B., & Foddis, W. F. (2006). Should people with low self-esteem strive for high self-esteem? In M. H. Kernis (Ed.), *Self-esteem issues and answers: A source book of current perspectives* (pp. 288–296). New York, NY: Psychology Press.

Self-Regulation

Richie L. Lenne and Traci Mann

Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA

Self-regulation is the set of processes through which individuals make efforts to alter or exert control over their behaviors, cognitions, and emotions, or, more simply, the processes used for setting and pursuing goals.

Self-regulation plays an important role in health promotion and illness prevention because many of the most common causes of death in the United States have a behavioral component that requires ongoing management. Poor health may result from failing to regulate smoking, drug and alcohol use, eating, exercise, and sexual activity, and many treatments require individuals to adhere to medical regimens or do exercises to manage pain. Other health problems may result from failing to regulate anger, anxiety, or stress.

Self-regulation can be conceptualized as a feedback or cybernetic system. In this kind of system, there are three main processes. First, individuals have a standard or goal, imposed by themselves or by others, that they wish to obtain. Second, they monitor their current state and compare it to the standard. Third, if their current state does not match the desired standard, they make changes to their behaviors, cognitions, or emotions aimed at moving closer to the standard. A person may have a goal of weighing a certain weight, for example. They may compare their current weight with that goal weight, find that they weigh too much, and therefore change their eating or exercise behavior in an effort to get closer to the goal weight.

This conceptualization implies that self-regulation is conscious and deliberate, but that is not always the case. Self-regulation can also be automatic, operating through nonconscious processes, for example, when individuals engage in habitual behaviors, or when goals are primed by situational cues.

In addition, the real-life context of self-regulation is considerably more complicated than implied by a simple feedback system. At any given moment, individuals may have multiple competing goals that they are aiming to fulfill simultaneously. Each goal must not only be attended to but also be protected and shielded from distractions, disturbances, and temptations, many of which come from other goals. For example, the goal of avoiding sugar may conflict with the goal of being a doting parent and baking a cake for one's child's birthday.

Regardless of the overall conceptualization, most concepts that pertain to the topic can be incorporated in one of two components of the self-regulation process: goal setting and goal striving.

Goal Setting

Goal setting is the process of deciding what goals to pursue and by what criteria to judge successful goal attainment. These goals can be as small as deciding to take the stairs instead of the elevator or as large as living a more healthy lifestyle. Individuals adopt health goals for different reasons, and the goals themselves have different characteristics that can affect whether goal pursuit is successful. In addition, people abandon goals for different reasons.

Adopting Health Goals

Most people want to live a long and happy life and do not need to be convinced that it is a good idea to take care of their health. However, some form of information is usually the first component to the adoption of a health goal. Accurate health information is not always easy to find in a landscape saturated with often-conflicting accounts and competing incentives. It can be challenging to know what to believe, which can prevent people from forming health goals. According to theories of health behavior change, once people come to believe that certain behaviors carry health consequences, they are more likely to set goals, particularly if they believe they have the ability to control these behaviors and the behaviors align with what society and significant others deems appropriate (Ajzen, 1991).

Another influence on the goal setting process is an individual's attempts to maintain a consistent self-concept (Fishbein et al., 2001) and to move toward aspirational selves and away from feared selves (Markus & Nurius, 1986). Whether a person adopts a new health goal will also be determined by the fit of this goal into the person's larger system of goals (health related or otherwise). Goal systems theory makes predictions about the adoption and successful completion of a goal by accounting for how other goals compete with or support each other (Kruglanski et al., 2002). According to this perspective, goals can be interrelated such that many goals are associated with a common means (multifinal), or a single goal can be achieved through many means (equifinal). The theory postulates that the more goals are interconnected, the stronger motivational value they carry and the more likely people are to adopt and accomplish them.

Goal Characteristics

Despite the desire to live a healthy life, people struggle to set goals that will last long enough to make a difference in their health and well-being. Certain characteristics of goals have been shown to have meaningful implications for successful goal setting and pursuit. These features include motivational aspects of goals, the level of difficulty of the goal, and the type of goal.

Goals may differ based on their motivational focus. People can have goals that are approach oriented, which are focused on moving toward a positive end state, as well as goals that are avoidance oriented, which are focused on preventing a negative end state. Goal orientations are not merely linguistic differences, but they have consequences in terms of whether the goal is likely to be met and for whom. Overall, approach-oriented goals tend to lead to more positive outcomes than avoidance-oriented goals (Elliott & Dweck, 1988). Approach goals may be

more effective because it is harder to define success criteria for avoidance goals (i.e., not getting a disease), and to maintain motivation when faced with an obstacle, it is beneficial to have a clear idea of what steps are required for success. There are also individual differences in motivational orientations, with people tending to be oriented toward either approach or avoidance goals, and these differences may have implications for goal pursuit. For example, there is some evidence that it may be beneficial to match health messages to an individual's motivational orientation (Mann, Sherman, & Updegraff, 2004).

An important predictor of successful goal setting and pursuit, according to self-determination theory (Deci, Koestner, & Ryan, 1999), is whether a person perceives that they have autonomous control over the decision to change their behavior. In fact, social psychologists as early as Lewin (1935) have proposed that self-imposed goals are more motivating than goals imposed by others. Evidence for the importance of self-set goals in the health domain is mixed, however. Interventions in which healthcare providers discuss patient values and support the patient's goal choice instead of recommending a course of action support this hypothesis (Silva et al., 2011). But other research has found similar outcomes for patient and provider set goals (Alexy, 1985), as well as employee and employer set goals (Locke & Latham, 1990).

Goals imposed by others may be less motivating because this undermines the intrinsic value of the goal (Deci et al., 1999). Meta-analytic evidence indicates that extrinsic rewards reduce intrinsic motivation (Deci et al., 1999). In situations where people lack internal motivation, however, extrinsic motivation may be an effective motivator, such as when external reinforcements are added to employee health promotion programs. Having recognized the cost savings that come from investing in the long-term health of the workforce, many employers have adopted material incentive programs designed to encourage their employees to engage in healthy behaviors, such as exercise and smoking cessation. There is little empirical research on the effectiveness of such programs and what, if any, payment structures are the most effective.

Goals also differ in their level of difficulty. Some research suggests that successful goal pursuit requires that the goal is feasible, but other work has found that unrealistic goals may actually inspire goal pursuit (Linde, Jeffery, Finch, Ng, & Rothman, 2004). Research in vocational settings also suggests that high demands (i.e., the goal is challenging) lead to better performance than easier goals as long as self-efficacy is sufficient (Latham & Locke, 2007). In support of this view, when the goal is highly desirable but unrealistic, there is evidence that people will take actions to better achieve the goal, instead of changing the goal itself.

A single goal, such as a weight-loss goal, can be cast in multiple ways. A person may set a goal to develop the knowledge and cooking skills required to eat a healthy diet (i.e., a "mastery goal"), or they may aim to demonstrate their success by achieving a certain weight (i.e., "performance goal"). There is evidence that mastery goals lead to better outcomes than performance goals (Elliott & Dweck, 1988). For example, mastery goals lead to deeper processing, whereas performance goals trigger superficial processing (for a review, see Covington, 2000). Mastery goals have also been shown to bolster self-efficacy, increase amount of time spent on task, augment the belief that effort leads to success, and help lead to persistence in the face of obstacles, whereas performance goals have been found to undermine intrinsic motivation and increase avoidance of difficult tasks. While it is reasonable to believe that this distinction applies to health behavior, it has not often been applied.

Goal Abandonment

Goal abandonment is common and often happens soon after goal setting. People frequently abandon their goals when other goals compete for time and energy or when there are no

facilitating goals that share a common means of pursuit. Research tends to focus on ways to bolster goal persistence, but this is not always the most advantageous response. Abandoning a goal that is not attainable is an adaptive strategy that can free up time and energy (Latham & Locke, 2007), reduce negative feelings that come from repeated failure to achieve goals, and minimize stress from setting goals without the proper resources to attain them (Schönpflug, 1986). Goal abandonment is also advantageous when goals are beyond personal control, such as the case of those who strive toward the goal of having a “perfect” thin body.

Goal Striving

Goal striving encompasses all actions, either deliberate or automatic, to achieve goals. This involves not only carrying out actions that directly promote goal attainment but also protecting those goals from being disrupted by competing goals or temptations. Individuals use many strategies in goal striving, which can be loosely categorized into planning strategies, automatic strategies, cognitive strategies, and effortful inhibition.

Planning Strategies

Planning strategies involve making arrangements before one is in a situation in which goal-directed behavior will be needed, so that either (a) temptation to engage in behavior counter to the goal can be avoided or (b) the individual is ready to meet the challenge when it occurs. By thinking ahead of time about potential obstacles that may arise, individuals can plan ways to overcome those obstacles. A simple example would be changing one’s route to work so that they do not pass the fast-food restaurant with a drive-through window, which they know would dangerously tempt them from their weight-loss goal. Choosing to keep tempting foods out of one’s home or at a distance are other examples.

Mentally rehearsing or simulating a strategy ahead of time is also a form of planning. People may envision themselves engaging in each of the steps necessary to achieve their goal so that they know what to expect and are ready with the appropriate response when the time comes. This strategy has been found effective among students mentally simulating the process of studying and preparing for exams, compared with students imagining the outcome of successful studying (Pham & Taylor, 1999). Focusing on the process rather than the outcome is an important aspect of the strategy, as only a process focus will reveal potential obstacles and challenges that may be encountered.

Automatic Strategies

Automatic strategies operate without an individual’s conscious awareness, or with less than full awareness. Automatic strategies are advantageous in many circumstances. They can operate when individuals are not motivated to comply with their goals and when individuals do not have sufficient attentional resources to dedicate to carefully monitoring and altering their behavior. When goal striving is automated, individuals are less likely to consciously talk themselves out of engaging in the relevant goal-directed behavior and are less susceptible to interference from competing goals.

Habits, once formed, operate automatically and can be an effective strategy for reaching a goal. Habits are formed when a behavior is paired with a particular environmental cue many times. After sufficient pairing, the behavior operates automatically whenever that environmental

cue is present. An individual may put on their seat belt when they get into their car so often that this becomes a habit, and they eventually do it without giving it any conscious thought.

Behaviors that only happen infrequently are less likely to become habits, as they do not have enough opportunities to be paired with a particular contextual cue. However, they may still be made automatic with the use of implementation intentions. Implementation intentions are if-then plans that specify a particular action to perform in a particular situation, such as “If I am in the produce section of the store, then I will buy apples.” Forming an implementation intention and repeating it several times has been shown to lead to improved regulation of many health-related behaviors (for further reading, see Gollwitzer & Sheeran, 2006).

Another way in which goal pursuit may become automatic is if alterations are made to one’s environment (planning) so that the goal-consistent behavior is the more likely response (or the only possible response). This may be possible by changing which behavioral options are available or possible, or by changing how the options are presented. Using “nudge”-type interventions, for example, keeps all options available but makes certain options more likely to be chosen, by, for example, making certain options more salient, closer, easier, more convenient, or cheaper. The fact that people have behaved based on responding to such changes is generally outside of their conscious awareness.

Cognitive Strategies

The category of cognitive strategies includes efforts to change the way one thinks about one’s goals and the obstacles to those goals. There are many ways one might think about the same goal or obstacle, and these different construals affect whether an individual is likely to be successful at attaining the goal. For example, temporal construal theory (Trope & Liberman, 2003) postulates that distant-future events are evaluated primarily in terms of their desirability, whereas near-future events are evaluated primarily in terms of their feasibility. When people set a goal, they tend to focus on desirable outcomes in the distant future, but during goal pursuit, these outcomes lose focus, challenges become more apparent, and persistence becomes more difficult, frequently leading to failure to achieve the goal. Individual differences in the extent to which people focus on proximal or distal goals influence goal attainment, with a more distal focus leading to better outcomes.

Individuals can be induced to focus on the future by having them think about possible events that could occur, and this serves as an effective self-regulatory strategy. It should be noted, however, that there is also evidence for the reverse position and that focusing on the means to success rather than on the ends of success leads to more successful goal pursuit and attainment, particularly if the task is difficult. Differences in these perspectives may be explained by the reward value of goal-directed behavior. A process focus may increase the intrinsic value of a rewarding behavior (e.g., engaging in a desired sport for exercise) but may simply highlight the drudgery of a less rewarding one (e.g., resisting desired foods).

Individuals also think about temptations and obstacles differently when those challenges are abstract than when they are specific. Challenges that are more vague and abstract seem less daunting than challenges that are concrete and detailed. For example, when children were trained to think about a tempting marshmallow in an abstract way (as “fluffy clouds”) instead of as a specific yummy treat, they were able to resist eating it for longer (Mischel, Shoda, & Rodriguez, 1989). Merely taking on an overall abstract mindset can work as well. Thinking about general categories of animals instead of specific animals led individuals in one study to think more abstractly, and they were more likely to successfully resist a tempting snack (Fujita & Han, 2009).

Effortful Inhibition of Impulses

The final category of goal striving strategies is effortfully inhibiting an impulse that would interfere with goal pursuit, by fighting off or suppressing the counterproductive thoughts, feelings, or behaviors. This subset of self-regulation processes is sometimes specifically referred to as self-control (although in many other cases, the terms self-control and self-regulation are used interchangeably), willpower, or delaying gratification. Effortfully inhibiting an impulse is, by definition, not automatic, as it requires conscious effort, and therefore it is subject to derailment or interference when these resources—whether they are cognitive or motivational—are scarce (for further reading, see Inzlicht & Schmeichel, 2012).

Dual-process models suggest that impulsive behaviors may be automatically enacted when there are not sufficient cognitive resources to challenge them. For example, when cognitive resources are scarce, individuals may not notice that they are violating their standards or may not have the ability to consider another option for how to behave. Individuals with impairments to their cognitive functioning, such as impaired working memory or executive function, have less success in regulating their behaviors. In addition, other factors that temporarily occupy these cognitive resources, including divided attention, alcohol intoxication, or fatigue, also reduce self-regulatory success.

Effortful inhibition may also be undermined by a lack of motivation. In particular, an extensive literature on the phenomenon of ego depletion shows that inhibiting one behavior makes it difficult to inhibit another behavior, due to the temporary depletion of a motivational resource. For example, in one study, after resisting eating a tempting cookie, participants performed worse on an unrelated self-regulation task, persisting in their efforts to solve a difficult puzzle (Baumeister, Bratslavsky, Muraven, & Tice, 1998). Even behaviors that seem to only require a small amount of effort to regulate (such as making a choice between two pens or suppressing one's facial expression) can still make it difficult to regulate another behavior soon after. Because of the ease and frequency with which both motivational and cognitive resources can become depleted, effortful inhibition is a strategy that is prone to failure.

While there is very little evidence that individuals can be taught to increase their effortful inhibition ability, there is some evidence that the depletion effect may be prevented with interventions that provide cash incentives, induce a positive mood, or reduce fatigue. In addition, depletion may be prevented by having participants form an implementation intention beforehand, by reminding them of their core values or even by teaching them that willpower is not limited.

Relying solely on effortful inhibition is not likely to be an effective strategy for goal attainment, but making use of multiple strategies may be more successful. For example, cognitive strategies might be used in an effort to help resist temptation, or planning strategies might be used so that individuals are less likely to encounter temptation. Similarly, if automatic strategies have been used to form habits or implementation intentions, effortful inhibition may not be necessary.

Future research on self-regulation might aim to focus on delineating the types of situations in which particular strategies may be effective, as well as the role of partners and family members in aiding an individual's self-control. This work may ultimately lead to effective interventions to help people successfully control their health behaviors.

Author Biographies

Richie L. Lenne is a PhD student in psychology at the University of Minnesota. He received his BA from Lewis & Clark College in 2011. He spent 3 years conducting research at the

Oregon Health & Science University before starting his doctorate. His research applies social psychological theory to public health and behavior change interventions with the dual purpose of informing theory and developing effective and efficient health programs.

Traci Mann is a professor of psychology at the University of Minnesota. She received her PhD in 1995 from Stanford University and spent ten years on the faculty at UCLA. She is interested in basic science questions about cognitive mechanisms of self-control and in applying social psychology research to promoting healthy behavior—particularly eating—in individuals' daily lives. Her research has been funded by NIH, NASA, and the USDA.

References

- Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, 50, 179–211.
- Alexy, B. (1985). Goal setting and health risk reduction. *Nursing Research*, 34, 283–288.
- Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology*, 74(5), 1252–1265. doi:10.1037/0022-3514.74.5.1252
- Covington, M. V. (2000). Goal theory, motivation, and school achievement: An integrative review. *Annual Review of Psychology*, 51(1), 171–200.
- Deci, E. L., Koestner, R., & Ryan, R. M. (1999). A meta-analytic review of experiments examining the effects of extrinsic rewards on intrinsic motivation. *Psychological Bulletin*, 125(6), 627–668; discussion 692–700. doi:10.1037/0033-2909.125.6.627
- Elliott, E. S., & Dweck, C. S. (1988). Goals: An approach to motivation and achievement. *Journal of Personality and Social Psychology*, 54(1), 5–12. doi:10.1037/0022-3514.54.1.5
- Fishbein, M., Triandis, H. C., Kanfer, F. H., Becker, M., Middlestadt, S. E., & Eichler, A. (2001). Factors influencing behavior and behavior change. In T. A. R. A. Baum & J. E. Singer (Eds.), *Handbook of health psychology* (pp. 3–17). Mahwah, NJ: Lawrence Erlbaum Associates.
- Gollwitzer, P. M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. *Advances in Experimental Social Psychology*, 38, 69–119. doi:10.1016/S0065-2601(06)38002-1
- Inzlicht, M., & Schmeichel, B. J. (2012). What is ego depletion? Toward a mechanistic revision of the resource model of self-control. *Perspectives on Psychological Science*, 7(5), 450–463. doi:10.1177/1745691612454134
- Kruglanski, A. W., Shah, J. Y., Fishbach, A., Friedman, R., Woo Young, C., & Sleeth-Keppler, D. (2002). A theory of goal systems. *Advances in Experimental Social Psychology*, 34, 331–378. doi:10.1016/S0065-2601(02)80008-9
- Latham, G. P., & Locke, E. A. (2007). New developments in and directions for goal-setting research. *European Psychologist*, 12(4), 290–300. doi:10.1027/1016-9040.12.4.290
- Lewin, K. (1935). *A dynamic theory of personality: Selected papers of Kurt Lewin*. New York, NY: McGraw-Hill.
- Linde, J. A., Jeffery, R. W., Finch, E. A., Ng, D. M., & Rothman, A. J. (2004). Are unrealistic weight loss goals associated with outcomes for overweight women? *Obesity Research*, 12(3), 569–576. doi:10.1038/oby.2004.65
- Locke, E. A., & Latham, G. P. (1990). *A theory of goal setting & task performance*. Englewood Cliffs, NJ: Prentice-Hall.
- Mann, T., Sherman, D., & Updegraff, J. (2004). Dispositional motivations and message framing: A test of the congruency hypothesis in college students. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, 23(3), 330–334. doi:10.1037/0278-6133.23.3.330

- Markus, H., & Nurius, P. (1986). Possible selves. *American Psychologist*, 41(9), 954–969. doi:10.1037/0003-066X.41.9.954
- Mischel, W., Shoda, Y., & Rodriguez, M. (1989). Delay of gratification in children. *Science*, 244(4907), 933–938. doi:10.1126/science.2658056
- Pham, L. B., & Taylor, S. E. (1999). From thought to action: Effects of process-versus outcome-based mental simulations on performance. *Personality and Social Psychology Bulletin*, 25(2), 250–260. doi:10.1177/0146167299025002010
- Schönplflug, W. (1986). Behavioral economics as an approach to stress theory. In M. H. Appley & R. Trumbull (Eds.), *Dynamics of stress: Physiological, psychological, and social perspectives* (pp. 81–98). New York, NY: Plenum Press.
- Silva, M. N., Markland, D., Carraça, E. V., Vieira, P. N., Coutinho, S. R., Minderico, C. S., ... Teixeira, P. J. (2011). Exercise autonomous motivation predicts 3-yr weight loss in women. *Medicine and Science in Sports and Exercise*, 43(4), 728–737. doi:10.1249/MSS.0b013e3181f3818f
- Trope, Y., & Liberman, N. (2003). Temporal construal. *Psychological Review*, 110(3), 403–421. doi:10.1037/0033-295X.110.3.403

Suggested Reading

- De Ridder, D. T. D., Lensvelt-Mulders, G., Finkenauer, C., Stok, F. M., & Baumeister, R. F. (2012). Taking stock of self-control: A meta-analysis of how trait self-control relates to a wide range of behaviors. *Personality and Social Psychology Review: An Official Journal of the Society for Personality and Social Psychology, Inc.*, 16(1), 76–99. doi:10.1177/1088868311418749
- Gollwitzer, P. M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. *Advances in Experimental Social Psychology*, 38, 69–119. doi:10.1016/S0065-2601(06)38002-1
- Inzlicht, M., & Schmeichel, B. J. (2012). What is ego depletion? Toward a mechanistic revision of the resource model of self-control. *Perspectives on Psychological Science*, 7(5), 450–463. doi:10.1177/1745691612454134
- Mann, T., de Ridder, D., & Fujita, K. (2013). Self-regulation of health behavior: Social psychological approaches to goal setting and goal striving. *Health Psychology*, 32(5), 487–498. doi:10.1037/a0028533

Sex Differences and Health

Vicki S. Helgeson and Katilyn Mascatelli

Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA

One of the seemingly contradictory findings in health psychology is the paradox that women are sicker than men, yet women outlive men. Women report feeling more pain and physical symptoms than men, perceive their health to be worse than men, and are more likely to be depressed than men. Yet men are more likely than women to die from 9 of the 10 leading causes of death in the United States. There are robust sex differences in psychological and physical health. The goal of this encyclopedia entry is first to orient the reader to the nature of these differences and then to offer explanations rooted in both culture and biology for the disparities. We use the phrase “sex differences” rather than “gender differences” when describing these statistics because we are comparing the biological categories rather than the social categories of male and female.

Mortality

There are long-standing sex differences in mortality in the United States. More males than females die at every age, with the ratio of male-to-female deaths peaking in adolescence and young adulthood. For example, for every 121 males who die between the ages of 20 and 24, 44 females die, which results in a male-to-female ratio of 2.75 (Centers for Disease Control and Prevention, 2015). This phenomenon makes the finding that women have longer life expectancies than men unsurprising. In 2013, the life expectancy was 81.2 years for females and 76.4 years for males (Centers for Disease Control and Prevention, 2015). This sex difference remains even when examining individual races and ethnicities. Life expectancy for Black women is 78.4 and for Black men is 72.3, whereas White women can expect to live to 81.4 and White men to 76.7 years. Latina women have a life expectancy of 83.8, and Latino men have a life expectancy of 79.1.

This sex difference in mortality favoring women has been documented for over a century, although the size of the difference has varied over the decades. In 1900, the life expectancy for

men was 46 years and for women was 48 years. As lifespans increased for both men and women over the twentieth century due to improvements in medicine and technology, the sex difference in mortality steadily increased, peaking in 1979 when women outlived men by nearly 8 years. This increased sex difference can be attributed to the reduction in women's mortality during childbirth and the increase in men's mortality from heart disease and lung cancer, which can be directly linked to smoking behavior. In recent decades, the sex difference in mortality has narrowed. This change has been credited to the relatively greater decrease in mortality from heart disease and cancer among men compared with women, in part due to men's higher quit rates of smoking compared with women.

Females outlive males in countries outside of the United States, but the magnitude of this difference is not consistent. Developing countries have shorter life expectancies than Westernized countries, and the sex difference in mortality is more variable and oftentimes smaller than that in more developed nations. The smaller sex difference in developing countries might be explained in part by the greater status difference between women and men, which contributes to female infanticide, higher maternal mortality, and poverty-linked mortality.

Morbidity

While lifespans have increased over the past century, rates of illness have increased. This trend reflects the fact that people who once would have died from acute illness live longer lives and acquire chronic diseases with which they can often live. In the early part of the twentieth century, it was much more common for people to die from acute illnesses such as influenza, but now they are able to survive these diseases and live long enough to contract illnesses that are more chronic in nature. Although men have higher death rates from the two major chronic diseases and the two leading causes of death (heart disease and cancer), women suffer from more acute and nonfatal chronic illnesses than men. Women suffer more than men from immune-related disorders, such as lupus, rheumatoid arthritis, and multiple sclerosis, as well as painful disorders, such as migraines, back pain, and carpal tunnel syndrome, and women report more overall disability than men and poorer self-ratings of health.

A primary contributor to the higher rate of morbidity among women compared with men is depression and depressive symptoms. Women experience more depressive symptoms than men in the general population and are also more likely than men to be diagnosed with clinical depression. This finding holds across many different countries, although the size of the difference varies. Interestingly, this difference emerges at the start of adolescence. That is, before age 13, boys and girls are equally likely to be depressed. Thus, any explanation of gender differences in depression must therefore account for this age-linked phenomenon.

Biology

There are a variety of biological explanations for sex differences in health. First, there are clearly some disorders that are linked to chromosomes that may place women or men at a disadvantage. Females are resistant to some X-linked chromosomal abnormalities because they have a second X chromosome. This may partly explain why women are less likely than men to suffer from hemophilia, meningitis, and muscular dystrophy. There are also disorders that are linked to the XX genotype, such as breast cancer, osteoarthritis, and glaucoma.

Hormones play a role in health, and men and women have varying levels of different hormones. Estrogen is a hormone that is thought to protect women from heart disease until menopause, after which estrogen levels decrease and women's levels of heart disease dramatically increase (Fetters, Peterson, Shaw, Newby, & Califf, 1996). The relation between estrogen and heart disease specifically and health more generally is not that simple, however. For example, oral contraceptives can increase the risk for heart disease (especially if combined with smoking), and hormone replacement therapy after menopause has not been linked to a reduction in heart disease (Lowe, 2004). Estrogen is thought to play a role in the development of some cancers, such as breast cancer. Thus, although hormones clearly play a role in health, they are likely to interact with genetic and environmental factors to influence health.

A third biological explanation for sex differences in health involves the immune system. It has been suggested that the nature of women's and men's immune systems differs, but the nature of these differences is not clear. Women are clearly more vulnerable than men to a host of immune-related disorders, such as lupus and rheumatoid arthritis. Immune factors also are likely to interact with hormones to influence health.

Health Behaviors

Smoking

There are a variety of health behaviors for which women and men differ that are directly linked to sex differences in morbidity and mortality. One is smoking. Smoking tobacco is one of the leading causes of heart disease and stroke and contributes to over a dozen kinds of cancer. While its ill effects are well known, the link between sex and smoking, as well as its long-term implications, is best understood within an historical perspective. In the early twentieth century, smoking was socially unacceptable for women. The smoking rate was twice as high among men than women, until the middle of the twentieth century. At that time, social norms relaxed, and advertising agencies focused their sights on selling cigarettes to women as part of a "liberated" lifestyle, a technique that allowed them to sell their product to a previously untapped other half of the population. Thus, smoking among women increased in the 1960s and 1970s as it became associated with women's fight for equality. In 1970, 31% of women and 44% of men were smokers. Overall smoking rates for both men and women have decreased dramatically as the health hazards of smoking became more widely known, and in 2013 about 15% of women and 20% of men smoked (Jamal et al., 2014).

Paralleling the changes in the rates of smoking for women and men, the sex difference in lung cancer has correspondingly shifted. The incidence of lung cancer has decreased for men but increased for women since the 1980s. In 1960, the male–female ratio of lung cancer was 6.7; in 1990, it was 2.3; and in 2015, it was projected to be 1.1 (American Cancer Society, 2015). Because lung cancer develops over the two to three decades following smoking, the changes in men's and women's rates of lung cancer can be directly tied to the changes in their rates of smoking.

Although both women and men have been quitting smoking, smoking cessation attempts are less successful for women than for men (Piper et al., 2010). Both nicotine replacement therapy and interventions that combine prescription smoking cessation medication with antidepressants are more effective in helping men than women quit smoking. One reason that women have more trouble quitting smoking than men is that women are more likely to be depressed than men, and smoking is more strongly associated with mood enhancement among women than men.

Other potential explanations are that smoking is more of a social experience for women than men and that women place higher value on the perceived weight-loss benefits of smoking than men.

Alcohol

Men drink more than women and have more alcohol-related problems than women. Women tend to have an older age of onset of regular drinking, to drink alcohol less frequently and to be more likely to abstain compared with men.

Regardless of the level of alcohol intake, the consequences of alcohol are different for women and men. It takes proportionately less alcohol to have the same effect on a woman as a man. For a woman and a man of the same weight who consume the same amount of alcohol, the woman will have a higher blood-alcohol level than the man due to the greater ratio of fat to water in a woman's compared with a man's body. This means that men have more water available in their systems to dilute consumed alcohol. The greater impact of alcohol on women compared with men may be why alcohol is more strongly associated with cirrhosis of the liver in women than in men. The progression from the first drink to an alcohol-related problem is faster among women than men. Thus, women are more vulnerable than men to both acute and chronic ailments of alcohol.

Despite women being more vulnerable to the health consequences of alcohol compared with men, men suffer more alcohol-related problems than women. The percentage of all deaths attributable to alcohol consumption is greater for males and females of all races: among Whites, 12.4% for males compared with 3.9% for females; among Blacks, 9.9% for males compared with 4.8% for females; among Native Americans, 25.8% for males compared with 11.6% for females; and among Asian/Pacific Islanders, 6.0% for males compared with 2.2% for females (Shield et al., 2013).

Obesity and Exercise

The United States has seen a dramatic rise in obesity rates over the past four decades. Men are more likely than women to be overweight, but women are more likely than men to be obese. Overweight is defined as a body mass index that exceeds 25, whereas obesity is defined as a body mass index that exceeds 30. In the majority of countries in the world, women are more likely than men to be obese. In the United States, the rate of obesity is the same for White women and men (33% women, 32% men), whereas the rate of obesity is much higher for females compared with males among Blacks (50 vs. 37%) and Hispanics (45 vs. 36%; Flegal, Carroll, Ogden, & Curtin, 2010). Aside from the fact that obesity is a risk factor for all causes of mortality for both women and men, the social and economic implications of obesity impact women more than men.

Women are more likely than men to be obese for two reasons. First, there are life events that are associated with obesity in women, such as having children and menopause. Women are also more likely to gain weight after marriage than men. Second, women are less likely than men to engage in physical activity, which is associated with a reduced risk of obesity. In addition, physical activity is associated with a lower risk of heart disease, hypertension, type 2 diabetes, and depression for both sexes. One reason that women have lower rates of exercise than men is that men tend to be motivated to be active by competition, whereas women are motivated by concerns about appearance and weight control. Unfortunately, body-shape motives are associated with lower levels of physical activity compared with other motives for exercise (Segar, Spruijt-Metz, & Nolen-Hoeksema, 2006).

Sexuality and Reproductive Health

The importance of understanding the impact of sexuality on health was brought to the forefront in recent decades, mostly due to the emergence of the AIDS epidemic. In particular, the idea that people belonging to sexual and gender minorities (i.e., lesbian, gay, bisexual, and transgender individuals, hereafter referred to as LGBT) might have unique healthcare needs gained validation only after the American Psychiatric Association acknowledged that homosexuality was not a psychiatric illness in 2000. LGBT youth are at greater risk for suicide attempts than non-LGBT youth (Silenzio, Pena, Duberstein, Cerel, & Knox, 2007). Lesbians are more likely than non-lesbian women to be overweight and obese (Boehmer, Bowen, & Bauer, 2007), putting them at risk for cardiovascular disease, glucose intolerance, and other obesity-related morbidities. These health problems are exacerbated by unique barriers LGBT persons encounter when attempting to access healthcare, such as reluctance to disclose sexual orientation to healthcare providers and structural barriers that impede access to health insurance for LGBT persons and their partners.

Although men tend to have more permissive attitudes about sex than women, this difference has decreased in magnitude over the past several decades, possibly due to an increase in women becoming more permissive. One potential reason for women's change in attitudes toward sex is the increased safety, efficacy, and availability of birth control. Virtually all women who have ever engaged in sexual intercourse in 2006–2010 have used some form of contraception, compared with about a third of women in the 1970s (Daniels, Mosher, & Jones, 2013). Today, the majority of women who use birth control are on oral contraceptives (the pill), although the proportion of women using other hormonal methods (the patch or shot) has increased from 4.5% in 1995 to 23% in 2006–2010 (Daniels et al., 2013). The proportion of women using long-acting reversible contraceptives (e.g., the intrauterine device) has also increased. Another tool women in the United States use to control their reproductive health is abortion, rates of which have been steadily declining over the past two decades. The abortion rate in 2010 for women aged 15–44 (14.6 per 1,000) was 32% lower than the rate in 1990 (27.4 per 1,000; Curtin, Abma, Ventura, & Henshaw, 2013). This decrease in the abortion rate has coincided with a national decline in the birth rate (16.7 per 1,000 women in 1990 compared with 13.0 per 1,000 women in 2010), and the most probable cause for these trends is improved or increased contraceptive use. By using birth control, women are able to delay, plan, and space their pregnancies.

Unintended pregnancy is not the only potential consequence of sexual activity—the risk of contracting a sexually transmitted infection (STI) is notably present for both males and females. In 2012, nearly 50,000 individuals were newly diagnosed; new cases occurred disproportionately among men who have sex with men, African Americans and people in their 20s (Centers for Disease Control and Prevention, 2014). The rate of HIV infections in the United States has remained stable since the late 1990s due to widespread prevention and testing efforts.

Sociocultural Factors

There are also a set of sociocultural factors that are implicated in the relation of gender to health. Here we consider three such factors: gender-related traits that are socialized in men and women, risky behavior that is socialized in men, and nurturant roles that are socialized in women.

Gender-Related Traits

As indicated in Janet Hyde's (2005) summary of 46 meta-analyses that compare women and men on a host of cognitive and social abilities, there are far more similarities than differences between men and women. Some of these differences may be attributed to the social roles that men and women possess in our society. Men are socialized to be agentic, and women are socialized to be communal. There is a strong body of research that shows the gender-related traits of agency and communion are more predictive of health than simply the categorical variable of sex (Helgeson, 2012).

In fact, there is strong evidence that agency is associated with good mental health outcomes and can account for sex differences in those outcomes (Helgeson, 2012). Agency is also related to good health practices, such as exercise, healthy eating, and good dental hygiene. However, communion is typically unrelated to health and health behaviors. Instead, communion is related to more supportive relationships and can account for sex differences in support. However, as discussed later, relationships are a double-edged sword for women. Thus, agency but not communion may account for some of men's lower rates of morbidity compared with that of women.

Aside from agency and communion, two more specific gender-related traits have been examined in their connection with health—unmitigated agency and unmitigated communion (Helgeson, 2012). Unmitigated agency is characterized by an excessive focus on the self and a negative view of others. Unmitigated agency has been linked to reckless driving, substance abuse, poor health behavior, risky behavior, and poor adjustment to disease (Helgeson, 2012). Unmitigated communion reflects a focus on others to the exclusion of the self and is defined by overinvolvement with others and self-neglect. Unmitigated communion has been linked to depressive symptoms, poor healthcare, and poor adjustment to disease (Helgeson, 2012). Even though some of the relations to health are the same, the reasons differ. For example, unmitigated agency is related to poor health behaviors due to feelings of superiority, beliefs in invulnerability, and possibly psychological reactance—that one does not need to engage in specific behaviors in order to be healthy. By contrast, unmitigated communion is related to poor health behavior due to an involvement with others that detracts from tending to one's own needs.

Risky Behavior

Men are socialized to take risks, whereas women are not. Parents are more encouraging of risk taking in boys than girls. For example, one study had parents watch a video of children on a playground and asked what they would say (Morrongiello & Dawber, 2000). Parents were more likely to intervene and warn of injury when they thought the child was a girl than a boy. Not surprisingly, men report engaging in more risky behaviors than women (Byrnes, Miller, & Schafer, 1999). As evidence of risk-taking behavior, men work in more hazardous jobs compared with women and have much higher rates of fatal work injuries compared with women; men handle more risky chores around the house compared with women, such as climbing on the roof; men are more aggressive drivers than women; and men engage in riskier leisure activities compared with women, such as skiing, mountain biking, and skydiving. Men are also more likely than women to own guns, and a greater use of guns contributes to a greater number of fatal gun accidents. Thus, risk-taking behavior may account partly for men's greater mortality from accidents and injuries compared with women. The male-to-female ratio of death from injuries—intentional and unintentional—persists across ethnic groups in the United States but is largest for Hispanic Whites and lowest for Asian/Pacific Islanders.

Nurturant Roles

Women report more supportive relationships than men, but these relationships have been cast as a double-edged sword. Relationships are not only sources of support for women but also sources of strain. That is, women not only receive but also provide support. Women are socialized to be caretakers, and caregiving has costs to health. According to the “nurturant role hypothesis,” women’s caretaking leads to increased exposure to communicable diseases, fatigue and vulnerability to illness from taking care of others, and self-neglect because caring for others takes precedence over caring for the self (Gove, 1984). In the United States, women are more likely than men to engage in volunteer work, women are more likely than men to be the primary caregivers of children, and women are more likely than men to take care of sick family members—spouses, parents, or children. In addition, caregiving is associated with greater burden and greater distress among women than men (Pinquart & Sörensen, 2006).

Conclusion and Future Directions

Health—psychological and physical—is one domain for which sex differences persist, although the size of these differences has shifted substantially over the last century. We reviewed several different classes of explanations for these differences—those involving biology, health behaviors, reproduction, and sociocultural variables – but note that these explanations have largely been examined in isolation of one another. It is increasingly recognized that biological, behavioral, and social factors are likely to interact to influence health. Future research aimed to increase our understanding of sex differences in health would benefit from a multidisciplinary approach. Research in this area also is expanding in terms of the methods used to study sex differences in health. Correlational research is being replaced with longitudinal studies, research on middle-class White persons is increasingly cross-cultural, and experimental approaches have been used to understand how we react to men and women as well as how hormones influence behavior. Finally, it is increasingly recognized that the category of being male versus female intersects with multiple other categories, such as race, ethnicity, and social class. Future research on sex differences in health would benefit from incorporating greater intersectionality in its approach.

Author Biographies

Vicki S. Helgeson is professor of psychology at Carnegie Mellon University where she studies the intersections of gender, relationships, and health. She studies how people adjust to chronic illness with a focus on the implications of gender-role socialization and social environmental factors for health. Her most recent work involves couples in which one person has diabetes, with a focus on communal coping. She recently completed the 5th revision of her textbook titled *Psychology of Gender*.

Katilyn Mascatelli is a graduate student in the social/health psychology program at Carnegie Mellon University in Pittsburgh, Pennsylvania. Her research interests include gender, stereotyping, and discrimination based on violations of gender stereotypes.

References

- American Cancer Society. (2015). Cancer facts & figures 2015. Retrieved from <http://www.cancer.org/acs/groups/content/@editorial/documents/document/acspc-044552.pdf>

- Boehmer, U., Bowen, D. J., & Bauer, G. R. (2007). Overweight and obesity in sexual-minority women: Evidence from population-based data. *American Journal of Public Health, 97*(6), 1134–1140. doi:10.2105/AJPH.2006.088419
- Byrnes, J. P., Miller, D. C., & Schafer, W. D. (1999). Gender differences in risk taking: A meta-analysis. *Psychological Bulletin, 125*, 367–383. doi:10.1037/0033-2909.125.3.367
- Centers for Disease Control and Prevention. (2014). Sexually transmitted disease surveillance 2012. Retrieved from <http://www.cdc.gov/std/stats12/surv2012.pdf>
- Centers for Disease Control and Prevention. (2015). Deaths: Final data for 2013 (Vol. 64). Retrieved from http://www.cdc.gov/nchs/data/nvsr/nvsr64/nvsr64_02.pdf
- Curtin, S. C., Abma, J. C., Ventura, S. J., & Henshaw, S. (2013). Pregnancy rates for U.S. women continue to drop. *National Center for Health Statistics, 136*, 1–8.
- Daniels, K., Mosher, W. D., & Jones, J. (2013). Contraceptive methods women have ever used: United States, 1982–2010. *National Health Statistics Reports, 62*, 1–15.
- Fetters, J. K., Peterson, E. D., Shaw, L. J., Newby, K., & Califf, R. M. (1996). Sex-specific differences in coronary artery disease risk factors, evaluation, and treatment: Have they been adequately evaluated? *American Heart Journal, 131*(4), 796–813. doi:10.1016/S0002-8703(96)90289-6
- Flegal, K. M., Carroll, M. D., Ogden, C. L., & Curtin, L. R. (2010). Prevalence and trends in obesity among U.S. adults, 1999–2008. *Journal of the American Medical Association, 303*, 235–241. doi:10.1001/jama.2009.2014
- Gove, W. R. (1984). Gender differences in mental and physical illness: The effects of fixed roles and nurturant roles. *Social Science & Medicine, 19*(2), 77–91. doi:10.1016/0277-9536(84)90273-9
- Helgeson, V. S. (2012). Gender and health: A social psychological perspective. In A. Baum, T. Revenson, & J. Singer (Eds.), *Handbook of health psychology* (2nd ed., pp. 519–537). New York, NY: Psychology Press.
- Hyde, J. S. (2005). The gender similarities hypothesis. *American Psychologist, 60*, 581–592. doi:10.1037/0003-066X.60.6.581
- Jamal, A., Agaku, I. T., O'Connor, E., King, B. A., Kenemer, J. B., & Neff, L. (2014). Current cigarette smoking among adults—United States, 2005–2013. *Morbidity and Mortality Weekly Report, 63*(47), 1108–1112.
- Lowe, G. D. O. (2004). Hormone replacement therapy and cardiovascular disease: Increased risks of venous thromboembolism and stroke, and no protection from coronary heart disease. *Journal of Internal Medicine, 256*, 361–374. doi:10.1111/j.1365-2796.2004.01400.x
- Morrongioello, B. A., & Dawber, T. (2000). Mothers' responses to sons and daughters engaging in injury-risk behaviors on a playground: Implications for sex differences in injury rates. *Journal of Experimental Child Psychology, 76*, 89–103. doi:10.1006/jecp.2000.2572
- Pinquart, M., & Sörensen, S. B. (2006). Gender differences in caregiver stressors, social resources, and health: An updated meta-analysis. *Journal of Gerontology, 61B*, 33–45. doi:10.1093/geronb/61.1.P33
- Piper, M. E., Cook, J. W., Schlam, T. R., Jorenby, D. E., Smith, S. S., Bolt, D. M., & Loh, W. Y. (2010). Gender, race, and education differences in abstinence rates among participants in two randomized smoking cessation trials. *Nicotine & Tobacco Research, 12*, 647–657. doi:10.1093/ntr/ntq067
- Segar, M., Spruijt-Metz, D., & Nolen-Hoeksema, S. (2006). Go figure? Body-shape motives are associated with decreased physical activity participation among midlife women. *Sex Roles, 54*, 175–187. doi:10.1007/s11199-006-9336-5
- Shield, K. D., Gmel, G., Kehoe-Chan, T., Dawson, D. A., Grant, B. F., & Rehm, J. (2013). Mortality and potential years of life lost attributable to alcohol consumption by race and sex in the United States in 2005. *PLoS One, 8*, e51923. doi:10.1371/journal.pone.0051923
- Silenzio, V. M., Pena, J. B., Duberstein, P. R., Cerel, J., & Knox, K. L. (2007). Sexual orientation and risk factors for suicidal ideation and suicide attempts among adolescents and young adults. *American Journal of Public Health, 97*(11), 2017–2019. doi:10.2105/AJPH.2006.095943

Suggested Reading

- Bogart, L. M., Revenson, T. A., Whitfield, K. E., & France, C. R. (2014). Introduction to the special section on lesbian, gay, bisexual, and transgender (LGBT) health disparities: Where we are and where we're going. *Annals of Behavioral Medicine*, 47, 1–4. doi:10.1007/s12160-013-9574-7
- Case, A., & Paxson, C. (2005). Sex differences in morbidity and mortality. *Demography*, 42, 189–214. doi:10.1353/dem.2005.0011
- Fischer, J., Jung, N., Robinson, N., & Lehmann, C. (2015). Sex differences in immune responses to infectious diseases. *Infection*, Advance online publication. doi:10.1007/s15010-015-0791-9
- Rogers, R. G., Everett, B. G., Onge, J. M. S., & Krueger, P. M. (2010). Social, behavioral, and biological factors, and sex differences in mortality. *Demography*, 47(3), 555–578. doi:10.1353/dem.0.0119
- Sanchez-Lopez, M. d. P., & Cuellar-Flores, I. (2012). The impact of gender roles on health. *Women & Health*, 52, 182–196. doi:10.1080/03630242.2011.652352

Social-Evaluative Threat

Sally S. Dickerson

Department of Psychology, Pace University, New York, NY, USA

Have you ever thought that others were negatively judging you while you were giving an important talk or presentation? Have you felt criticized by a relationship partner, friend, or acquaintance or felt that you were judged or rejected by others on the basis of a stigma, devalued characteristic, or an aspect of your identity? These types of situations can be inherently difficult, painful, and threatening—and they all share an underlying core element of social evaluation. Social-evaluative threat (SET) occurs when an important aspect of the self could be judged by others (Dickerson & Kemeny, 2004). Theoretical and empirical work has documented how these social-evaluative situations may be associated with patterns of emotional and physiological reactivity.

Why are social-evaluative situations threatening? Humans have a fundamental need to belong, and maintaining the social acceptance of others is critical to well-being and survival (Baumeister & Leary, 1995; Holt-Lunstad, Smith, & Layton, 2010). Situations with the potential for evaluation or rejection therefore jeopardize this fundamental goal. We have proposed that social-evaluative situations may elicit a coordinated psychobiological response, characterized by emotional and physiological changes (e.g., Dickerson, Gruenewald, & Kemeny, 2004). Our theoretical and empirical work has documented that SET can lead to increases in self-conscious cognitions and emotions (e.g., shame, embarrassment) and other negative cognitive and affective states. Physiologically, SET can elicit increases in cortisol, an important hormone that is involved in energy mobilization, and can help organize the body's response to stressors; SET can also lead to changes in the cardiovascular and immune systems. If the physiological activation elicited by SET is experienced chronically or persistently, there could be implications for health.

Social-Evaluative Threat and Cortisol Reactivity

A great deal of research has tested whether SET can elicit the hormone cortisol. In a meta-analytic review of 208 acute laboratory stressor studies that assessed cortisol as an outcome

(Dickerson & Kemeny, 2004), we found that stressors characterized by SET (e.g., evaluative audience present) were associated with substantially larger cortisol responses than those without this element (e.g., completing difficult math problems alone). The effect size for social-evaluative stressors was nearly three times the magnitude of the non-SET tasks ($d = 0.67$ vs. $d = 0.15$). The increase in cortisol was particularly pronounced when stressors were both *social-evaluative* and *uncontrollable*. Social-evaluative, uncontrollable stressors were associated with large increases in cortisol ($d = 0.92$); however, those without either element did not on average lead to increases in this parameter. Taken together, this meta-analysis demonstrates that social-evaluative stressors—particularly when uncontrollable—can elicit strong, substantial cortisol reactivity.

Subsequent studies have manipulated the social-evaluative context of the stressor to experimentally test the conditions in which cortisol reactivity is observed. Gruenewald, Kemeny, Aziz, and Fahey (2004) randomly assigned participants to deliver a speech and complete a computerized math task either in front of an evaluative audience (SET) or alone in a room (non-SET). Participants in both conditions reported that the task was challenging and difficult and required effort (e.g., “stressful”); however, only the SET condition elicited increases in cortisol. Performing the identical task in the absence of social evaluation did not lead to cortisol changes, demonstrating that the social-evaluative nature of the stressor can shape the physiological response.

This SET manipulation has now been used in a number of studies and the primary findings replicated using laboratory performance stressors (e.g., Bosch et al., 2009; Dickerson, Mycek, & Zaldivar, 2008; Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009; Taylor et al., 2010) and physical stressors (e.g., cold pressor task; Schwabe, Haddad, & Schachinger, 2008). Other studies have examined whether naturally occurring instances of SET (e.g., real-life competitions) can also elicit cortisol reactivity. Rohleder, Beulen, Chen, Wolf, and Kirschbaum (2007) collected cortisol from ballroom dancers during both rehearsal and competition days. They found that cortisol levels were much higher on the competition days, when their performances were being judged by an evaluative panel, compared with days in which they were rehearsing (and not evaluated). Further, the dancers who reported feeling more stressed by the judges also showed greater increases in cortisol. Another study examined cortisol responses during a competitive audition for a musical theater production (Boyle, Lawton, Arkbage, Thorell, & Dye 2013). Audition exposure led to increases in cortisol, and cortisol reactivity correlated with subjective ratings of SET. Taken together, these studies demonstrate that naturally occurring performance contexts characterized by social evaluation can also trigger increases in cortisol and that cortisol can be linked to perceptions of social evaluation.

Components of Social-Evaluative Threat Linked with Cortisol Reactivity

We have been interested in unpacking the critical components of the social-evaluative context that elicit increases in cortisol. For example, is the mere social presence of others enough to trigger cortisol reactivity, or does the social evaluation need to be explicit to lead to increases in this parameter? To test this question, we conducted a study with three experimental groups (Dickerson et al., 2008). Similar to Gruenewald et al. (2004), participants delivered a speech in front of an evaluative audience (SET) or alone in a room (non-SET). We also included a third experimental condition, in which a research assistant was inattentive (working on a laptop in the room) (PRES). We found that the SET condition elicited a robust increase in cortisol;

however, the non-SET and PRES conditions did not lead to increases in this parameter. This suggests that simply having others present during a performance stressor may not be enough to elicit increases in cortisol; instead, the perceived evaluation of others may be critical for activating this physiological system.

Others have examined whether SET that occurs remotely can trigger cortisol reactivity; do the evaluative others need to be physically present to elicit a response? Several studies have found that remote evaluation (e.g., participants believe they are being evaluated through a one-way mirror) is sufficient to increase cortisol levels (e.g., Kelly, Matheson, Martinez, Merali, & Anisman, 2007); evaluation by avatars in an immersive virtual reality environment has also been shown to elicit cortisol reactivity (Fallon, Careaga, Sbarra, & O'Connor, 2016). One study that experimentally manipulated whether the evaluators were physically present or remote (virtual audience or one-way mirror; Kelly et al., 2007) found having an audience that was physically present elicited about cortisol increases of about three times the magnitude of more remote forms of social evaluation. While future research should test key moderators (e.g., immersive nature or believability of the remote evaluation), it is clear that when perceptions of evaluation are evoked (whether the evaluation is in person or virtual), the cortisol system can be activated.

The threat inherent in a social-evaluative context is multidimensional and could lead to concerns of loss of social standing or loss of acceptance. Smith and Jordan (2015) conducted a study to delineate whether status or acceptance concerns are driving cortisol responses to SET. They randomly assigned participants to complete a public speaking task in one of four conditions: a low-threat condition, in which they were told they would not be evaluated; a high-acceptance threat condition, in which they would be judged in terms of how likeable, friendly, and interesting they were; a high-status threat condition, in which they were judged on how competent, skilled, and intelligent they appeared; and a high-acceptance/high-status condition, where the instructions were combined. They found that both status and acceptance threats elicited greater increases in cortisol compared with the low-threat condition and the largest increases were observed for the high-status/high-acceptance threat group. This suggests that both status concerns and acceptance concerns can lead to increases in cortisol and contexts that trigger both are potent elicitors of this physiological parameter.

The behavior of the evaluators could also be an element that influences cortisol reactivity to SET. Cundiff, Smith, Baron, and Uchino (2016) found that an evaluative interaction with a highly dominant individual (e.g., assertive voice) led to greater cortisol responses compared with interactions with someone low on dominance (e.g., spoke deferentially). This suggests that qualities and behaviors of the evaluative others can also shape responses to SET.

Taken together, these experimental studies demonstrate that contexts that heighten the evaluative potential (e.g., explicit evaluation, dominant interaction partner, threaten salient goals) lead to pronounced increases in cortisol. Future research should continue to unpack the critical elements of the social-evaluative situation that trigger this system and identify important moderators of reactivity.

Emotional and Cognitive Effects of Social-Evaluative Threat

SET can elicit robust increases in cortisol, but what are the emotional experiences of individuals in these situations? We have been interested in the affective changes that occur under social-evaluative contexts. Consistent with theoretical and empirical literature that self-conscious emotions and affective self-evaluative states are sensitive to assessments of declining status or

acceptance (e.g., Gilbert, 2000; Leary, 2007), we have found that the SET conditions are associated with greater increases in self-conscious cognitions and emotions compared with the non-SET conditions (Dickerson et al., 2008; Gruenewald et al., 2004). Further, there were no differences between conditions in other emotional states, such as fear or sadness, demonstrating that the self-conscious states were most sensitive to the social-evaluative context of the stressor. In both of these studies, we also found correlations between the self-conscious emotions and cortisol; those who showed the greatest increases in self-conscious emotions also showed the greatest increases in cortisol. There was not a relationship between cortisol reactivity and other negative emotions assessed. Taken together, this suggests that social-evaluative conditions can elicit both self-conscious emotions and cortisol reactivity and these systems could be activated in parallel under SET. However, this self-conscious emotion/cortisol association could vary depending on the type of SET experienced (e.g., status vs. acceptance threats; Smith & Jordan, 2015) or individual difference factors (e.g., subjective social status; Gruenewald, Kemeny & Aziz, 2006); additional research on the emotional correlates of SET, and accompanying physiological changes, is warranted.

For some, the social-evaluative experience may not end when the interaction is over. SET can lead to rumination, or repetitive, unwanted, past-oriented thoughts. In our experimental studies that manipulate SET, we have assessed post-task negative thoughts to test if social-evaluative contexts are more likely to trigger rumination and if rumination is linked to persistent cortisol activation. Consistent with this premise, we have found that explicit social-evaluative conditions elicit more negative thoughts (e.g., “I looked stupid”) in the 10 min post-task compared with other conditions (Zoccola, Dickerson, & Zaldivar, 2008). Additionally, those who reported higher levels of post-task rumination also had greater cortisol reactivity, and levels remained elevated longer compared with those who reported lower levels of rumination following the stressor.

We conducted another study to test whether the SET-rumination link persists beyond the immediate post-task period (Zoccola, Dickerson, & Lam, 2012). Replicating our previous finding, post-task rumination was higher in the SET condition compared with a non-SET condition 10 min post-stressor. In this study, we also assessed rumination 40 min post-task, later that evening and 3–5 days later. At all time points, participants in the social-evaluative condition reported higher levels of rumination compared with those in the non-SET condition. Further, we found this effect was mediated by self-conscious cognitions and emotions; the SET condition led to greater increases in self-conscious cognitions and emotions, which in turn was associated with greater rumination. This demonstrates that SET contexts can elicit ruminative thought even days beyond the initial stressor, potentially through the ability to elicit self-conscious cognitions and emotions.

Social-Evaluative Threat Activates Other Physiological Systems

While much of the research on SET has examined activation of the cortisol system, these situations can elicit changes in a wide range of physiological parameters. SET can lead to increases in cardiovascular (e.g., systolic blood pressure, diastolic blood pressure, heart rate) and sympathetic (e.g., epinephrine, norepinephrine, alpha-amylase) activity in both laboratory (Kirschbaum, Pirke, & Hellhammer, 1993; Nater & Rohleder, 2009) and naturally occurring contexts (Lehman & Conley, 2010). Studies that have manipulated the social context of the stressor often observe greater cardiovascular reactivity under conditions that were social evaluative or where the evaluative potential was emphasized (e.g., Bosch et al., 2009).

SET can also lead to changes in immunologic parameters. For example, we conducted a study in which we randomly assigned participants to a SET or non-SET laboratory stressor (Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009). We found that the SET condition elicited increases in the production of the pro-inflammatory cytokine TNF- α compared with a non-SET condition. Further, we found that those who felt the most evaluated during the task showed the greatest increases in TNF- α production, suggesting a link between perceptions of social evaluation and markers of inflammation. Future research should examine the different systems that can be activated under SET and their interactions.

Implications for Health

While acute instances of SET can elicit short-term increases in cortisol and changes in other systems, experiencing chronic forms of SET could take a physiological toll. For example, a meta-analysis found that chronic social stressors were associated with higher morning and evening cortisol levels compared with nonsocial stressors (Miller, Chen, & Zhou, 2007). Over-activation and/or dysregulation in cortisol and other physiological systems is thought to impact health over time and put individuals at greater risk of disease (e.g., McEwen, 1998). Future research should pair acute laboratory reactivity methodologies with longitudinal assessments, to further clarify how patterns of physiological reactivity to SET may be associated with long-term negative health outcomes.

Author Biography

Sally S. Dickerson is the associate provost for research and a professor of psychology at Pace University. Her research integrates psychological and biological levels of analysis by applying theories of social and emotional processes to the context of stress and disease. She examines how social-evaluative threat and accompanying self-conscious emotional responses affect neuroendocrine and immune outcomes and how individual differences may heighten vulnerability to these psychological and physiological changes.

References

- Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, 117, 497–529. <http://dx.doi.org/10.1037/0033-2909.117.3.49>
- Bosch, J. A., De Gues, E. J. C., Carroll, D., Goedhart, A. D., Anane, L. A., Veldhuizen, et al. (2009). A general enhancement of autonomic and cortisol responses during social-evaluative threat. *Psychosomatic Medicine*, 71, 877–885. <http://dx.doi.org/10.1097/PSY.0b013e3181baef05>
- Boyle, N. B., Lawton, C., Arkbage, K., Thorell, L., & Dye, L. (2013). Dreading the boards: Stress response to a competitive audition characterized by social-evaluative threat. *Anxiety, Stress and Coping*, 26, 690–699. <http://dx.doi.org/10.1080/10615806.2013.766327>
- Cundiff, J. M., Smith, T. W., Baron, C. E., & Uchino, B. N. (2016). Hierarchy and health: Physiological effects of interpersonal experiences associated with socioeconomic position. *Health Psychology*, 35, 356–365. <http://dx.doi.org/10.1037/hea0000227>
- Dickerson, S. S., Gable, S. L., Irwin, M. R., Aziz, N., & Kemeny, M. E. (2009). Social-evaluative threat and proinflammatory cytokien regulation: An experimental laboratory investigation. *Psychological Science*, 20, 1237–1244. <http://dx.doi.org/10.1111/j.1467-9280.2009.02437.x>

- Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality*, 72, 1191–1216. <http://dx.doi.org/10.1111/j.1467-6494.2004.00295.x>
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130, 355–391. <http://dx.doi.org/10.1037/0033-2909.130.3.355>
- Dickerson, S. S., Mycek, P. M., & Zaldivar, F. P. (2008). Negative social evaluation, but not mere social presence, elicits cortisol responses to a laboratory stressor task. *Health Psychology*, 27, 116–121. <http://dx.doi.org/10.1037/0278-6133.27.1.116>
- Fallon, M. A., Careaga, J. S., Sbarra, D. A., & O'Connor, M. F. (2016). Utility of a virtual Trier Social Stress Test: Initial findings and benchmarking comparisons. *Psychosomatic Medicine*, 78, 835–840. <http://dx.doi.org/10.1097/PSY.0000000000000338>
- Gilbert, P. (2000). Varieties of submissive behavior as forms of social defense: Their evolution and role in depression. In L. Sloman & P. Gilbert (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy* (pp. 3–45). Mahwah, NJ: Erlbaum.
- Gruenewald, T. L., Kemeny, M. E., & Aziz, N. (2006). Subjective social status moderates cortisol responses to threat. *Brain, Behavior and Immunity*, 20, 410–419. <https://doi.org/10.1016/j.bbi.2005.11.005>
- Gruenewald, T. L., Kemeny, M. E., Aziz, N., & Fahey, J. L. (2004). Acute threat to the social self: Shame, social self-esteem, and cortisol activity. *Psychosomatic Medicine*, 66, 915–924. <http://dx.doi.org/10.1097/01.psy.0000143639.61693.ef>
- Het, S., Rohleder, N., Schoofs, D., Kirschbaum, C., & Wolf, O. T. (2009). Neuroendocrine and psychometric evaluation of a placebo version of the “Trier Social Stress Test”. *Psychoneuroendocrinology*, 34, 1075–1086. <http://dx.doi.org/10.1016/j.psyneuen.2009.02.008>
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7, e1000316. <https://doi.org/10.1371/journal.pmed.1000316>
- Kelly, O., Matheson, M., Martinez, A., Merali, Z., & Anisman, H. (2007). Psychosocial stress evoked by a virtual audience: Relation to neuroendocrine activity. *Cyberpsychology & Behavior*, 10(5), 655–662. <http://dx.doi.org/10.1089/cpb.2007.9973>
- Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The “Trier Social Stress Test”—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76–81. <http://dx.doi.org/10.1159/000119004>
- Leary, M. R. (2007). Motivational and emotional aspects of the self. *Annual Review of Psychology*, 58, 317–344. <http://dx.doi.org/10.1146/annurev.psych.58.110405.085658>
- Lehman, B. J., & Conley, K. M. (2010). Momentary reports of social-evaluative threat predict ambulatory blood pressure. *Social Psychological and Personality Science*, 1, 51–56. <http://dx.doi.org/10.1177/1948550609354924>
- McEwen, B. S. (1998). Stress, adaptation, and disease. Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 840, 33–44. <http://dx.doi.org/10.1111/j.1749-6632.1998.tb09546.x>
- Miller, G. E., Chen, E., & Zhou, E. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, 133, 25–45. <http://dx.doi.org/10.1037/0033-2909.133.1.25>
- Nater, U. M., & Rohleder, N. (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology*, 34, 486–496. <http://dx.doi.org/10.1016/j.psyneuen.2009.01.014>
- Rohleder, N., Beulen, S. E., Chen, E., Wolf, J. M., & Kirschbaum, C. (2007). Stress on the dance floor: The cortisol stress response to social-evaluative threat in competitive ballroom dancers. *Personality and Social Psychology Bulletin*, 33, 69–84. <http://dx.doi.org/10.1177/0146167206293986>
- Schwabe, L., Haddad, L., & Schachinger, H. (2008). HPA axis activation by a socially evaluated cold-pressor task. *Psychoneuroendocrinology*, 33, 890–895. <http://dx.doi.org/10.1016/j.psyneuen.2008.03.001>

- Smith, T. W., & Jordan, K. D. (2015). Interpersonal motives and social-evaluative threat: Effects of acceptance and status stressors on cardiovascular reactivity and salivary cortisol response. *Psychophysiology*, 52, 269–276. <http://dx.doi.org/10.1111/psyp.12318>
- Taylor, S. E., Seeman, T. E., Eisenberger, N. I., Kozanian, T. A., Moore, A. N., & Moons, W. G. (2010). Effects of a supportive or unsupportive audience on biological and psychological responses to stress. *Journal of Personality and Social Psychology*, 98, 47–56. <http://dx.doi.org/10.1037/a0016563>
- Zoccola, P. M., Dickerson, S. S., & Lam, S. (2012). Eliciting and maintaining ruminative thought: The role of social-evaluative threat. *Emotion*, 12, 673–677. <http://dx.doi.org/10.1037/a0027349>
- Zoccola, P. M., Dickerson, S. S., & Zaldivar, F. P. (2008). Rumination and cortisol responses to laboratory stressors. *Psychosomatic Medicine*, 70, 661–667. <http://dx.doi.org/10.1097/PSY.0b013e31817bbc77>

Suggested Reading

- Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2011). Physiological effects of social threat: Implications for health. In J. Decety & J. Cacioppo (Eds.), *Handbook of social neuroscience* (pp. 787–803). New York, NY: Oxford University Press.
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130, 355–391.
- Miller, G. E., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biological plausible models linking the social world and physical health. *Annual Review of Psychology*, 60, 501–524.
- Zoccola, P. M., & Dickerson, S. S. (2012). Assessing the relationship between rumination and cortisol: A review. *Journal of Psychosomatic Research*, 73, 1–9.

Social Factors in Diet and Obesity

Gbolahan O. Olanubi and A. Janet Tomiyama

Psychology Department, University of California Los Angeles, Los Angeles, CA, USA

Introduction

When attempting to understand factors that influence diet, or food choice, people are quick to assume that people eat when they are hungry and stop when they are full. Hunger, however, turns out to be one of the least important factors in eating. This is because there are a multitude of other social, economic, and psychological factors at play that shape our eating choices, which can over time affect weight and body mass index (BMI).

The purpose of this encyclopedia entry is to describe the social factors that are involved in eating, dietary choices, and obesity. We organize our discussion of social factors roughly from the macro- to microlevel. The literatures we cover are enormous, but space and reference constraints allow only for highlights of each topic. Throughout the entry, we note when there are seminal review papers in each area. Ultimately, our goal is to highlight the role of social factors in diet and obesity and to position psychology in its rightful place as a key player in diet and obesity research.

Culture

Culture has been shaping food choice for millennia. Rozin and Vollmecke (1986) noted that if you want to know as much as possible about a person's food preference, simply ask, "What is your culture or ethnic group?" Culture, a way of life of a society or an amalgam of shared practices and meanings among a group that guides interactions and is shaped by experience, plays a major role in diet and food choice. Culture influences attitudes toward food, our preferences, and perceptions of what is healthy compared with what is unhealthy.

Everyone needs to eat, but not everyone needs to be happy about eating. Among Americans there is an attitude not only that food is "nutrient," a source of nourishment, but also that it may be dangerous—almost as dangerous as abstaining from food (Rozin, 1996). Americans

also endorse attitudes that even trace amounts of salt and fat may be harmful (Rozin, 1996). In contrast to American attitudes, the French have a more positive attitude toward food, viewing it as both nutrient and a source of pleasure. Considering these cultural differences in attitudes, there are also cultural differences in consumption. The French, compared with Americans, eat more high-fat foods (yet tend to be healthier—a phenomenon known as the French paradox; Ferrières, 2004). Although Americans have more negative attitudes than the French about food, Americans eat larger servings of food.

Expanding on this research Rozin, Fischler, Imada, Sarubin, and Wrzesniewski (1999) examined cultural differences in food attitudes and behavior in the United States, France, Belgium, and Japan. Congruent with previous research, they found that Americans had the highest concern about healthiness of food choice, worried about the fattening effects of food as opposed to the savoring of food, and were more likely to make nutritional, as opposed to culinary, associations with food. Americans also had the lowest food-positive attitudes, considering food less important and drawing little pleasure from food. Finally, despite the worry, concern, and attitudes toward food, Americans also reported viewing themselves as the least healthy eaters compared with the other countries.

Socioeconomic Status

Food choice is predicated upon access to foods. Socioeconomic status, comprising income, education, and occupation, plays a focal role in determining purchasing power and food choice. Cost has been one of the most important factors in food choice for low-income individuals. Ironically, the cost of food is actually more expensive in low-income neighborhoods compared with high-income neighborhoods because of a dearth of low-cost supermarkets and presence of higher-priced convenience stores (see Morland, Wing, Roux, & Poole, 2002).

If low-income families have a restricted budget but more expensive foods, what are they eating? Low-income families may be cornered into low-cost energy dense diets (Darmon, Ferguson, & Briend, 2003). Energy density refers to calories per edible weight of a food item. Energy dense diets consist of eating foods high in energy density (cereals, meats, sweets) more so than foods low in energy density (fruits, vegetables, whole grains). A low-cost energy dense diet would essentially attempt to maximize calories per dollar while still maintaining sufficient nutrients.

Supporting this perspective, Darmon et al. (2003) used a computer simulation to model the food choices of individuals under economic constraint. Under increasing economic constraint the model selected for less fruits and vegetables (FV), meats, fish, and cheese and higher contents of milk, sweets, and starchy foods compared with a normal diet.

Fortunately, this barrier appears surmountable. Researchers have intervened to remove financial barriers to increase selection and consumption of FV for low-income individuals and found that subsidizing FV costs increases consumption for low-income individuals (Anderson et al., 2001).

Social Norms

Norms surrounding food consumption are powerful drivers of eating behavior (see Herman, Roth, & Polivy, 2003). In general, an inhibitory social norm surrounding eating exists in Western countries, based on widespread weight stigma and negative stereotypes of those who

overeater (Vartanian, Herman, & Polivy, 2007). This inhibitory norm appears to be most pronounced in eating among strangers, due to impression management concerns, and true particularly for women, who are seen as more feminine and socially appealing when they eat less (Vartanian et al., 2007). Inhibitory norms are highest with a non-eating observer, according to a meta-analysis of studies that experimentally manipulated awareness of one's eating being observed (Robinson, Hardman, Halford, & Jones, 2015).

In contrast, eating in the presence of close others appears to trigger social facilitation of eating (de Castro, 2002). In one illuminating study, Howland, Hunger, and Mann (2012) experimentally manipulated eating norms in existing friend groups. Two members of a friend trio were enlisted to set an eating norm, and the third member's eating was examined. Participants followed whatever eating norm was set, and these eating norms carried over into situations where participants were then eating alone.

A subtype of norms are informational norms, wherein information about eating behavior is communicated (e.g., through visual cues of leftover food). In a meta-analysis of 15 studies examining experimental manipulations, Robinson, Thomas, Aveyard, and Higgs (2014) found that both high and low informational intake norms had moderate effects on eating behavior. Thus, there appear to be general social norms that affect eating behavior, but these processes are amenable to manipulation, pointing to potential intervention targets.

An interesting offshoot related to norms is research that investigates the weight of others in determining an individual's food intake. Some studies have found that non-dieters consume more calories when their food server is overweight compared with when she is normal weight (e.g., McFerran, Dahl, Fitzsimons, & Morales, 2010).

Social Support

Social support, characterized as instrumental, emotional, or informational, is related to eating behavior and diet quality (for a review, see Vesnaver & Keller, 2011). Social support may be particularly relevant in the context of healthy, as opposed to unhealthy, eating behavior. For example, the national representative National Health and Nutrition Examination Survey found that greater frequency of contact with family, friends, neighbors, coworkers, and others was related to eating at least five servings of FV per day (Ford, Ahluwalia, & Galuska, 2000). A systematic review of environmental determinants of FV intake (Kamphuis et al., 2006) identified social support as a contributor to more intake of FV. One rigorous study included in this review was a brief intervention in low-income participants, where FV consumption was confirmed via plasma/urine biomarkers (Steptoe, Perkins-Porras, Rink, Hilton, & Cappuccio, 2004). Baseline social support specifically for dietary change was related to 12-month increases in FV intake, independent of experimental group or other controls, suggesting the effects of social support are robust to a variety of settings and populations (Kamphuis et al., 2006).

Social support is a component of Bandura's social cognitive theory (SCT; Bandura, 1997), and in a study testing multiple components of SCT, social support was an important correlate of FV intake (Anderson, Winett, & Wojcik, 2007). In the context of an online SCT-based lifestyle intervention (Anderson-Bill, Winett, & Wojcik, 2011), perceived social support (from friends and family) was a predictor of better nutrition.

The relationship between social support and obesity is best tested in longitudinal studies, and such studies find mixed results. Moreover, positive and negative qualities of social relationships may affect BMI differently. In 4,724 Dutch adults, low positive social support was related concurrently to FV intake, and high negative social support was related concurrently to overweight

status, but neither was related to future weight change (Croezen et al., 2012). Negative relationships, however, were related to future risk of gains in BMI and waist circumference in another longitudinal study (Kouvonen et al., 2011). To inform this conflicted literature, Kershaw et al. (2014) examined a time frame when obesity tends to develop using 10-year longitudinal data in 3,074 participants. They found that those with continuously high levels of social support had lower likelihood of gaining (by more than 10%) in BMI or waist circumference than those with continuously low levels of social support. Those who increased in frequency of negative social contacts had higher likelihood of increasing waist circumference, but there was no relationship with BMI, and increasing frequency of positive social contacts showed no effect.

Social Networks

One of the most famous findings in the area of modern social network analysis is that obesity spreads across networks. Christakis and Fowler (2007) demonstrated this in the 12,067 participants of the longitudinal Framingham Heart Study (FHS; 1971–2003). Individuals' risk of becoming obese was higher if their spouses, siblings, and friends previously became obese, with stronger concordance among same-sex social ties. This phenomenon was not driven merely by similar BMI individuals forming relationships, nor was it driven by neighborhood/geographic distance effects.

Although much of the work examining social network effects on diet have been cross-sectional, Pachucki, Jacques, and Christakis (2011) used 10-year longitudinal data from the FHS to examine social influences (spouses, siblings, and friends) on dietary patterns. Using data classification procedures, they extracted dietary patterns based on food frequency questionnaires and found longitudinal social influence was exerted on those such as “alcohol and snacks” and “meat and soda” as well as those such as “healthier” and “caffeine avoidant.” This indicates that both healthy and unhealthy eating patterns are subject to social influence. A study in adolescents in Australia across three waves of data in 1 year found that friends appear to influence low-nutrient, energy dense food consumption (de la Haye, Robins, Mohr, & Wilson, 2013). This effect, however, was not mediated by beliefs about unhealthy food, indicating that social network influences may be operating implicitly.

Family Relationships and Parenting

Parents, caregivers, and families exert enormous influence on eating and diet in children. Food preferences of parents affect children's eating (Patrick, Nicklas, Hughes, & Morales, 2005), due to simple availability of those foods. Attachment style is also related to eating behavior.

In their review, Ventura and Birch (2008) distinguish between parenting styles (attitudes and interaction styles) and parenting practices (behavioral patterns). Moreover, there is likely a bidirectional relationship with children shaping parent's behavior (e.g., a child rejecting broccoli, causing the parent to avoid serving it again). Authoritative feeding styles are linked to better quality eating (Patrick et al., 2005), whereas authoritarian (“clean your plate!”), permissive, and neglectful parenting styles are associated with greater likelihood of having an overweight child (see Ventura & Birch, 2008). Using longitudinal design, steeper BMI trajectories and less leveling off of BMI were observed in authoritarian and disengaged parenting styles as opposed to balanced parenting (Fuemmeler et al., 2012).

Examples of parenting practices are using food as reward, restricting access to food, or modeling eating (Ventura & Birch, 2008; see Modeling section below). Using food as a reward was

shown in experimental studies to increase consumption of the target food but decrease liking. Restricting food can backfire, where children work harder to obtain restricted snacks and eat and prefer them more.

Families create a social context around mealtimes (for a review, see Patrick et al., 2005). Families eating together is related to better diet quality. When TV watching is part of mealtime, however, diet quality is poorer, likely due to exposure to advertising.

Given the importance of parenting for child weight and diet, interventions such as the “Lifestyle Triple P” have targeted parenting. In a randomized controlled trial, this intervention positively affected intermediate outcomes such as soda consumption, but saw no differences at 12 months between intervention and control in terms of weight or body composition (Gerards et al., 2015).

Romantic Relationships

Romantic relationships have effects on healthy and unhealthy eating. Women appear to play a larger role than men in terms of controlling eating behaviors of the couple (note: the literature has focused on heterosexual relationships; we know nearly nothing about eating behavior in same-sex romantic relationships). This is likely due to cultural norms surrounding the idea that women should be the ones who cook, and marriage appears to have differential effects on the eating of men versus women. For example, when wives report poorer marital quality, they also report more unhealthy dieting behaviors, particularly if they have low self-esteem (Markey, Markey, & Birch, 2001). In this same study, however, no marital quality variables were significantly related to healthy dieting behaviors. However, the literature on marital status and diet/nutritional quality is very mixed (reviewed in Vesnaver & Keller, 2011), indicating that it may not be marital status per se, but living alone that imparts risk for poor eating.

Marriage is associated with greater BMI, and spouses tend to have similar BMI (Jeffery & Rick, 2002). Longitudinal studies indicate that marriage is related to BMI gain, with effects most pronounced for African American women. Spouses’ BMIs also predict one another’s longitudinal BMI change (Jeffery & Rick, 2002). Divorce, in this study, was related to 2-year weight loss, although other studies have found divorce and other indices of marital dissatisfaction are related to increased BMI trajectories.

Modeling

Many food choices are derived from modeling—a behavioral mechanism related to social norms. Nisbett and Storms (1974) were among the first to test the effects of social influence on eating. Initially, they hypothesized that normal weight and overweight participants’ eating behavior would be differentially affected by social suppression or social facilitation cues of eating compared with a no-cue control condition. Participants completed a task with a confederate where there happened to be crackers on the table. In the suppression condition, the confederate ate one cracker; in the facilitation condition, the confederate ate 20 crackers, and there was no confederate in the control condition. Rather than a differential effect, they found that regardless of participant weight, participants typically mimicked the consumption behavior of the confederate.

This finding launched a line of research into the modeling effect and underlying mechanisms of modeling behavior in food consumption (reviewed in Herman et al., 2003). Researchers have tested if these effects were moderated by participant dieting status, participant satiation,

and personality characteristics such as extraversion and self-monitoring and found that the expected main effect of modeling occurs regardless of these moderators. Researchers have even found that participants model behavior when left alone but are given information on others' behavior (e.g., with a bogus "data sheet" left behind with information on how much the prior participant had ostensibly eaten; see also informational norms section above).

Conclusion

People often think of diet and obesity as matters of individual will—individuals exert control over their food choice, and the choices they make directly impact their weight. However, in this entry we covered a wide array of evidence that suggests this is not entirely the case. Culture forms our attitudes around food, socioeconomic status impacts our access to foods, norms guide behavior around food, and social connections with friends, family, and even strangers modify our behavior around food. In addition we showed that weight and obesity are also tied to social factors. Changes in BMI travel through social networks and are affected by social support and the status of our marriages.

In considering future directions, it is important to note that these factors we have discussed in isolation are deeply connected and build on one another to create our attitudes and subsequent eating behavior. Future directions should integrate these factors to build better interventions with potentially cascading impacts. For example, a future intervention aiming to change eating norms or attitudes toward food within a community should first examine the community's social network to identify particular individuals or groups who will have the strongest impact. On a larger scale, another future direction could aim to change eating norms in parents and also to explicitly test whether this also changes norms in children through modeling, with implications for the norms within both parent and child social networks. And, after some time, since culture is shaped by the people within it, these changes in norms in parents could even lead to lasting cultural changes in attitudes toward food and healthier diets.

Author Biographies

Gbolahan O. Olanubi, C. Phil, is a doctoral student in the Department of Psychology at the University of California, Los Angeles. His research investigates concealable identities and stereotype threat, and of particular relevance to this entry, he is interested in how race affects perceptions of the healthiness and quality of food and the downstream health implications of these perceptions.

A. Janet Tomiyama, PhD, is an associate professor in the Department of Psychology at the University of California, Los Angeles. Her research investigates stress, eating, dieting, and weight stigma.

References

- Anderson, E. S., Winett, R. A., & Wojcik, J. R. (2007). Self-regulation, self-efficacy, outcome expectations, and social support: Social cognitive theory and nutrition behavior. *Annals of Behavioral Medicine*, 34(3), 304–312. doi:10.1007/BF02874555
- Anderson, J. V., Bybee, D. I., Brown, R. M., McLean, D. F., Garcia, E. M., Breer, M. L., & Schillo, B. A. (2001). 5 a day fruit and vegetable intervention improves consumption in a low income

- population. *Journal of the American Dietetic Association*, 101(2), 195–202. doi:10.1016/S0002-8223(01)00052-9
- Anderson-Bill, E. S., Winett, R. A., & Wojcik, J. R. (2011). Social cognitive determinants of nutrition and physical activity among web-health users enrolling in an online intervention: The influence of social support, self-efficacy, outcome expectations, and self-regulation. *Journal of Medical Internet Research*, 13(1). doi:10.2196/jmir.1551
- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Macmillan.
- Christakis, N. A., & Fowler, J. H. (2007). The spread of obesity in a large social network over 32 years. *New England Journal of Medicine*, 357(4), 370–379. doi:10.1056/NEJMsa066082
- Croezen, S., Picavet, H. S. J., Haveman-Nies, A., Verschuren, W. M., de Groot, L. C., & van't Veer, P. (2012). Do positive or negative experiences of social support relate to current and future health? Results from the Doetinchem Cohort Study. *BMC Public Health*, 12(1), 65. doi:10.1186/1471-2458-12-65
- Darmon, N., Ferguson, E., & Briand, A. (2003). Do economic constraints encourage the selection of energy dense diets? *Appetite*, 41(3), 315–322. doi:10.1016/S0195-6663(03)00113-2
- de Castro, J. M. (2002). Age-related changes in the social, psychological, and temporal influences on food intake in free-living, healthy, adult humans. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 57(6), M368–M377. doi:10.1093/gerona/57.6.M368
- de la Haye, K., Robins, G., Mohr, P., & Wilson, C. (2013). Adolescents' intake of junk food: Processes and mechanisms driving consumption similarities among friends. *Journal of Research on Adolescence*, 23(3), 524–536. doi:10.1111/jora.12045
- Ferrières, J. (2004). The French paradox: lessons for other countries. *Heart*, 90(1), 107–111.
- Ford, E. S., Ahluwalia, I. B., & Galuska, D. A. (2000). Social relationships and cardiovascular disease risk factors: Findings from the third national health and nutrition examination survey. *Preventive Medicine*, 30(2), 83–92. doi:10.1006/pmed.1999.0606
- Fuemmeler, B. F., Yang, C., Costanzo, P., Hoyle, R. H., Siegler, I. C., Williams, R. B., & Østbye, T. (2012). Parenting styles and body mass index trajectories from adolescence to adulthood. *Health Psychology*, 31(4), 441. doi:10.1037/a0027927
- Gerards, S. M., Dagnelie, P. C., Gubbels, J. S., van Buuren, S., Hamers, F. J., Jansen, M. W., ... Kremers, S. P. (2015). The effectiveness of lifestyle triple P in the Netherlands: A randomized controlled trial. *PLOS One*, 10(4), 1–18. doi:10.1371/journal.pone.0122240
- Herman, C. P., Roth, D. A., & Polivy, J. (2003). Effects of the presence of others on food intake: A normative interpretation. *Psychological Bulletin*, 129(6), 873. doi:10.1037/0033-2909.129.6.873
- Howland, M., Hunger, J. M., & Mann, T. (2012). Friends don't let friends eat cookies: Effects of restrictive eating norms on consumption among friends. *Appetite*, 59(2), 505–509. doi:10.1016/j.appet.2012.06.020
- Jeffery, R. W., & Rick, A. M. (2002). Cross-sectional and longitudinal associations between Body Mass Index and marriage-related factors. *Obesity Research*, 10(8), 809–815. doi:10.1038/oby.2002.109
- Kamphuis, C., Giskes, K., de Bruijn, G. J., Wendel-Vos, W., Brug, J., & Van Lenthe, F. J. (2006). Environmental determinants of fruit and vegetable consumption among adults: A systematic review. *British Journal of Nutrition*, 96(04), 620–635. doi:10.1079/BJN20061896
- Kershaw, K. N., Hankinson, A. L., Liu, K., Reis, J. P., Lewis, C. E., Loria, C. M., & Carnethon, M. R. (2014). Social relationships and longitudinal changes in body mass index and waist circumference: The coronary artery risk development in young adults study. *American Journal of Epidemiology*, kwt311. doi:10.1093/aje/kwt311
- Kouvonen, A., Stafford, M., De Vogli, R., Shipley, M. J., Marmot, M. G., Cox, T., ... Kivimäki, M. (2011). Negative aspects of close relationships as a predictor of increased body mass index and waist circumference: The Whitehall II study. *American Journal of Public Health*, 101(8), 1474–1480. doi:10.2105/AJPH.2010.300115
- Markey, C. N., Markey, P. M., & Birch, L. L. (2001). Interpersonal predictors of dieting practices among married couples. *Journal of Family Psychology*, 15(3), 464. doi:10.1037//0893-3200.15.3.464

- McFerran, B., Dahl, D. W., Fitzsimons, G. J., & Morales, A. C. (2010). Might an overweight waitress make you eat more? How the body type of others is sufficient to alter our food consumption. *Journal of Consumer Psychology*, 20(2), 146–151. doi:10.1016/j.jcps.2010.03.006
- Morland, K., Wing, S., Roux, A. D., & Poole, C. (2002). Neighborhood characteristics associated with the location of food stores and food service places. *American Journal of Preventive Medicine*, 22(1), 23–29. doi:10.1016/S0749-3797(01)00403-2
- Nisbett, R. E., & Storms, M. D. (1974). Cognitive and social determinants of food intake. In H. London & R. E. Nisbett (Eds.), *Thought and feeling: Cognitive alternation of feeling states* (pp. 190–208). Chicago: Aldine.
- Pachucki, M. A., Jacques, P. F., & Christakis, N. A. (2011). Social network concordance in food choice among spouses, friends, and siblings. *American Journal of Public Health*, 101(11), 2170–2177. doi:10.2105/AJPH.2011.300282
- Patrick, H., Nicklas, T. A., Hughes, S. O., & Morales, M. (2005). The benefits of authoritative feeding style: Caregiver feeding styles and children's food consumption patterns. *Appetite*, 44(2), 243–249. doi:10.1016/j.appet.2002.07.001
- Robinson, E., Hardman, C. A., Halford, J. C., & Jones, A. (2015). Eating under observation: A systematic review and meta-analysis of the effect that heightened awareness of observation has on laboratory measured energy intake. *The American Journal of Clinical Nutrition*, 102(2), 324–337. doi:10.3945/ajcn.115.111195
- Robinson, E., Thomas, J., Aveyard, P., & Higgs, S. (2014). What everyone else is eating: A systematic review and meta-analysis of the effect of informational eating norms on eating behavior. *Journal of the Academy of Nutrition and Dietetics*, 114(3), 414–429. doi:10.1016/j.jand.2013.11.009
- Rozin, P. (1996). Towards a psychology of food and eating: From motivation to module to model to marker, morality, meaning, and metaphor. *Current Directions in Psychological Science*, 18–24. doi:10.1111/1467-8721.ep10772690
- Rozin, P., Fischler, C., Imada, S., Sarubin, A., & Wrzesniewski, A. (1999). Attitudes to food and the role of food in life in the USA, Japan, Flemish Belgium and France: Possible implications for the diet–health debate. *Appetite*, 33(2), 163–180. doi:10.1006/appe.1999.0244
- Rozin, P., & Vollmecke, T. A. (1986). Food likes and dislikes. *Annual Review of Nutrition*, 6(1), 433–456. doi:10.1146/annurev.nu.06.070186.002245
- Steptoe, A., Perkins-Porras, L., Rink, E., Hilton, S., & Cappuccio, F. P. (2004). Psychological and social predictors of changes in fruit and vegetable consumption over 12 months following behavioral and nutrition education counseling. *Health Psychology*, 23(6), 574. doi:10.1037/0278-6133.23.6.574
- Vartanian, L. R., Herman, C. P., & Polivy, J. (2007). Consumption stereotypes and impression management: How you are what you eat. *Appetite*, 48(3), 265–277. doi:10.1016/j.appet.2006.10.008
- Ventura, A. K., & Birch, L. L. (2008). Does parenting affect children's eating and weight status? *International Journal of Behavioral Nutrition and Physical Activity*, 5(1), 15. doi:10.1186/1479-5868-5-15
- Vesnaver, E., & Keller, H. H. (2011). Social influences and eating behavior in later life: A review. *Journal of Nutrition in Gerontology and Geriatrics*, 30(1), 2–23. doi:10.1080/01639366.2011.545038

Suggested Reading

Food and Culture

- Rozin, P. (2007). Food and eating. In S. Kitayama & D. Cohen (Eds.), *Handbook of cultural psychology* (pp. 391–416). New York: Guilford.

Norms/Modeling Eating Behaviors

Herman, C. P., Roth, D. A., & Polivy, J. (2003). Effects of the presence of others on food intake: A normative interpretation. *Psychological Bulletin*, 129(6), 873. doi:10.1037/0033-2909.129.6.873

Socioeconomic Status

Morland, K., Wing, S., Roux, A. D., & Poole, C. (2002). Neighborhood characteristics associated with the location of food stores and food service places. *American Journal of Preventive Medicine*, 22(1), 23–29. doi:10.1016/S0749-3797(01)00403-2

Parenting

Ventura, A. K., & Birch, L. L. (2008). Does parenting affect children's eating and weight status? *International Journal of Behavioral Nutrition and Physical Activity*, 5(1), 15. doi:10.1186/1479-5868-5-15

Social Relationships

Vesnaver, E., & Keller, H. H. (2011). Social influences and eating behavior in later life: A review. *Journal of Nutrition in Gerontology and Geriatrics*, 30(1), 2–23. doi:10.1080/01639366.2011.545038

Social Factors in Neuroendocrine Function

Theodore F. Robles and Ben W. Shulman

Psychology Department, University of California, Los Angeles, CA, USA

Social Neuroendocrinology

Social neuroendocrinology is an interdisciplinary field that examines bidirectional links between neuroendocrine function and social behavior. This entry reviews key theory and evidence, focusing on neuroendocrine systems that are key players in responses to stressful events, because such systems are biologically plausible mechanisms through which social relationships can “get under our skin” and impact physical health (Miller, Chen, & Cole, 2009). Specifically, we focus on two main neuroendocrine axes: the autonomic nervous system (ANS) (subdivided into the sympathetic and parasympathetic nervous system [SNS and PSNS], respectively) and the hypothalamic–pituitary–adrenal (HPA) axis.

The ANS is critical for mounting rapid bodily responses to help individuals adapt to environmental challenges. For example, the SNS can speed heart rate and increase sweating through norepinephrine and epinephrine signaling, and the PSNS can promote bodily growth and restoration at rest through acetylcholine signaling from parasympathetic nerves to their target organs. The HPA axis is a multi-hormone cascade that culminates in the adrenal cortex releasing cortisol, which is capable of signaling to virtually all cells in the body. Because of their critical roles in mobilizing stored energy, and the multiple effects their signaling hormones have throughout the body, both axes have received the most attention in research linking social factors to health.

We note however that other neuroendocrine axes and hormones are also relevant for social neuroendocrinology, in part by interacting with the ANS and the HPA axis. These include estrogen and the hypothalamic–pituitary–gonadal axis (where prolactin and testosterone are the major signaling hormones), posterior pituitary hormones (including oxytocin and vasopressin), and hormones involved in regulating growth and energy metabolism (including growth hormone, insulin, leptin, and glucagon). We direct interested readers to several reviews and empirical papers (Carter, 2014; Robles & Carroll, 2011).

Social influences on neuroendocrine function begin in our earliest days of life (Repetti, Robles, & Reynolds, 2011). However, this entry primarily focuses on adulthood, which has received the most empirical attention. In addition, we restrict our discussion to hormone *signals* that are released by nerves and glands to the rest of the body, as opposed to the *effects* of those signals on target tissues and cells. For example, we discuss social factors and SNS responses, such as norepinephrine production, but do not discuss the effect of these responses on targets like the heart (in terms of increasing heart rate or blood pressure) or immune cells. That said, this entry will discuss the association between social factors and cardiovascular measures that are considered “pure” indices of sympathetic or parasympathetic activity. For instance, SNS activity is indexed by pre-ejection period (a time-based indicator of how quickly the heart contracts, assessed by impedance cardiography), and PSNS activity is indexed by high-frequency heart rate variability (assessed using electrocardiography and referred to in this entry simply as HRV).

This entry is divided into four sections. First, we describe key conceptualizations and measures and examine links between stress-related neuroendocrine function and structural aspects of social networks, such as the number or frequency of social contacts. We then review links with functional aspects of social networks, both positive (social support) and negative (social strain and conflict). Finally, we conclude by reviewing the neural and psychological mechanisms that link social relationships to stress-related neuroendocrine activity.

Key Concepts and Research Approaches

A key distinction must be made between two different aspects of neuroendocrine function: basal activity and physiological responses to environmental challenges (Robles & Carroll, 2011). Basal activity refers to the steady-state activity of biological systems, when the organism is not facing environmental challenges, in contrast to physiological responses to such challenges. Each aspect is measured differently. For example, a researcher could examine basal activity by measuring hormone levels at known low points, such as bedtime or at “rest” prior to a laboratory challenge, or measuring hormone levels at consistent times during the day over several days. To measure reactivity to environmental challenges, researchers frequently examine changing hormone levels in response to a laboratory stressor.

Models linking social functioning to physical health also distinguish between two types of associations, which stem from broader models linking social functioning to physical health (Cohen, Underwood, & Gottlieb, 2000). Specifically, social functioning may be associated with neuroendocrine function regardless of whether the individual is facing an environmental challenge, which would be consistent with a “main-effect” model. Alternatively, social functioning may be related only to neuroendocrine responses to an environmental challenge or stressor, which would be consistent with a “buffering” (protective) or “reactivity” (exacerbating) model.

Finally, the association between social functioning and neuroendocrine activity can be interrogated in a number of research designs. Correlational methods examine concurrent or prospective associations between neuroendocrine measures and social functioning (self- or other-reported), such as perceived social support or loneliness. Nonexperimental laboratory methods involve assessing social functioning in the laboratory, such as through observational coding of a social interaction paradigm, and correlating measures obtained during the interaction (self-reported, objectively observed) with neuroendocrine measures. Experimental methods involve manipulating social functioning, such as having an individual go through a

laboratory stressor either with or without the presence of a friend, and testing whether the manipulation changes neuroendocrine activity.

Structural Aspects of Social Networks

Structural features of social networks, also described as social integration, refer to the number of social relationships in which a person is involved and the degree of connection between the people they know (Taylor, 2011). For instance, some individuals have more interconnected social networks (in which most people know each other), while others have less interconnected social networks. Most social neuroendocrinology research has focused on number and frequency of social contacts, with few studies of social interconnections.

ANS functioning is linked to social integration in prairie voles (a social mammal). When subjected to social isolation, these animals show reduced HRV, which may be due to both increased SNS and reduced PSNS influence on the heart. In humans, the evidence suggests a similar picture. In several large studies, high social integration was related to greater resting HRV and lower basal levels of SNS hormones. In particular, weekly religious attendance was specifically related to less likelihood of having high urinary epinephrine, and being married was related to lower urinary epinephrine. Finally, in a small prospective study, international students were followed from arrival in their host country to 2 and 5 months later. Individuals with low social integration, particularly low social contact with people from other cultures, showed lower HRV over time, an association that grew stronger over the course of the study. Notably, baseline HRV did not predict changes in social integration over time, suggesting the causal direction may be from social structure to parasympathetic function, rather than the reverse.

In terms of HPA axis functioning, living alone and reporting less social contact were related to higher cortisol levels at bedtime, when cortisol is expected to be at its lowest levels, and greater cortisol production throughout the day. These findings were observed in middle-aged and older adults, and work in younger samples shows a similar pattern. In a social network analysis of nursing students, those who were less likely to nominate their classmates as friends had higher daily cortisol levels.

The overall pattern of findings across the ANS and HPA axis suggests that lower social connection is related to elevated basal SNS and HPA activity, as well as lower basal PSNS activity. This pattern indicates that neuroendocrine systems designed to respond during stress are upregulated, even when the individual is not facing a challenge. Considerably less is known about links between social structure and neuroendocrine responses during stress, but the literature reviewed suggests plausible neuroendocrine pathways through which low social integration may negatively impact health over time (Holt-Lunstad, Smith, & Layton, 2010).

Positive Aspects of Social Relationships: Social Support

A central benefit of social relationships is the experience of being cared for by others and being valued in a network of mutual dependence and assistance (Taylor, 2011). Social support can be informational (advice), instrumental (tangible assistance), or emotional (validation, comforting). Moreover, the way support is measured varies, and this entry focuses on two broad distinctions: perceived available support (often described as perceived support), which is the expectation of being able to call on others for support when needed, and received support,

which refers to individuals' self-report of being the beneficiary of helpful behavior. Perceived and received support are only moderately correlated, and greater perceived support is more consistently related to psychological and physical well-being compared with greater received support (Uchino, 2009).

Perceived Support

Research on perceptions of support availability is mostly correlational and focused on basal functioning. Perceived support is generally associated with lower SNS and higher PSNS activation. For instance, among people living with chronic stress, those with higher perceived support have lower catecholamine levels. For HPA axis function, the results are mixed. Some studies have found no association between 24-hr cortisol output and perceived support, both at work and at home (although greater perceived work support was related to higher cortisol output on weekends). Other studies have found that perceived support at work is linked to lower daily cortisol.

Received Support

While observational studies assess received support by self-report, experimental studies manipulate the social conditions of individuals facing laboratory stressors. In such experiments, received support consistently reduces blood pressure and heart rate responses to stress (Thorsteinsson & James, 1999). However, these outcomes are not pure indices of SNS or PSNS function. Received support is related to increased activity in pure markers of PSNS function. For instance, received support was related to elevated HRV when facing a laboratory stress task (particularly for individuals high in dispositional compassion). Similarly, received support is related to decreased activity in pure markers of SNS function. For example, receiving support from a member of one's self-identified social group was related to lower galvanic skin response. However, the benefits of received support may be offset by the added evaluative threat of another person's presence. Even in the presence of a supportive companion, people performing a speech task in an evaluative context had increased SNS activity compared with performing the speech alone, as indicated by larger decreases in cardiovascular pre-ejection period.

One of the earliest and most cited examples of received support and HPA reactivity to stress involved participants either receiving support from an opposite-sex confederate, receiving support from an opposite-sex romantic partner, or being alone (no support) prior to going through a speech and arithmetic task. Interestingly, men who received support from their female partners showed a smaller cortisol response during the stressor compared with when alone or receiving support from a stranger. In contrast, women receiving support from their male romantic partners did not show smaller cortisol responses compared with the other conditions. In other studies, men, but not women, receiving verbal support from their best friends showed reduced cortisol responses to a laboratory stress task. Women, in contrast, showed an increase in cortisol responses when receiving verbal support from their male partners but showed buffered responses after a partner's massage. Finally, men receiving support from a stranger, with whom they had a brief closeness manipulation, showed higher cortisol responses, but women showed no difference. Similar to the literature on autonomic responses, evaluative threat influences HPA axis responses and may explain the mixed pattern of results. Indeed, one study found that performing speech and mental arithmetic tasks in front of a supportive audience showed higher cortisol responses than those performing the task alone. Moreover, the

benefits of social support in attenuating cortisol responses to stress may depend on oxytocin. A specific polymorphism (rs53576) in the oxytocin receptor gene moderates the benefits of social support on cortisol response to stress, and oxytocin administration can improve the stress-buffering cortisol reduction of social support.

Summary

Research on perceived support and stress-related neuroendocrine function is largely correlational, with a focus on basal activity. In contrast, research on received support is largely experimental, focusing on reactivity to stress. Interestingly, while findings linking perceived support and health are more consistent than those linking received support and health (Uchino, 2009), the opposite seems to be the case for stress-related neuroendocrine activity. Received support is robustly related to ANS and HPA responses to stress. However, factors like gender of the support provider, the degree of evaluation potential, and other neuroendocrine systems (i.e., oxytocin) can modify those links. A smaller literature suggests that greater perceived support is related to lower basal SNS and higher PSNS activity, while research on basal HPA axis activity is mixed.

Negative Aspects of Social Relationships: Social Negativity and Loneliness

Social Negativity

Relationships can be sources of negativity when people face aversive or unwanted behavior from another person (Brooks & Dunkel Schetter, 2011). Such negativity can involve provoking conflict, being insensitive to an individual's needs, and disrupting an individual's ability to pursue goals. Most research to date has focused on conflict, and even research examining insensitivity to an individual's needs has done so in the context of conflict.

Most research on social conflict has focused on cardiovascular reactivity in close, romantic relationships. However, a few studies have measured pure indicators of autonomic activity (Robles, Slatcher, Trombello, & McGinn, 2014). In newlywed couples, greater hostile and negative behavior during problem discussions was related to elevated epinephrine and norepinephrine responses during the discussion. Older couples showed similar effects, with negative behavior related to increased epinephrine levels. In terms of PSNS activity, among young married couples, marital conflict was associated with lower HRV, but only for women.

Conflict in many different kinds of relationships has been linked to HPA activity (reviewed in Robles et al., 2014). People reporting higher levels of negativity from friends, family, and romantic partners had lower morning cortisol levels and flatter cortisol slopes across the day. In newlywed couples, greater hostile behavior while discussing problems in the relationship was related to elevated adrenocorticotrophic hormone levels (ACTH, which stimulates cortisol release by the adrenal cortex) during and after the discussion. Similarly, for these newlyweds, the combination of low supportive behavior and high hostile behavior was related to elevated cortisol levels. Older couples showed similar effects, but only among women, whose levels of cortisol and ACTH increased following more conflictual discussions. Moreover, conflicts in which one spouse withdraws from the other's demands (e.g., avoids the topic, a notable example of social insensitivity) are viewed as particularly destructive for relationships. Accordingly,

in newlywed couples, greater probability of husbands withdrawing from their wives' demands was related to elevated wives' cortisol over a 24-hr period.

Loneliness

Loneliness is distinct from low social integration. People may live contently in solitude or have active social lives and still feel lonely (Hawkley & Cacioppo, 2010). Few studies have examined links between loneliness and pure indicators of autonomic function, although thus far research suggests that loneliness is not related to SNS or PSNS influences on the heart. However, loneliness was associated with elevated epinephrine in overnight urine samples in middle-aged and older adults. Thus, the vasculature (arteries), but not the heart, may be targets for loneliness-related elevations in SNS activity. This pattern could explain the consistent elevations in blood pressure related to loneliness. Regarding HPA functioning, loneliness is related to elevated basal cortisol levels in a number of studies. No studies have examined HPA reactivity to stress, but several studies have found that greater loneliness is related to larger cortisol responses to awakening.

Neural and Psychological Mechanisms

We conclude this entry by discussing the mechanisms that may explain how social functioning can impact stress-related neuroendocrine function and linking those mechanisms to models of how social relationships influence health. To directly influence neuroendocrine functioning, the neural circuits involved in social information processing must interface with circuits that regulate the HPA axis and the ANS. Over the past decade, neuroimaging research in humans has identified the circuitry involved in regulating neuroendocrine function, which had been previously outlined in animal models. Specifically, a “medial prefrontal–brain stem axis” involves evolutionarily newer brain structures processing information about the social environment (e.g., prefrontal cortex, anterior cingulate; Eisenberger, 2013) and then sending signals to evolutionarily older structures that modulate HPA and ANS activity (e.g., amygdala, hypothalamus). More recent work is now identifying links between social relationships and neural structure and function.

In terms of neural structure, most work has focused on social integration (Platt, Seyfarth, & Cheney, 2016). Early studies found that greater social integration was specifically associated with greater amygdala volume. This pattern parallels work in macaques, where living in larger social groups (2–7 per group) vs. alone was related to increased gray matter in the amygdala. Living in larger social groups was also related to more gray matter in regions that track attention and process social information and face and body movement. Similarly, in humans, larger social networks were related to greater orbitofrontal cortex volume, a region specifically implicated in social cognition and affective processing, but were not related to dorsal prefrontal cortex volume, a region involved in planning and executive function. In terms of neural connectivity, greater social integration was related to stronger connections between the amygdala and brain regions involved in social perception and affiliation. In addition, greater social integration was related to stronger interconnections (greater white matter integrity) in a frontal region involved in interconnecting other regions involved in social information processing and reward processing (Molesworth, Sheu, Cohen, Gianaros, & Verstynen, 2015).

Studies linking the quality of social interactions to neural mechanisms implicate the same medial prefrontal axis structures. For example, Eisenberger, Taylor, Gable, Hilmert, and Lieberman (2007) had participants complete a brief laboratory stressor, 10 days of repeated

measures on the degree of supportiveness in their daily social interactions, and finally a neuro-imaging task assessing neural responses to social rejection. Greater daily reports of feeling supported during social interactions were related to lower neural activity in several regions of the medial prefrontal axis during social rejection. Moreover, activity in several medial prefrontal axis regions explained associations between daily social support and cortisol reactivity during the laboratory stressor.

Main effects models suggest that a network of high-quality relationships provides more opportunities for social influence from others, support provision, and benefits for mood that then influence neuroendocrine responses (Cohen et al., 2000). However, some researchers have argued that social relationships are so intrinsic to human experience that the presence of other people should be regarded as a default or baseline state (Coan & Sbarra, 2015). Because people adapted to living in close proximity and connection to other people, the brain expects to assess social resources and functions best under such conditions. Deviations from this social baseline (perceiving fewer social resources) are perceived as threats to well-being, and the individual must be prepared to respond. In the context of social neuroendocrinology, mounting ANS and HPA responses, even at rest, prepares the organism to deal with what it perceives as a threatening environment (mainly by increasing energy available for use by the brain and body).

Stress-buffering models suggest that perceiving greater availability of social resources, or actual receipt of social resources, should affect neuroendocrine mechanisms through mitigating negative cognitive (e.g., reducing threat appraisals) and emotional responses (e.g., reducing negative mood) (Cohen et al., 2000). However, to date, the literature is mixed as to whether positive aspects of social functioning are linked to neuroendocrine function via psychological mechanisms (Uchino, Bowen, Carlisle, & Birmingham, 2012). Fortunately, a number of potential ways forward have been suggested, including improving research designs, rethinking how social relationships are conceptualized and measured, and including a focus on automatic social cognitive processes (Uchino et al., 2012). Increased attention to these issues will hopefully yield advances in research on social neuroendocrinology.

Author Biographies

Theodore F. Robles is an associate professor in the Department of Psychology at the University of California, Los Angeles. His research examines the biological mechanisms that explain how close relationships impact health. His empirical work includes both allostatic and restorative processes, such as HPA responses, inflammation, and skin barrier recovery, and covers the life course, including childhood, young adulthood, parenting age, and older ages.

Ben W. Shulman is a graduate student in health psychology at the University of California, Los Angeles, where he studies how challenging self-regulation affects conflict in romantic relationships. He was a research assistant in the Culture Cognition and Coevolution Lab at the University of British Columbia and at the Center for Health Coping Studies. There, he studied the development of human culture and the ways spouses support each other through the stress of chronic disease.

References

- Brooks, K. P., & Dunkel Schetter, C. (2011). Social negativity and health: Conceptual and measurement issues. *Social and Personality Psychology Compass*, 5, 904–918. doi:10.1111/j.1751-9004.2011.00395.x

- Carter, C. S. (2014). Oxytocin pathways and the evolution of human behavior. *Annual Review of Psychology*, 65, 17–39. doi:10.1146/annurev-psych-010213-115110
- Coan, J. A., & Sbarra, D. A. (2015). Social baseline theory: The social regulation of risk and effort. *Current Opinion in Psychology*, 1, 87–91. doi:10.1016/j.copsyc.2014.12.021
- Cohen, S., Underwood, L., & Gottlieb, B. H. (2000). Social relationships and health. In S. Cohen, L. Underwood, & B. H. Gottlieb (Eds.), *Social support measurement and intervention* (pp. 3–25). New York, NY: Oxford University Press.
- Eisenberger, N. I. (2013). An empirical review of the neural underpinnings of receiving and giving social support: Implications for health. *Psychosomatic Medicine*, 75(6), 545–556. doi:10.1097/PSY.0b013e31829de2e7
- Eisenberger, N. I., Taylor, S. E., Gable, S. L., Hilmert, C. J., & Lieberman, M. D. (2007). Neural pathways link social support to attenuated neuroendocrine stress responses. *Neuroimage*, 35, 1601–1612.
- Hawkey, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine*, 40(2), 218–227. doi:10.1007/s12160-010-9210-8
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7(7), e1000316. doi:10.1371/journal.pmed.1000316
- Miller, G. E., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, 60, 501–524. doi:10.1146/annurev.psych.60.110707.163551
- Molesworth, T., Sheu, L. K., Cohen, S., Gianaros, P. J., & Verstynen, T. D. (2015). Social network diversity and white matter microstructural integrity in humans. *Social Cognitive and Affective Neuroscience*, 10(9), 1169–1176. doi:10.1093/scan/nsv001
- Platt, M. L., Seyfarth, R. M., & Cheney, D. L. (2016). Adaptations for social cognition in the primate brain. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 371, 20150096. <http://dx.doi.org/10.1098/rstb.2015.0096>
- Repetti, R. L., Robles, T. F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology*, 23, 921–938. doi:10.1017/S095457941100040X
- Robles, T. F., & Carroll, J. E. (2011). Restorative processes and health. *Social and Personality Psychology Compass*, 5, 518–537. doi:10.1111/j.1751-9004.2011.00368.x
- Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological Bulletin*, 140, 140–187. doi:10.1037/a0031859
- Taylor, S. E. (2011). Social support: A review. In H. S. Friedman (Ed.), *Oxford handbook of health psychology* (pp. 189–214). New York, NY: Oxford University Press.
- Thorsteinsson, E. B., & James, J. E. (1999). A meta-analysis of the effects of experimental manipulations of social support during laboratory stress. *Psychology & Health*, 14(5), 869–886. doi:10.1080/08870449908407353
- Uchino, B. N. (2009). Understanding the links between social support and physical health: A life-span perspective with emphasis on the separability of perceived and received support. *Perspectives on Psychological Science*, 4, 236–255. doi:10.1111/j.1745-6924.2009.01122.x
- Uchino, B. N., Bowen, K., Carlisle, M., & Birmingham, W. (2012). Psychological pathways linking social support to health outcomes: A visit with the “ghosts” of research past, present, and future. *Social Science & Medicine*, 74(7), 949–957. doi:10.1016/j.socscimed.2011.11.023

Suggested Reading

- Miller, G. E., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, 60, 501–524. doi:10.1146/annurev.psych.60.110707.163551

- Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological Bulletin*, 140, 140–187. doi:10.1037/a0031859
- Taylor, S. E. (2011). Social support: A review. In H. S. Friedman (Ed.), *Oxford handbook of health psychology* (pp. 189–214). New York, NY: Oxford University Press.

Social Factors in Sleep

Zlatan Krizan and Garrett Hisler

Department of Psychology, Iowa State University, Ames, IA, USA

Humans spend about a third of their life sleeping, typically once every night for 8 hr. Sleep is behaviorally defined as a reversible state of perceptual disengagement from—and unresponsiveness to—the environment. In an adult, sleep proceeds in alternating cycles of slow-wave sleep (non-rapid eye movement, NREM) and more active rapid eye movement (REM) sleep associated with emotional dreams (Carskadon & Dement, 2011). Modern neuropsychology reveals that circadian (daily) rhythms in wakefulness and sleep are controlled by an internal biological clock located in the suprachiasmatic nuclei of the hypothalamus. This mechanism controls most physiological activity, including daily (i.e., circadian) rhythms in body temperature, hormone levels, psychomotor vigilance, and affect (Van Dongen & Dinges, 2003). Although these rhythms are internally generated, they are synchronized with external time cues, the most important of which is the light-dark cycle. Critically, sleep plays a vital role in health given its many key functions such as toxin disposal from the brain, physical rest and physiological regeneration, and encoding and integration of newly learned information and emotional experiences (Walker, 2010).

Any significant deviation in sleep quantity or timing relative to the optimal sleep needed by a given individual (typically between 7 and 9 hr of uninterrupted sleep during the night) is deemed sleep disruption. Critically, various forms of sleep disruption (i.e., total deprivation, partial restriction, or fragmentation) can produce similarly deleterious neurobehavioral effects. These include reduced attention, lowered energy and enthusiasm, compromised executive functioning, and poor self-control (Reynolds & Banks, 2010; Van Dongen & Dinges, 2003). In this vein, sleep problems often present themselves alongside anxiety, mood, impulse control, and substance abuse disorders. Conversely, improving sleep problems can alleviate emotional problems such as depression. Sleep problems are also a major risk factor for physical conditions such as obesity, diabetes, and cardiovascular diseases. Sleep problems are even associated with increased all-cause mortality (Hublin, Partinen, Koskenvuo, & Kaprio, 2007). Although the exact causal mechanisms between sleep and health problems are yet to be elucidated, sleep plays an essential role in maintaining good health, earning its status as the third “pillar of health” alongside diet and exercise.

Social Behavior and the Sleep–Wake Cycle Are Mutually Dependent

Although sleep is a biological phenomenon, it is intrinsically connected to social behavior. The organization of human social and cultural activities has always been constrained by the rhythm of the biologically mandated sleep–wake cycle. Humans need to be awake and alert in order to meaningfully interact with others, so most social interaction is confined to daylight hours (although the need to protect the community from threats also meant some individuals needed to be awake at night). Throughout the day, social behavior such as work and recreation tends to be further concentrated around times of circadian peaks in alertness (i.e., late morning and early evening). Indeed, both the drive to engage socially and the ability to successfully interact with others reflect the endogenous circadian rhythm (Hasler, Mehl, Bootzin, & Vazire, 2008). Social behavior is thus heavily dependent on daily rhythms in biological capacities for psychological and motor engagement. However, any individual's level of alertness or sleep timing is also highly flexible, showing sensitivity to a variety of internal (e.g., motivation, drug effects) and external modifiers (e.g., threats, social incentives). As illustrated in the next section, many of the most important modifiers of human sleep behavior are social in nature.

The Impact of Social Behavior on Sleep

Social Behavior as a Time Cue

The general propensity toward social behavior is constrained by the daily rhythm of alertness, yet this rhythm can also be entrained to the regular timing of social interactions. Although light exposure plays the most important role (Czeisler et al., 1999), socially influenced behaviors such as collectively determined sleep–wake schedules, exercise, or eating food can shift the internal clock (Mistlberger & Skene, 2004). Moreover, regularity of social activities may be an important support to a regular sleep–wake cycle and optimal sleep quality (Monk, Petrie, Hayes, & Kupfer, 1994). The social zeitgeber theory goes so far as to suggest that stressful life events lead to depression because they disrupt social rhythms needed for a stable cycle in mood and motivation (Ehlers, Frank, & Kupfer, 1988). Fatigue, negative affect, and impaired processing resulting from sleep disruption may then further perpetuate depressive symptoms. Given the extensive confounding of social activity with light exposure and food intake (e.g., most interactions occur under light, and most eating involves socializing), social factors exert considerable impact on our biological rhythms by affecting when we engage in arousing activities (e.g., going for a run when it fits others' schedules), go outside into natural light (e.g., leaving the house to go to work), or eat (e.g., having dinner when the family is available). In short, socializing modulates human biological rhythms through other variables, even if not in itself an essential time cue.

Social Interactions as Disruptors of the Sleep–Wake Cycle

Although not the chief entrainment signal for our biological rhythms, social factors play a major role in how and when individuals sleep. First, social experiences are often the source of stressors that create insomnia (problems with initiating or maintaining sleep) by undermining onset, subjective quality, or consolidation of sleep. The most stressful events in life are social

in nature; losing one's parent or spouse, being fired, being romantically rejected, and failing to be selected for a promotion are all essentially social experiences that draw their power from impact on social identity and connections (Almeida, 2005). Such stressful experiences carry with them physiological arousal, anxiety, and ruminative thinking, which disrupt sleep as individuals aim to cope with the stressors.

Critically, these markers of stress play a key role in the formation and development of insomnia, with negative social events often being a precipitating or perpetuating factor (e.g., work and home conflict; Riemann et al., 2010). For example, embroilment in aggression and interpersonal conflict can fuel worry and rumination about one's safety or guilt about one's own behavior and its consequences on others; all these can undermine sleep over time, especially among romantic partners (Krizan & Herlache, 2016). Conversely, having trust and intimate relationships with others who can provide social support during a time of crisis may reduce the impact of stressful events on one's sleep. For people who live and sleep with romantic partners, there seems to be an especially close tie between social interactions and sleep—one study found that when women reported less negatively toned social interactions with their male partner, *both* individuals slept more efficiently the next night (Hasler & Troxel, 2010). As a result, social experiences not only play a major role in development and perpetuation of sleep problems such as insomnia but also can provide a buffer against them in the form of social support (Maume, 2013). Such processes also contribute to why sociocultural factors such as personal socioeconomic status or neighborhood quality negatively impact sleep and health among disadvantaged populations.

Second, conformity to social norms (going out late) and pursuit of personal social goals (getting up early to help an ill partner) exert a large impact on behavioral choices that impact sleep. Correspondingly, perceived norms have been found to predict intentions toward behaviors that promote optimal sleep, referred to as *sleep hygiene*. Standard recommendations to aid proper sleep include avoiding psychoactive substances that interfere with sleep (e.g., caffeine, alcohol), exercising regularly (although not before bed), maintaining a regular sleep-wake schedule, reducing stimulation in the sleeping environment (e.g., bedroom noise) that is only used for sleeping, and avoiding excessive daytime napping. All of these factors have been linked to optimal sleep in some way, although their potential as public health interventions is still being evaluated (Irish, Kline, Gunn, Buysse, & Hall, 2015).

Despite the fact that sleep hygiene recommendations are made to individuals, whether any given individual actually follows good sleep practices often comes down to the social influence of others, be it because of concerns about what others will think or because of others' pleas and persuasive attempts. For example, the pressure to be constantly available on social media and respond quickly has pushed teenagers to stay up late, use electronic devices in bed, and resultantly displace or harm their sleep (Woods & Scott, 2016). Even more disconcerting is the rise of "sleep texting," wherein individuals wake up to respond to text messages with little or no memory of the interaction the next morning. Unfortunately, sleep practices can spread through social networks such that one's friend adopt similarly bad sleep habits.

In a similar vein, the use of psychoactive drugs that interfere with sleep typically occurs within social milieus. The impact of social ties on sleep-relevant behavior is especially strong in adolescence given increased concerns about acceptance (Maume, 2013). In fact, teenagers are more likely to use substances such as tobacco and alcohol (both disruptive to sleep) as a result of both active influence by others (e.g., being offered a cigarette) and more passive modeling of others' behavior or alignment with social norms (e.g., drinking because others are drinking). In short, understanding individuals' social ties is critical for understanding why people engage in poor sleep hygiene such as substance use and bedtime delays.

Third and finally, whether people lie down in bed regularly and keep the bedroom free of distractions strongly depends on people with whom they live, be they family members, roommates, or romantic partners. Romantic partners are likely to play the most significant role given the sharing of both evening activities and the bedroom; for example, couples are more concordant in their behaviorally measured sleep than are random pairs of individuals (Gunn, Buysse, Hasler, Begley, & Troxel, 2014). Moreover, bed times and rise times are typically a function of preferences and responsibilities of both partners, requiring frequent compromise. How people balance work and family demands is constantly shaped by interactions with one's partner, so understanding the nature of intimate relationships is critical for understanding sleep.

The Impact of Sleep on Social Behavior

Social processes undoubtedly play an important role in shaping human sleep, and this role represents one pathway by which social behavior affects health. At the same time, sleep itself molds social and health behavior. Although scientific evidence on this score is only emerging, accumulating evidence converges on the conclusion that when and how humans sleep carries considerable consequences for what people do, how they think and feel about others, and how they interact with them.

How Optimal Sleep Timing (Chronotype) Shapes Social Experience

A typical person usually sleeps during the night, but individuals' ideal times for going to sleep and waking up vary considerably. A key reason for this variability stems from individual differences in timing of biological rhythms, namely, circadian phase or *chronotype*. Although behavior of every individual follows his or her internal biological clock, this clock is often slightly ahead or slightly behind the earth's 24-hr light-dark cycle. As a result, some individuals' internal cycle starts and ends earlier than the 24-hr day; these "larks" are said to have a morning chronotype as they rise earlier, feel more energetic in the morning, and have difficulties extending wakefulness late into the night. Conversely, others' internal cycle starts and ends later than the 24-hr day; these "owls" have an evening chronotype as they rise later, feel most energetic in the evening, and find it easy to stay up late (Roenneberg, Wirz-Justice, & Mellow, 2003). Most individuals ("hummingbirds") fall somewhere in between these extremes along a continuum. As we outline next, individuals' chronotype exerts a powerful impact on the social environments people inhabit, their psychophysiological state during social interactions at a particular time of day, and weekly patterns of rising and falling asleep that impact socializing.

First, because people's chronotypes bias sleep propensity toward the most optimal time for the individuals' biological clock, they partially determine when people are awake and participating in social activities. Critically, the nature of social environments accessible to a person throughout the day varies, as early morning affords chatting over a cup of coffee (but not grabbing a beer), while later evening or night affords socializing in entertainment establishments that serve alcohol or provide adult entertainment such as bars and clubs. Because chronotypes partially determine the time of day people have the energy and interest to participate in (social) activities, they also constrain available social opportunities and resultantly the likelihood of participating in them. As a result, evening-oriented individuals should be more likely to come in contact with vices such as drugs, alcohol, or sex (more available at night) than

are more morning-oriented individuals. Consistent with this premise, evening-oriented individuals are more likely to smoke, drink, and behave promiscuously (Wittmann, Dinich, Merrow, & Roenneberg, 2006). This pattern may partially result from individuals' self-selection into these nighttime environments, but persistent exposure to them may promote the development of substance use problems, impulsive behavior, and social conflicts associated with nighttime environments and intoxication.

Second, chronotype also strongly impacts individuals' psychophysiological state during a given time of day. This resultantly affects both the momentary ability and motivation of an individual to cope with situational and social demands occurring at a particular time of day. Specifically, "larks" are more likely to feel alert and interested, be vigilant, perform better physically, and think carefully earlier rather than later in the day, whereas the opposite is true for "owls." These interactive patterns between the time of day and chronotype have significant consequences for numerous social variables such as engagement with others, group performance, perspective taking, and impression formation. For example, morning-oriented individuals maintain exercise more effectively when such activity occurs during morning hours than evening hours (Hisler, Phillips, & Krizan, 2017).

Third and finally, because the "social" clock (i.e., timing of socially formalized activities such as work) is more aligned with the "biological" clock of morning-oriented – rather than evening-oriented – individuals, the latter experience "social jet lag," that is, a misalignment of biological and social time. As normal work schedules are best suited for morning chronotypes, evening individuals who fall asleep later accrue a sleep debt during the week, for which they compensate by sleeping in on the weekends (Wittmann et al., 2006). This dilemma also leads to greater daytime tiredness, more psychosomatic disturbances, and more emotion dysregulation for evening-oriented individuals during a typical weekday. Given that many social behaviors (e.g., drinking, smoking, exercising) have important implications for health, these findings point to the essential role that chronotypes play in social behavior and especially to the importance of match between people's biological clocks and the social opportunities and demands that fluctuate with time of day.

The Effects of Sleep Disruption on Social Behavior

Losing sleep has far-reaching consequences on people's health and well-being, yet chronic sleep restriction seems to be prevalent in the US population, with 30% of American adults reporting less than 6 hr of sleep a night (the National Sleep Foundation recommends 7–9 hr of sleep a night). Furthermore, this population has increased over the past two decades. Because sleep impacts virtually all aspects of cognitive and emotional functioning, existing evidence suggests that it creates downstream consequences for how we perceive and interact with the social world.

First, sleep disruption leads to feelings of fatigue, loss of energy, and lack of motivation. Specifically, sleep loss leads students to prefer easier tasks, to spend less time on activities such as dressing neatly and fashionably, and to engage in more time wasting during work (Engle-Friedman et al., 2003). These choices seem logical because they dovetail with subjective perceptions of increased effort following lost sleep, evident in higher perceptions of difficulty for athletic challenges, physical tasks, and intellectual problems (Engle-Friedman et al., 2003). These consequences carry direct implications for people's motivation and ability to pursue numerous social goals. Dressing less neatly and fashionably lowers perceptions of status and competence, while loafing or avoidance of taxing tasks such as exercise creates opportunities

for sedentary social behaviors such as texting or use of social media. In short, the reduced effort and amotivation following sleep loss impacts how individuals allocate their time, which can produce a wide variety of social and health consequences.

Second, in concert with feelings of fatigue and lack of drive, many of the cognitive and emotional effects of sleep loss broadly impair self-regulation, namely, the ability to select, enact, and monitor progress toward important goals (Krizan & Hisler, 2016). Sleep loss not only saps energy and motivation needed to enact goal-directed behavior but also impairs vigilance and executive functions (i.e., selective attention, working memory, task switching) needed to appropriately select and monitor goals (Killgore, 2010). For instance, poorly slept individuals are less aware of abstract goals or standards such as morals, perceive less value in rewards that require effort, fail to incorporate feedback into ongoing goal pursuit, and prefer riskier courses of action.

A key aspect of how sleep loss hinders optimal goal selection and pursuit involves blunted positive emotions necessary for goal pursuit (e.g., desire and enthusiasm) and heightened negative emotions that often impede goal striving (e.g., anxiety). Specifically, those who are sleep deprived tend to report more negative affect and less positive affect, perceive neutral stimuli as having negative connotations, have more intense emotional reactions, and show an impaired capacity to reappraise and inhibit these emotions (Tempesta et al., 2010). The importance of sleep for effective self-regulation is further suggested by broader links between sleep and interpersonal aggression, relationship satisfaction and conflict, and unethical behavior (Barnes, Schaubroeck, Huth, & Ghumman, 2011; Krizan & Herlache, 2016; Troxel, 2010). As many health behaviors require self-regulation because they are burdensome (e.g., exercising, getting a mammography) or require restraint (e.g., not smoking, not eating fast food), how sleep relates to self-regulation is key for understanding initiation and maintenance of health behaviors.

Third and finally, all these findings underscore that losing sleep (or experiencing fragmented sleep) impairs vigilance, working memory, monitoring of changes in oneself and the environment, and the ability to change thinking strategies or inhibit automatic responses. At a more basic level, then, disrupting an individual's sleep seems to compromise the person's ability to engage in reflexive, rule-based processing of social information that requires effort and is dependent on motivation and cognitive capacity (Kahneman & Frederick, 2005). As a result, individuals may be more likely to engage in low-effort intuitive and associative processing of social information while pursuing social goals. Because processing of social information central to understanding most social phenomena (e.g., social judgment, attribution, persuasion, aggression, emotions) involves a more intuitive, associative system (impulsive; System 1) and a more rational, rule-based system (reflexive; System 2), sleep may be one key factor in how these systems are deployed in service of social cognition (Kahneman & Frederick, 2005). Although these implications of sleep for social processing are largely unexplored, existing evidence does suggest that losing sleep due to daylight savings (i.e., moving the clock forward in the spring) increases police officer's reliance on racial stereotypes when making traffic stop decisions (Wagner, Barnes, & Guarana, 2016), while a night of poor sleep impairs individuals' stop decisions and undermines taking the perspective of one's romantic partner (Gordon & Chen, 2014). How sleeplessness impacts elaboration of persuasive messages, attitude change, activation of stereotypic knowledge representations, and social perceptions of warmth and competence remains to be investigated. How sleep affects these social cognitive processes is of particular importance to health intervention endeavors as the cognitive and affective repercussions of sleep loss during exposure to health interventions likely represent an often overlooked barrier to health change.

Conclusion

Sleep is intrinsically tied to social behavior—socializing shapes chronic and acute sleep patterns, and how and when people sleep impacts their ability to regulate social behavior. Because sleep is an essential health outcome in itself while influencing many other health outcomes and behaviors, the dynamics between sleep and social behavior represent key considerations in theories of health behavior and their application toward interventions.

Author Biographies

Zlatan Krizan, PhD, is an associate professor of psychology and an affiliate for the Center for Study of Violence at Iowa State University in Ames, Iowa. He directs the Sleep, Self, and Personality Laboratory that investigates how sleep and dreams intersect with social behavior and individual differences in personality. A key focus of the research is how sleep intersects with self-control processes, personality, and health behaviors.

Garrett Hisler, MS, is a McNair scholar who is currently completing his doctoral training as a graduate assistant in Dr. Zlatan Krizan's Sleep, Self, and Personality Laboratory at Iowa State University's Department of Psychology. His research focuses on the bidirectional relationship of sleep and self-regulation and the role of personality in these dynamics.

References

- Almeida, D. M. (2005). Resilience and vulnerability to daily stressors assessed via diary methods. *Current Directions in Psychological Science*, 14(2), 64–68.
- Barnes, C. M., Schaubroeck, J., Huth, M., & Ghumman, S. (2011). Lack of sleep and unethical conduct. *Organizational Behavior and Human Decision Processes*, 115(2), 169–180.
- Carskadon, M. A., & Dement, W. C. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 16–26). St. Louis, MO: Elsevier Saunders.
- Czeisler, C. A., Duffy, J. F., Shanahan, T. L., Brown, E. N., Mitchell, J. F., Rimmer, D. W., ... Dijk, D. J. (1999). Stability, precision, and near-24-hour period of the human circadian pacemaker. *Science*, 284(5423), 2177–2181.
- Ehlers, C. L., Frank, E., & Kupfer, D. J. (1988). Social zeitgebers and biological rhythms: A unified approach to understanding the etiology of depression. *Archives of General Psychiatry*, 45(10), 948–952.
- Engle-Friedman, M., Riela, S., Golan, R., Ventuneac, A. M., Davis, C. M., Jefferson, A. D., & Major, D. (2003). The effect of sleep loss on next day effort. *Journal of Sleep Research*, 12(2), 113–124.
- Gordon, A. M., & Chen, S. (2014). The role of sleep in interpersonal conflict: Do sleepless nights mean worse fights? *Social Psychological and Personality Science*, 5(2), 168–175.
- Gunn, H. E., Buysse, D. J., Hasler, B. P., Begley, A., & Troxel, W. M. (2014). Sleep concordance in couples is associated with relationship characteristics. *Sleep*, 38(6), 933–939.
- Hasler, B. P., Mehl, M. R., Bootzin, R. R., & Vazire, S. (2008). Preliminary evidence of diurnal rhythms in everyday behaviors associated with positive affect. *Journal of Research in Personality*, 42(6), 1537–1546.
- Hasler, B. P., & Troxel, W. M. (2010). Couples' nighttime sleep efficiency and concordance: Evidence for bidirectional associations with daytime relationship functioning. *Psychosomatic Medicine*, 72(8), 794–801.
- Hisler, G., Phillips, A. L., & Krizan, Z. (2017). Individual differences in chronotype and time-of-exercise interact to predict physical activity. *Annals of Behavioral Medicine*, 51, 391–401. doi:10.1007/s12160-016-9862-0

- Hublin, C., Partinen, M., Koskenvuo, M., & Kaprio, J. (2007). Sleep and mortality: A population-based 22-year follow-up study. *Sleep*, 30(10), 1245.
- Irish, L. A., Kline, C. E., Gunn, H. E., Buysse, D. J., & Hall, M. H. (2015). The role of sleep hygiene in promoting public health: A review of empirical evidence. *Sleep Medicine Reviews*, 22, 23–36.
- Kahneman, D., & Frederick, S. (2005). A model of intuitive judgment. In K. J. Holyoak & R. G. Morrison (Eds.), *The Cambridge handbook of thinking and reasoning* (pp. 267–293). New York, NY: Cambridge University Press.
- Killgore, W. D. S. (2010). Effects of sleep deprivation on cognition. *Progress in Brain Research*, 185, 105–129.
- Krizan, Z., & Herlache, A. D. (2016). Sleep disruption and aggression: Implications for violence and its prevention. *Psychology of Violence*, 6(4), 542.
- Krizan, Z., & Hisler, G. (2016). The essential role of sleep in self-regulation. In K. Vohs & R. Baumeister (Eds.), *The handbook of self-regulation* (3rd ed., pp. 182–202). New York, NY: Guilford.
- Maume, D. J. (2013). Social ties and adolescent sleep disruption. *Journal of Health and Social Behavior*, 54(4), 498–515.
- Mistlberger, R. E., & Skene, D. J. (2004). Social influences on mammalian circadian rhythms: Animal and human studies. *Biological Reviews*, 79(3), 533–556.
- Monk, T. H., Petrie, S. R., Hayes, A. J., & Kupfer, D. J. (1994). Regularity of daily life in relation to personality, age, gender, sleep quality and circadian rhythms. *Journal of Sleep Research*, 3(4), 196–205.
- Reynolds, A. C., & Banks, S. (2010). Total sleep deprivation, chronic sleep restriction and sleep disruption. *Progress in Brain Research*, 185, 91–103.
- Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews*, 14(1), 19–31.
- Roenneberg, T., Wirz-Justice, A., & Mellow, M. (2003). Life between clocks: Daily temporal patterns of human chronotypes. *Journal of Biological Rhythms*, 18(1), 80–90.
- Tempesta, D., Couyoumdjian, A., Curcio, G., Moroni, F., Marzano, C., De Gennaro, L., et al. (2010). Lack of sleep affects the evaluation of emotional stimuli. *Brain Research Bulletin*, 82, 104–108.
- Troxel, W. M. (2010). It's more than sex: Exploring the dyadic nature of sleep and implications for health. *Psychosomatic Medicine*, 72(6), 578–586.
- Van Dongen, H. P. A., & Dinges, D. F. (2003). Investigating the interaction between the homeostatic and circadian processes of sleep-wake regulation for the prediction of waking neurobehavioural performance. *Journal of Sleep Research*, 12(3), 181–187.
- Wagner, D. T., Barnes, C. M., & Guarana, C. L. (2016). Law and error: Daylight saving time, race and police harassment. *American Economic Review*, Manuscript in preparation.
- Walker, M. P. (2010). Sleep, memory and emotion. *Progress in Brain Research*, 185, 49–68.
- Wittmann, M., Dinich, J., Mellow, M., & Roenneberg, T. (2006). Social jetlag: Misalignment of biological and social time. *Chronobiology International*, 23, 497–509.
- Woods, H. C., & Scott, H. (2016). #Sleepyteens: Social media use in adolescence is associated with poor sleep quality, anxiety, depression and low self-esteem. *Journal of Adolescence*, 51, 41–49.

Suggested Reading

- Hasler, B. P., Mehl, M. R., Bootzin, R. R., & Vazire, S. (2008). Preliminary evidence of diurnal rhythms in everyday behaviors associated with positive affect. *Journal of Research in Personality*, 42(6), 1537–1546.
- Irish, L. A., Kline, C. E., Gunn, H. E., Buysse, D. J., & Hall, M. H. (2015). The role of sleep hygiene in promoting public health: A review of empirical evidence. *Sleep Medicine Reviews*, 22, 23–36.

- Krizan, Z., & Hisler, G. (2016). The essential role of sleep in self-regulation. In K. Vohs & R. Baumeister (Eds.), *The handbook of self-regulation* (3rd ed., pp. 182–202). New York, NY: Guilford.
- Troxel, W. M. (2010). It's more than sex: Exploring the dyadic nature of sleep and implications for health. *Psychosomatic Medicine*, 72(6), 578–586.
- Wittmann, M., Dinich, J., Merrow, M., & Roenneberg, T. (2006). Social jetlag: Misalignment of biological and social time. *Chronobiology International*, 23, 497–509.

Social Identity

S. Alexander Haslam, Catherine Haslam, Jolanda Jetten,
Tegan Cruwys, and Genevieve A. Dingle

*Faculty of Health and Behavioural Sciences, School of Psychology, University of
Queensland, St. Lucia, QLD, Australia*

People's social lives and the social worlds they inhabit have a critical bearing on their health. For most health psychologists this is an uncontroversial observation, as there is abundant evidence both (a) that social contact, social connections, and a rich social life are good for one's well-being and (b) that those who are impoverished in this respect (e.g., because they are socially isolated as a result of poverty, prejudice, or other forms of social exclusion) suffer from relatively poor health outcomes. However, the social identity approach to health provides a more nuanced analysis of such patterns in suggesting that particular forms of social interaction have an especially important role to play in health dynamics. These are those that are grounded in shared social identity—a person's internalized sense of shared group membership and an associated sense that they are part of an "us" that is bigger than "me" alone.

This argument lies at the heart of the social identity approach to health: a theoretical perspective on health and well-being that has become increasingly influential in recent years. In what follows, we set out the core tenets of this approach and explain why its ideas are so central to a range of issues that are central to health psychology.

Social Identity as a Basis for Group Behavior

When people refer to "the self," they are typically referring to something singular about a person. A person who is self-confident, for example, is understood to be assured of their personal place in the world. Importantly, though, we also talk about the self in the first-person plural, in terms of our social identities as "us" and "we." Accordingly, we can define ourselves as "us Australians," "us psychologists," "us Democrats," and so on and be self-confident as members of these groups. The fact that we do this speaks to the capacity for the self to be

defined not only in unique terms but also in terms of attributes and qualities we share with other people.

The significance of this point for psychology was first illustrated in a famous series of experiments conducted by Henri Tajfel and his colleagues that came to be known as the minimal group studies. In these, schoolboys were assigned to essentially meaningless groups (e.g., supposedly on the basis of their liking for abstract paintings by Klee or Kandinsky) and had the task of assigning points (signifying small amounts of money) to other members of both their own group (their ingroup) and another group (the outgroup). What the researchers found was that even these most minimal of conditions were sufficient to produce group behavior—with many of the boys adopting a strategy in which they awarded points preferentially to members of their ingroup than to those in the outgroup. Moreover, they did so even if this meant their group would end up with worse outcomes than it might have had otherwise (e.g., if the participants had adopted a strategy in which both groups received a high number of points). Seeking to make sense of these findings, researchers came to understand that they reflected the fact that for participants in the studies, the process of acting in terms of their group membership was a way of making an otherwise meaningless situation meaningful. In particular, the actions they engaged in were part of a strategy to attain and maintain a positive and distinctive social identity (as “us in the Klee group” as opposed to “them in the Kandinsky group”).

Building upon this analysis, John Turner subsequently noted that it was the participants’ capacity to act in terms of social identity that made this group behavior possible (Turner, 1982). In other words, it was only because the boys could categorize themselves as “us in the Klee group” that they were able to act as members of the Klee group. This, he argued, is true of all other groups too. So it is only because (and to the extent that) Australian people can define themselves in terms of a social identity as Australians that they are able to behave as an organized, coordinated national entity. And the same is true for all other groups—teams, clubs, societies, communities, unions, churches, and so on.

Social Identity as a Basis for Health

The foregoing observation is clearly important when it comes to understanding the psychology of group behavior—for example, if we want to explain why people sometimes engage in social competition and conflict. But why is social identity important for health? The core reason is that humans are social animals who live, and have evolved to live, in social groups. In short, group life makes us human and is a key source of personal meaning, purpose, and worth. Among other things, this means that, like hunger and thirst, physical and psychological isolation are inimical to our makeup and design. And in this regard, the fundamental significance of social identity is that, without it, as Turner (1982) argued, group behavior is impossible.

If one considers a football team, for example, it is the fact that its members share social identity that allows them to coordinate their play and function as a meaningful social and psychological entity. Shared social identity (as “us members of Team X”) thus allows team members to harness the power and access the benefits of the group, and this at least partly explains why feeling part of a team is generally good for health and well-being (Steffens, Haslam, Schuh, Jetten, & van Dick, 2016). Moreover, the more that team members define themselves in terms of shared identity—that is, the higher their social identification—the more true this will tend to be.

Of course this point is not restricted to football teams, but is true of all collectives (e.g., recreational, religious, occupational, political, and community groups). The general point here, then, is that social identities and social identifications allow us to fulfill our potential as humans and hence will have important—and generally positive—implications for health. Consistent with this point, recent meta-analyses have found (a) that people's social identification with a broad range of groups is negatively and reliably associated with depression ($r = -.25$; Cruwys, Haslam, Dingle, Haslam, & Jetten, 2014) and (b) that employees' social identification with work teams is positively and reliably associated with both psychological well-being and the absence of stress ($r_s = .27, .18$, respectively; Steffens, Haslam, et al., 2016).

Further support for this proposition comes from research that used data from the English Longitudinal Survey on Ageing to examine the power of different types of social connection to predict the cognitive health of more than 3,000 English adults over time (Haslam, Cruwys, & Haslam, 2014). Contrary to the idea that all forms of social connection are equally protective of health, this analysis found that social group ties proved far more beneficial than individual ties. Indeed, only group engagement made a significant and sustained contribution to subsequent cognitive function, and this contribution became more pronounced as people became more vulnerable (i.e., with increasing age). Similarly, a study of over 800 Australian high school students showed that psychological health (in the form of personal self-esteem) was predicted not by students' interpersonal social network ties, but only by the quality of the students' group-based ties (Jetten et al., 2015).

Stated slightly differently, the social identity approach to health suggests that social identities are beneficial to health because they are the basis for meaningful forms of group life and hence give people access to important social and psychological resources (Jetten, Haslam, Haslam, Dingle, & Jones, 2014). Consistent with this idea, evidence from a large number of studies supports the argument that the more social identities a person has access to (i.e., the more group memberships they have internalized as part of their self), the better their physical, mental, and cognitive health. This prediction is confirmed by research with multiple populations including high school students (Jetten et al., 2015), retirees (Steffens, Cruwys, Haslam, Jetten, & Haslam, 2016), people recovering from a stroke (Haslam et al., 2008), or people staving off depression (Cruwys et al., 2013).

However, it also follows from this analysis that if our capacity to form meaningful social connections is compromised (e.g., because we lose or lack social identities or because our social identities are stigmatized), then this will limit our access to these same social and psychological resources and hence will threaten psychological and social functioning (e.g., Cruwys et al., 2014). This can also be true if, as a function of their particular nature, groups exert a negative influence on our lives because the content of a given social identity (e.g., "us depressives," "us heroin users") is harmful in some way (e.g., because it has negative connotations or prescribes toxic behavior; Dingle, Stark, Cruwys, & Best, 2015). A key point here, then, is that much of the power of the social identity approach to health derives from its capacity to understand the ways in which group and identity processes are implicated in negative health-related dynamics as well as those that are more positive (Haslam, Jetten, Cruwys, Dingle, & Haslam, 2018).

Social Identity as a Health-Enhancing Resource

Social identity theory (Tajfel & Turner, 1979) and self-categorization theory (Turner, 1982; Turner, Hogg, Oakes, Reicher, & Wetherell, 1987) together comprise the social identity approach.

Work informed by this approach is generally concerned with exploring the determinants of social identity (and social identification) as well as its consequences for social behavior (e.g., Haslam et al., 2018; Jetten et al., 2017). As well as having broad implications for the analysis of social behavior, this work has particular relevance for clinical and health psychology in allowing us to understand both the origins and the impact of health-related social identity resources.

Social Connection

One of the primary consequences of coming to see ourselves as sharing social identity with others is that this draws them closer to us psychologically. As spelled out within self-categorization theory, a key reason for this is that shared social identity transforms people who, as individuals, are different from the self (i.e., “others”) into people who, as group members, are part of the self (Turner et al., 1987).

By way of illustration, as individuals, passengers on the London Underground have no connection to each other and treat each other as strangers—with indifference. However, if circumstances were to lead them to see themselves as sharing social identity, then passengers would tend to see themselves as having much more in common, and their psychological orientation to each other would become much more positive as a result. This point was supported in research that documented the sense of solidarity and shared resolve that emerged among passengers who were victims of the London bombings in 2005. It has also been documented in work that examines passengers’ experiences of sharing train carriages with football supporters when those passengers are supporters of the same team, a different team, or no team at all. More prosaically, a range of controlled experimental studies—including those that involve minimal groups—show that manipulations that make salient a social identity that is shared with another person (e.g., as Australians, team members, or Klee likers) serve to make that person appear more similar to the perceiver.

Furthermore, as well as changing abstract perceptions of similarity and connectedness, such manipulations also change our openness and receptivity to others, as evidenced in perceptions of liking and trust. Not only do we understand others to be more yoked to us in such circumstances, but once we relate to them as ingroup members we also embrace them more enthusiastically. Consequently, we also experience a greater sense of belonging and feel more comfortable in their company. Indeed, one key reason why social identity (and social identification) tends to have positive implications for health is that it serves to counteract—and indeed is inherently antithetical to—a sense of psychological isolation (Cruwys et al., 2014).

Effective Social Support

As people come to define themselves in terms of shared group membership, it is not simply their perceptions of each other that change. So too does their behavior. Not least, this is because social identity provides group members with the motivation to engage with each other in ways that advance the identity they share—so that if a person sees themselves as part of a given group (e.g., a family, a team, an organization), then they are motivated to “do what it takes” to help the group thrive. One obvious way they can do this is by helping each other out by providing social support when it is perceived to be needed (e.g., if a member of the group is in difficulty). Such support can take an emotional, intellectual, or material form and again flows from the fact that shared social identity transforms “others” into “self.”

Another large body of empirical work supports this claim (for a review see Haslam, Reicher, & Levine, 2012). For example, studies show that when a football fan trips and falls, a passerby

is far more likely to stop and help them if the victim appears to be a supporter of the same team rather than of a rival one. However, if the observer's more general social identity as a football fan is made salient (rather than their identity as a supporter of a specific team), then they are more likely to help the supporter of the rival team than someone who appears not to support any team.

On a larger scale, studies of international aid show that charitable giving also follows the contours of shared identity. However, a person's status as an ingroup or outgroup member (and hence the amount of support they receive) varies as a function of the specific identity lens through which they are viewed. Studies have shown, for example, that British people donate more money to Italian earthquake victims if they are encouraged to define themselves as European (in which case they share identity with Italians) rather than as British (where they do not).

Importantly, though, effective support is not just a matter of aid provision; it also depends on recipients being receptive to such charity. And this receptiveness too is structured by shared social identity. Studies thus show that people are more positively oriented to support when it is offered by ingroup rather than outgroup members—in large part because they are more trusting of the motives that lie behind it (Haslam et al., 2012).

Together then, these two sides of the support equation combine to mean that support tends both to be more forthcoming and to be more effective when it occurs within, rather than across, the boundaries of social identity. The same is true for the process of *social influence* that is implicated in most forms of support (and in all forms of leadership; Haslam, Reicher, & Platow, 2011). In particular, whether people give useful advice, and whether others take it on board, depends very much on the degree to which they see themselves as sharing social identity. This is another point that has broad relevance for health psychology. For example, it helps us understand the therapeutic alliance between practitioners and clients as something that is predicated on social identity. And it also helps us understand why interventions tend to be less successful when they occur across identity fault lines (e.g., of ethnicity, culture, and social class).

Meaning and Worth

We noted above that within the minimal groups studies, Tajfel observed that it was the process of acting as group members (in terms of social identity) that allowed participants to make an otherwise meaningless task meaningful. At the time he did not make too much of this, but it turns out that this is largely true of most other forms of social behavior. Looked at from the outside, many (if not most) social activities have the capacity to appear meaningless—whether they involve kicking a football, singing an operatic aria, or playing board games. What makes them meaningful (and enjoyable) is the process of identifying with the groups that engage in these activities.

In this regard too, it is obviously the case that what people do in their groups (and as individual group members) is largely informed by the *content* of their identity and what it means to be part of that group. Football teams play football, choir members sing, and board gamers play board games. The significance of this observation for health and well-being is that in this way, social identities lend meaning, direction, and purpose to actions that would otherwise be meaningless. This point was brought home in Durkheim's groundbreaking study of suicide in which he noted that people are far less likely to kill themselves during wartime—a pattern that he attributed to the fact that war had the power to bring people together and focus their energies on a common goal. At the same time, the process of contributing to such goals (e.g., by exerting oneself in a football match, by singing enthusiastically in a concert, by taking a board

game seriously) tends to be valued and valorized by those with whom one shares social identity, and hence it instills in group members a sense that they and their efforts are worthwhile.

Control, Efficacy, and Power

Finally, because it provides a basis for people to participate in, and promote, collective projects, it is also the case that shared social identity tends to give people a sense of agency and control. As social identity theory suggests, at a group level, this should furnish them with a sense of being in charge of their own destiny and of having power in the world (Haslam et al., 2008, 2011). This is seen in a range of studies that show that as people's sense of social identity increases, so too does their sense of agency and power. Moreover, as studies of protest groups have shown (and as fans of perennially underperforming sports teams understand), it is not necessary for a group to achieve its aims in order for these benefits to accrue.

At an individual level too, shared social identity also furnishes group members with a sense of efficacy and of personal control over their lives. Research thus shows that people report a greater sense of personal control to the extent that they identify highly with their community, with a political party, with their nation, or with humanity as a whole (Greenaway et al., 2015). Again, this pattern is not contingent on group success—being apparent, for example, among highly identified Republicans immediately after their party had failed to win the 2012 presidential election. Here, then, internalized social identities help group members to interpret, understand, and recover from such failure.

An abundance of work on the importance of a sense of control for health underlines the importance of this observation. Indeed, the *World Happiness Report* identifies control as one of the “six pillars” of life satisfaction. Yet while perceived control is generally thought to have personal origins, social identity research suggests that it often has its basis in group identities and identifications.

Intervention: Groups 4 Health

A key message of the social identity approach to health is that social connectedness, and group connectedness especially, is central to health behavior and health outcomes. But how might we translate knowledge of group and social identity processes into applied interventions?

Evidence from group-based interventions points to three features that appear to be shared by those that are the most effective. First, interventions seem to be more effective to the extent that they have an integrated focus on issues of social process, cognition, and behavior—rather than looking at any one of these things in isolation. Second, interventions tend to be more effective to the extent that the groups at their heart are meaningful to their members and involve activities they value, rather than having an arbitrary or trivial basis. Third, more effective interventions create a sense of shared purpose that binds people together and enables them to work collectively toward common goals.

Building on these insights, researchers have used social identity theorizing as a basis for developing a manualized intervention—Groups 4 Health (G4H)—that seeks to improve health by enhancing people's group-based social connectedness in the context of a non-stigmatizing in vivo group experience (Haslam, Cruwys, Haslam, Dingle, & Chang, 2016). G4H is composed of five modules—the 5Ss—as shown in Figure 1. Together, these aim to give people the knowledge and skills not only to understand the nature and importance of their social identity resources but also to manage them effectively in the long term.

1. Schooling	• Raising awareness of the value of groups for health and of ways to harness this
2. Scoping	• Developing social maps to identify existing connections and areas for social growth
3. Sourcing	• Training skills to maintain and utilise existing networks and reconnect with valued groups
4. Scaffolding	• Using group as platform for new social connections and to train effective engagement
5. Sustaining	• Reinforcing key messages and troubleshooting (held one month later as a booster session)

Figure 1 The modules in Groups 4 Health: the 5Ss (Haslam et al., 2016; see also Haslam et al., 2018).

A proof-of-concept trial with participants experiencing social isolation and affective disturbance indicated that G4H reduced clinical symptoms of depression as well as anxiety and stress (Haslam et al., 2016). In addition, it proved enjoyable for both participants and facilitators and led to significant improvements in social anxiety, social functioning, self-esteem, and life satisfaction. Importantly too, follow-up analysis indicated that most of these improvements were sustained—or even enhanced—6 months after the program had ended (see Haslam et al., 2016). The reason for this, we suggest, is that, because they are foundational to adaptive social functioning, groups are an especially sustainable source of psychological health.

Future Directions

Because the social identity approach to health is relatively new, it is clearly the case that more work needs to be done to test and validate its core assumptions and to establish their practical relevance. Work of this form is currently underway in relation to a range of health topics including social disadvantage, stigma, stress, trauma, aging, depression, chronic mental health, eating behavior, brain injury, acute pain, addiction, and physical health (see Haslam et al., 2018). In order to establish the generalizability and robustness of G4H, further validation studies are also currently in progress to establish its applicability to a range of populations (e.g., students, new mothers, people recovering from addiction, retirees).

Conclusion

The theorizing and evidence that supports the social identity approach has been built up over nearly half a century. However, it is only in the past decade that the approach has been applied specifically to the domain of health (e.g., see Haslam et al., 2018; Haslam, Jetten, Postmes, & Haslam, 2009; Jetten, Haslam, & Haslam, 2012). Nevertheless, in this short time, the evidence base and impact of the approach has increased exponentially, with the result that there are now well over 500 publications that speak to its utility.

The model that this work supports is represented schematically in Figure 2. This points to the fact that there are important (if complex) interconnections between social identity, group

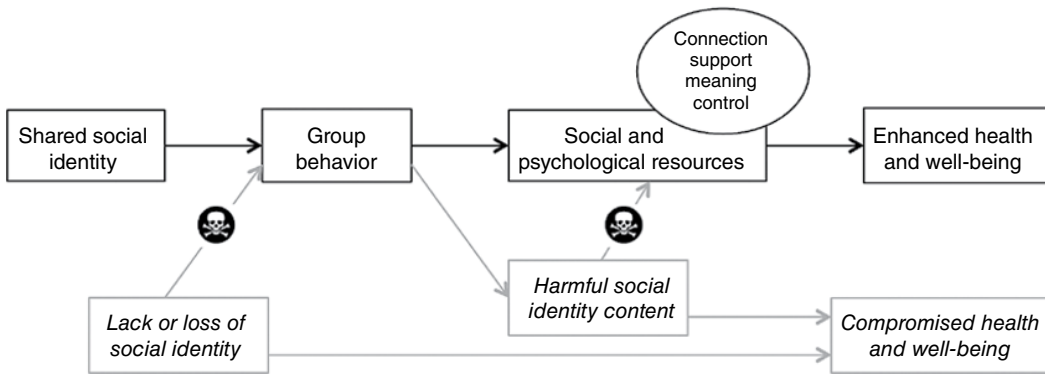


Figure 2 Schematic representation of the social identity approach to health.

behavior, and health and that the capacity for social identity to deliver positive health outcomes rests on its capacity to give individuals access to important social and psychological resources. However, it also suggests that health benefits can only be reaped from groups that are psychologically important for their members and, more generally, that the extent to which groups influence health—whether for good or for ill—depends on individuals’ strength of identification with them.

Importantly, the resources that social identities provide are not only psychological but also material. They thus relate not only to people’s sense of connection, support, meaning, and control but also to the social reality of these things. This is because, as social identity theorists grasped from the outset, groups are fundamental not only to our sense of self but also to our capacity to do things with others in the world and by this means to contribute to both social change and social progress (Tajfel & Turner, 1979). It is for this reason, then, that social identities are vital not just for the health of individuals but for that of society as a whole.

Acknowledgment

Work on this entry was supported by a fellowship from the Australian Research Council (FL110100199).

Author Biographies

S. Alexander Haslam is a professor of psychology and Australian Research Council Laureate Fellow at the University of Queensland. His research focuses on the study of group and identity processes in social, organizational, and health contexts. Together with colleagues he has written and edited 11 books and over 200 peer-reviewed articles on these topics including, most recently, *The New Psychology of Leadership: Identity, Influence and Power* (Psychology Press, 2011, with S. Reicher & M. J. Platow).

Catherine Haslam is professor of clinical psychology at the University of Queensland. Her research focuses on the social and cognitive consequences of identity-changing life transitions (e.g., trauma, disease, aging). She is an associate editor of the *British Journal of Psychology* and co-author of *The New Psychology of Health: Unlocking the Social Cure* (Routledge, in press; with J. Jetten, C. Cruwys, G. A. Dingle, and S. A. Haslam).

Jolanda Jetten is a professor of social psychology and research fellow at the University of Queensland. She is a former editor of the *British Journal of Social Psychology*. She has published over 140 peer-reviewed articles on topics related to social identity, group processes, and inter-group relations and previously co-edited *The Social Cure: Identity, Health and Well-Being* (Psychology Press, 2012; with C. Haslam & S. A. Haslam).

Tegan Cruwys is a lecturer in the School of Psychology, University of Queensland, a practicing clinical psychologist, and a recipient of an Australian Research Council Discovery Early Career Research Award. Her research investigates the social-psychological determinants of health, with a particular focus on health behaviors, mental health, and vulnerable populations.

Genevieve A. Dingle is a senior lecturer in the School of Psychology, University of Queensland. Her research focuses on social factors in addiction, depression, and chronic mental health problems, people experiencing homelessness, and the recovery process. She is on the editorial board of the *British Journal of Clinical Psychology*.

References

- Cruwys, T., Dingle, G., Haslam, S. A., Haslam, C., Jetten, J., & Morton, T. (2013). Social group memberships protect against future depression, alleviate depression symptoms, and prevent depression relapse. *Social Science and Medicine*, 98, 179–186. doi.org/10.1016/j.socscimed.2013.09.013
- Cruwys, T., Haslam, S. A., Dingle, G. A., Haslam, C., & Jetten, J. (2014). Depression and social identity: An integrative review. *Personality and Social Psychology Review*, 18, 215–238. doi:10.1177/1088868314523839
- Dingle, G. A., Stark, C., Cruwys, T., & Best, D. (2015). Breaking good: Breaking ties with social groups may be good for recovery from substance misuse. *British Journal of Social Psychology*, 54, 236–254.
- Greenaway, K., Haslam, S. A., Branscombe, N. R., Cruwys, T., Ysseldyk, R., & Heldreth, C. (2015). From “we” to “me”: Group identification enhances perceived personal control with consequences for health and well-being. *Journal of Personality and Social Psychology*, 109, 53–74. doi:10.1037/pspi0000019
- Haslam, C., Cruwys, T., & Haslam, S. A. (2014). “The we’s have it”: Evidence for the distinctive benefits of group engagement in enhancing cognitive health in aging. *Social Science and Medicine*, 120, 57–66. doi:10.1016/j.socscimed.2014.08.037
- Haslam, C., Cruwys, T., Haslam, S. A., Dingle, G., & Chang, M. X.-L. (2016). Groups 4 Health: Evidence that a social-identity intervention that builds and strengthens social group membership improves mental health. *Journal of Affective Disorders*, 194, 188–195. doi:10.1016/j.jad.2016.01.010
- Haslam, C., Holme, A., Haslam, S. A., Iyer, A., Jetten, J., & Williams, W. H. (2008). Maintaining group memberships: Social identity continuity predicts well-being after stroke. *Neuropsychological Rehabilitation*, 18, 671–691. doi:10.1080/09602010701643449
- Haslam, C., Jetten, J., Cruwys, C., Dingle, G. D., & Haslam, S. A. (2018). *The new psychology of health: Unlocking the social cure*. London: Routledge.
- Haslam, S. A., Jetten, J., Postmes, T., & Haslam, C. (2009). Social identity, health and well-being: An emerging agenda for applied psychology. *Applied Psychology*, 58, 1–23. doi:10.1111/j.1464-0597.2008.00379.x
- Haslam, S. A., Reicher, S. D., & Levine, M. (2012). When other people are heaven, when other people are hell: How social identity determines the nature and impact of social support. In J. Jetten, C. Haslam, & S. A. Haslam (Eds.), *The social cure: Identity, health, and well-being* (pp. 157–174). Hove, UK: Psychology Press.
- Haslam, S. A., Reicher, S. D., & Platow, M. J. (2011). *The new psychology of leadership: Identity, influence and power*. Hove, UK: Psychology Press.

- Jetten, J., Branscombe, N. R., Haslam, S. A., Haslam, C., Cruwys, T., Jones, J. M., ... Zhang, A. (2015). Having a lot of a good thing: Multiple important group memberships as a source of self-esteem. *PLoS One*, 10(6), e0131035. doi:10.1371/journal.pone.0131035
- Jetten, J., Haslam, C., & Haslam, S. A. (Eds.) (2012). *The social cure: Identity, health and well-being*. Hove, UK: Psychology Press.
- Jetten, J., Haslam, C., Haslam, S. A., Dingle, G., & Jones, J. M. (2014). How groups affect our health and well-being: The path from theory to policy. *Social Issues and Policy Review*, 8, 103–130. doi:10.1111/sipr.12003
- Jetten, J., Haslam, S. A., Cruwys, T., Greenaway, K., Haslam, S. A., & Steffens, N. R. (2017). Advancing the social identity approach to health and well-being: Progressing the social cure research agenda. *European Journal of Social Psychology*, 47(7), 789–802.
- Steffens, N. K., Cruwys, T., Haslam, C., Jetten, J., & Haslam, S. A. (2016). Social group memberships in retirement are associated with reduced risk of premature death: evidence from a longitudinal cohort study. *BMJ Open*, 6(2), e010164.
- Steffens, N. K., Haslam, S. A., Schuh, S., Jetten, J., & van Dick, R. (2016). A meta-analytic review of social identification and health in organizational contexts. *Personality and Social Psychology Review*, 21(4), 303–335. doi:10.1177/1088868316656701
- Tajfel, H., & Turner, J. C. (1979). An integrative theory of intergroup conflict. In W. G. Austin & S. Worchel (Eds.), *The social psychology of intergroup relations* (pp. 33–47). Monterey, CA: Brooks/Cole.
- Turner, J. C. (1982). Towards a redefinition of the social group. In H. Tajfel (Ed.), *Social identity and intergroup relations* (pp. 15–40). Cambridge, UK: Cambridge University Press.
- Turner, J. C., Hogg, M. A., Oakes, P. J., Reicher, S. D., & Wetherell, M. S. (1987). *Rediscovering the social group: A self-categorization theory*. Cambridge, MA: Basil Blackwell.

Suggested Reading

- Cruwys, T., Haslam, S. A., Dingle, G. A., Haslam, C., & Jetten, J. (2014). Depression and social identity: An integrative review. *Personality and Social Psychology Review*, 18, 215–238. doi:10.1177/1088868314523839
- Haslam, C., Jetten, J., Cruwys, C., Dingle, G. D., & Haslam, S. A. (2018). *The new psychology of health: Unlocking the social cure*. London, UK: Routledge.
- Haslam, S. A., Jetten, J., Postmes, T., & Haslam, C. (2009). Social identity, health and well-being: An emerging agenda for applied psychology. *Applied Psychology: An International Review*, 58, 1–23. doi:10.1111/j.1464-0597.2008.00379.x
- Jetten, J., Haslam, C., & Haslam, S. A. (Eds.) (2012). *The social cure: Identity, health and well-being*. Hove, UK: Psychology Press.
- Jetten, J., Haslam, S. A., Cruwys, T., Greenaway, K., Haslam, S. A., & Steffens, N. R. (2017). Advancing the social identity approach to health and well-being: Progressing the social cure research agenda. *European Journal of Social Psychology*.

Social Influence

Karen S. Rook, Dara H. Sorkin, and Kelly Biegler

Department of Psychological Science, University of California, Irvine, CA, USA

People who have more social ties are known to have a significantly lower risk of mortality and morbidity, and this lower risk is usually attributed to the health-protective benefits of social support provided by others (Cohen, 2004). Social support refers to the aid and care that social network members often provide in times of need, and it is important in helping to buffer people from the harmful effects of life stress. Providing support is not the only process, however, by which social network members may contribute to health. They can also be a source of social influence, as people monitor each other's health behavior and seek to foster change when a health behavior (or set of behaviors) is perceived to be health damaging. A wife who is concerned about her husband's smoking, for example, might tell him how upset she feels when he smokes, might warn him of the long-term negative consequences of smoking, or might otherwise try to convince him to discontinue smoking. Researchers use different terms to describe such attempts to exert social influence, but a commonly used term is social control. Social control refers to efforts by social network members to monitor each other's health behavior and to intervene to discourage health-damaging behavior (Lewis & Rook, 1999; Umberson, 1987). Social control, like social support, could be an important mechanism that explains why having close relationships is associated with better health and greater longevity. The two kinds of interpersonal processes differ, however, in other respects.

By serving as a source of aid and care in time of need, social support is typically experienced as welcome and affirming. Social control, in contrast, involves efforts to change or constrain a person's health behavior, which can be experienced as unwelcome and critical, rather than affirming (Rook, 2015). Yet theorists have argued that social control attempts have the potential to benefit health, even when they are not affirming. Pressuring a family member to follow a prescribed treatment regimen more closely may help to prevent serious illness complications, even if the pressure is frustrating or irritating. The idea that social control may contribute to improved health behavior even while it arouses psychological distress has come to be known as the dual effects hypothesis (Lewis & Rook, 1999). Researchers have recently extended this reasoning to suggest that social control might detract from a recipient's feelings of self-efficacy,

even though it is intended to benefit the recipient's health by fostering improved health behavior. Specifically, others' social control attempts may convey implicitly (or sometimes explicitly) the message that the recipient's self-control is poor, which could erode his or her feelings of self-efficacy (e.g., Franks et al., 2006) and could kindle feelings of guilt or embarrassment.

Does social control have these hypothesized dual effects? Existing evidence is somewhat mixed, but it currently suggests three conclusions. First, social control is sometimes associated with better health behavior, but it is often associated with worse health behavior. For example, more frequent social control has been related to less health-damaging behavior (such as smoking) and more health-enhancing behavior (such as exercise, sleep, sound dietary practices) in some studies, but it has been related to worse, rather than better, health behavior in numerous studies. Moreover, more frequent social control has been found to be related to hiding of unsound health behavior and pretended adoption of sound health behavior in some studies (Lewis & Rook, 1999; see review by Craddock, van Dellen, Novak, & Ranby, 2015).

Such evidence casts doubt on one component of the dual effects hypothesis—the idea that others' social control attempts tend to encourage better health behavior. Much of the evidence that casts doubt on this conclusion is derived from cross-sectional studies, however, and cross-sectional designs introduce ambiguity with respect to temporal order. Do unsound health behaviors encourage others to engage in social control attempts, or do others' social control attempts encourage people to engage in unsound health behaviors (e.g., by undermining self-efficacy or provoking behavioral reactance)? Longitudinal studies generally yield less ambiguous evidence, and some (though not all) longitudinal studies do suggest that greater social control is associated with improved health behavior over time (e.g., Knoll, Burkert, Scholz, Roigas, & Gralla, 2012; Stephens et al., 2009).

A second conclusion suggested by existing research is that social control attempts are often associated with psychological distress, including not only feelings of resentment and irritation but also feelings of guilt and shame (e.g., Rook, August, Stephens, & Franks, 2011). Links between greater social control and lower feelings of self-efficacy have also been reported (e.g., Berg et al., 2013). It is worth noting, however, that participants in some studies express appreciation for others' efforts to monitor their health behavior and nudge improvements (Rook et al., 2011). Individual differences exist in how people react to others' social control attempts, with evidence suggesting that factors such as gender (e.g., August & Sorkin, 2011; Martire et al., 2013), ethnicity (e.g., August & Sorkin, 2011), and expectations about the appropriateness of others' involvement in one's health (e.g., Rook et al., 2011) influence whether people react favorably or unfavorably to social control attempts. Similarly, features of the broader context in which social control attempts occur may also influence how people react to them. People in more satisfying relationships appear to respond more favorably to their partner's social control attempts, exhibiting greater health behavior change and little psychological distress (e.g., Knoll et al., 2012; Tucker, 2002). The specific health context in which social control occurs also appears to influence how people react. Individuals with a serious chronic illness who face the task of adhering to a complex set of health behaviors as part of their day-to-day illness management may understand that their partners' efforts to prompt or urge adherence, even if unwelcome, are well-intentioned (e.g., Badr, Yeung, Lewis, Milbury, & Redd, 2015). Similarly, if couples regard the day-to-day tasks of managing a chronic illness as a collaborative rather than an independent endeavor, patients may react more favorably to their partners' involvement in their health (Berg et al., 2008).

A third conclusion suggested by existing research is that the nature of the social control attempts themselves influence how people respond, with positive and negative strategies of social control eliciting markedly different responses. Positive control strategies refer to others'

attempts to persuade or motivate a person to improve his or her health behavior, whereas negative control strategies refer to others' attempts to pressure or coerce a person to make improvements (Stephens et al., 2009). Examples of positive strategies include reminding, persuading, bargaining, modeling, and providing contingent positive feedback. Examples of negative strategies include criticism, guilt induction, nagging, pressure, and withdrawal (Lewis & Butterfield, 2005). For example, a wife might try to persuade her husband to improve his adherence to a prescribed diet by reminding him of the health benefits of making better dietary choices, or, on the other hand, she might subtly criticize his food choices ("Are you really going to eat that?") or express exasperation ("I just do not understand how you can keep eating food that you know is bad for you.") Some researchers favor the terms persuasion and pressure to refer to these two different categories of social control tactics to avoid potential confusion between the nature of the influence attempt and its presumed effect on the recipient (Stephens et al., 2013). Across many studies, positive strategies have been found to be more likely to elicit improved health behavior and to avoid arousing psychological distress compared with negative strategies (Craddock et al., 2015). One challenge confronting researchers, however, in their efforts to identify distinctive effects of specific tactics of social control is that social network members frequently use multiple strategies of influence (Lewis & Butterfield, 2005).

The dual effects model of social control has continued to be elaborated in recent studies, with researchers investigating moderators that may indicate when the hypothesized effects are most likely to occur and mediating mechanisms that may explain why observed effects occur. Relationship characteristics and the health context of social control attempts, as noted earlier, are examples of potentially important moderators. The emotions aroused by social control attempts are examples of potentially important mediators, with social control expected to be more effective to the extent that the tactics used (such as reminders and explanations) elicit positive emotions and less effective, in contrast, to the extent that the tactics used (such as criticism or coercion) elicit negative emotions (e.g., Okun, Huff, August, & Rook, 2007; Tucker, Orlando, Elliott, & Klein, 2006). In the context of a serious chronic illness, however, even social control that arouses frustration or resentment may, nonetheless, help to foster adherence to a demanding self-care regimen (Badr et al., 2015). Thus, the specific health context in which social control attempts occur may influence the emotional and behavioral responses they elicit. Researchers have also begun to investigate the joint effects of social support and social control, positing, for example, that social control attempts may elicit more favorable responses in social relationships that also serve as a source of support (e.g., Khan, Stephens, Franks, Rook, & Salem, 2013).

Researchers have recently extended research on health-related social control to examine potential effects on the individuals engaged in social control attempts, such as spouses. It is plausible, for example, that having to monitor and seek to influence a partner's health behavior can be stressful for spouses, perhaps especially in the context of a chronic illness. Evidence consistent with this idea has emerged in studies of couples in which one individual is coping with type 2 diabetes (e.g., August, Rook, Franks, & Stephens, 2013; August, Rook, Stephens, & Franks, 2011). Whether negative effects of engaging in persistent health-related social control attempts would emerge in other health contexts is a question that warrants investigation in future studies.

Although most work on health-related social influence has emphasized actions by social network members that are intended to discourage health-compromising behavior, it is also important to note that others' social influence sometimes serves to encourage health-compromising behavior. In adolescence and young adulthood, for example, peer influence is known to play a role in the initiation of substance use (e.g., Wills, McNamara, Vaccaro, & Hirky, 1996). Even in the context of illness management, friends and family members sometimes

undermine rather than bolster adherence to a treatment regimen. For example, spouses of individuals with type 2 diabetes are known to tempt their partners occasionally to eat unhealthy food, thereby undermining the partner's adherence to a recommended dietary regimen (Henry, Rook, Stephens, & Franks, 2013). The particular circumstances that prompt such undermining remain poorly understood and represent a valuable focus of future research.

Social influence, like social support, is a common element of many close relationships. Health-related social control and social support often co-occur in close relationships, but these interpersonal processes differ conceptually and empirically and exhibit distinctive associations with health behavior (Rook, 2015). Social support has been investigated more extensively than has social control. Yet across different community samples, as many as 70–86% of participants report experiencing health-related social control attempts from their social network members (e.g., August & Sorkin, 2011; Lewis & Rook, 1999). Given how common this form of social influence is in close relationships, it warrants greater attention from researchers in future studies. It is particularly important to gain an understanding of the dynamics of health-related support and control as they unfold over the course of a chronic illness or other significant health transition. Initial expressions of social support may give way over time to increasingly insistent, and even caustic, forms of social control when individuals experiencing a serious health challenge exhibit limited interest or history of success in initiating and sustaining health behavior changes that are crucial to their health. Future research will hopefully shed light on ways that social network members can avoid engaging in caustic or coercive social influence attempts and, instead, can foster successful health behavior change and maintenance while preserving self-efficacy in the recipient and good will in the relationship.

Author Biographies

Karen S. Rook is a professor of psychology and social behavior at the University of California, Irvine. Her research investigates the role of social relationships in fostering or interfering with health-enhancing behavior, particularly in the context of chronic illness. Other research examines the effects of interventions designed to strengthen social relationships as a resource for successful health behavior change.

Dara H. Sorkin is an associate professor of medicine at the University of California, Irvine. Her research interests focus on social relationships and health, particularly as they may play a role in health disparities. She also conducts and evaluates behavioral health interventions in community settings that seek to reduce health risks and poor health outcomes among under-resourced populations.

Kelly Biegler is a research scientist at the University of California, Irvine. Her research focuses on examining the impact of health behaviors, quality of life, and chronic stress on disease management, clinical outcomes, and neurophysiologic and immunologic processes in patient populations. She also investigates the effects of community interventions on the health and well-being of at-risk, low-income populations.

References

- August, K. J., Rook, K. S., Franks, M. M., & Stephens, M. A. P. (2013). Spouses' involvement in their partners' diabetes management: Associations with spouse stress and perceived marital quality. *Journal of Family Psychology*, 27(5), 712–721. doi:10.1037/a0034181

- August, K. J., Rook, K. S., Stephens, M. A., & Franks, M. M. (2011). Are spouses of chronically ill partners burdened by exerting health-related social control? *Journal of Health Psychology, 16*(7), 1109–1119. doi:10.1177/1359105311401670
- August, K. J., & Sorkin, D. H. (2011). Support and influence in the context of diabetes management: Do racial/ethnic differences exist? *Journal of Health Psychology, 15*(5), 711–721. doi:10.1177/1359105310388320
- Badr, H., Yeung, C., Lewis, M. A., Milbury, K., & Redd, W. H. (2015). An observational study of social control, mood, and self-efficacy in couples during treatment for head and neck cancer. *Psychology & Health, 30*(7), 783–802. doi:10.1080/08870446.2014.994633
- Berg, C. A., Butner, J. E., Butler, J. M., King, P. S., Hughes, A. E., & Wiebe, D. J. (2013). Parental persuasive strategies in the face of daily problems in adolescent type 1 diabetes management. *Health Psychology, 32*(7), 719–728. doi:10.1037/a0029427
- Berg, C. A., Wiebe, D. J., Butner, J., Bloor, L., Bradstreet, C., Upchurch, R., ... Patton, G. (2008). Collaborative coping and daily mood in couples dealing with prostate cancer. *Psychology and Aging, 23*(3), 505–516. doi:10.1037/a0012687
- Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*, 676–684. doi:10.1037/0003-066X.59.8.676
- Craddock, E., vanDellen, M. R., Novak, S. A., & Ranby, K. W. (2015). Influence in relationships: A meta-analysis on health-related social control. *Basic and Applied Social Psychology, 37*(2), 118–130. doi:10.1080/01973533.2015.1011271
- Franks, M. M., Stephens, M. A. P., Rook, K. S., Franklin, B. A., Keteyian, S. J., & Artinian, N. T. (2006). Spouses' provision of health-related social support and control to patients participating in cardiac rehabilitation. *Journal of Family Psychology, 20*, 311–318. doi:10.1037/0893-3200.20.2.311
- Henry, S. L., Rook, K. S., Stephens, M. A. P., & Franks, M. M. (2013). Spousal undermining of older diabetic patients' disease management. *Journal of Health Psychology, 18*, 1550–1561. doi:10.1177/1359105312465913
- Khan, C. M., Stephens, M. A. P., Franks, M. M., Rook, K. S., & Salem, J. K. (2013). Influences of spousal support and control on diabetes management through physical activity. *Health Psychology, 32*, 739–747. doi:10.1037/a0028609
- Knoll, N., Burkert, S., Scholz, U., Roigas, J., & Gralla, O. (2012). *Anxiety, Stress & Coping, 25*(3), 291–307. doi:10.1080/10615806.2011.584188
- Lewis, M. A., & Butterfield, R. M. (2005). Antecedents and reactions to health-related social control. *Personality and Social Psychology Bulletin, 31*(3), 416–427. doi:10.1177/0146167204271600
- Lewis, M. A., & Rook, K. S. (1999). Social control in personal relationships: Impact on health behaviors and psychological distress. *Health Psychology, 18*, 63–71. doi:10.1037//0278-6133.18.1.63
- Martire, L. M., Stephens, M. A. P., Mogle, J., Schulz, R., Brach, J., & Keefe, F. J. (2013). Daily spousal influence on physical activity in knee osteoarthritis. *Annals of Behavioral Medicine, 45*(2), 213–223. doi:10.1007/s12160-012-9442-x
- Okun, M. A., Huff, B. P., August, K. J., & Rook, K. S. (2007). Testing hypotheses distilled from four models of the effects of health-related social control. *Basic and Applied Social Psychology, 29*(2), 185–193. doi:10.1080/01973530701332245
- Rook, K. S. (2015). Social networks in later life: Weighing positive and negative effects on health and well-being. *Current Directions in Psychological Science, 24*, 45–51. doi:10.1177/0963721414551364
- Rook, K. S., August, K. J., Stephens, M. A. P., & Franks, M. F. (2011). When does spousal social control provoke negative reactions in the context chronic illness? The pivotal role of patients' expectations. *Journal of Social and Personal Relationships, 28*, 772–789. doi:10.1177/0265407510391335
- Stephens, M. A. P., Fekete, E. M., Franks, M. M., Rook, K. S., Druley, J. A., & Greene, K. (2009). Spouses' use of pressure and persuasion to promote osteoarthritis patients' medical adherence after orthopedic surgery. *Health Psychology, 28*, 48–55. doi:10.1037/a0012385
- Stephens, M. A. P., Franks, M. F., Rook, K. S., Iida, M., Hempill, R., & Salem, J. K. (2013). Spouses' attempts to regulate day-to-day dietary adherence among patients with type 2 diabetes. *Health Psychology, 32*, 1029–1037. doi:10.1037/a0030018

- Tucker, J. S. (2002). Health-related social control within older adults' relationships. *Journal of Gerontology: Psychological Sciences*, 57, 387–395. doi:10.1093/geronb/57.5.P387
- Tucker, J. S., Orlando, M., Elliott, M. N., & Klein, D. J. (2006). Affective and behavioral responses to health-related social control. *Health Psychology*, 25(6), 715–722. doi:10.1037/0278-6133.25.6.715
- Umberson, D. (1987). Family status and health behaviors: Social control as a dimension of social integration. *Journal of Health and Social Behavior*, 28, 306–319.
- Wills, T. A., McNamara, G., Vaccaro, D., & Hirky, A. E. (1996). Escalated substance use: A longitudinal grouping analysis from early to middle adolescence. *Journal of Abnormal Psychology*, 105(2), 166–180. doi:10.1037/0021-843X.105.2.166

Suggested Reading

- August, K. J., & Sorkin, D. H. (2010). Marital status and gender differences in managing a chronic illness: The function of health-related social control. *Social Science & Medicine*, 71, 1831–1838. doi:10.1016/j.socscimed.2010.08.022
- Lewis, M. A., Butterfield, R. M., Darbes, L. A., & Johnston-Brooks, C. (2004). The conceptualization and assessment of health-related social control. *Journal of Social and Personal Relationships*, 21, 669–687. doi:10.1177/0265407504045893
- Rook, K. S., August, K. J., & Sorkin, D. H. (2011). Social network functions and health. In R. Contrada & A. Baum (Eds.), *Handbook of stress science: Biology, psychology, and health* (pp. 123–135). New York: Springer.
- Stephens, M. A. P., Hemphill, R. C., Rook, K. S., & Franks, M. M. (2014). It sometimes takes two: Marriage as a mechanism for managing chronic illness. In C. R. Agnew & S. C. South (Eds.), *Interpersonal relationships and health: Social and clinical psychological mechanisms* (pp. 109–132). New York: Oxford University Press.

Social Isolation and Health

Mona Moieni and Naomi I. Eisenberger

Psychology Department, University of California Los Angeles, Los Angeles, CA, USA

I don't know if anyone has noticed, but I only ever write about one thing: being alone. The fear of being alone, the desire to not be alone... the devastation of being left alone. The need to hear the words: You are not alone.

—Shonda Rhimes, television writer and producer

What drives a compelling story? What pushes people to tune in each week to watch a story unfold on their televisions? Shonda Rhimes, an award-winning writer, producer, and creator of multiple widely successful television shows such as *Grey's Anatomy* and *Scandal*, revealed her secret for creating a narrative that millions of people around the world want to follow each week: writing about loneliness. Her answer also tapped into the idea that humans have a “need to belong”—a fundamental need or drive to seek out and maintain social relationships. It has been suggested that this desire to connect with others is as essential to our existence as other basic needs such as food, water, and shelter, providing us with advantages for survival (Baumeister & Leary, 1995).

If being social is such a crucial aspect of being human, then we would expect that not having social relationships would be associated with disadvantages and detrimental outcomes for our health and survival. Indeed, decades of research support the notion that lacking social connections has consequences for physical and mental health, including a significantly higher risk for all-cause mortality. In this chapter, we first briefly discuss measurement issues relevant to social isolation and then highlight health outcomes associated with social isolation. We follow this by a discussion of mechanisms that have been proposed to explain the relationship between social isolation and health, as well as consideration of interventions to reduce social isolation.

Measuring Social Isolation

Social isolation can be conceptualized in many ways. To assess whether an individual is socially isolated, we could ask whether she is living with other people, how often she gets together

with or talks to people, or in how many organizations or social activities (e.g., work, volunteering) she participates. These kinds of measures that tap into an individual's social network size are typically referred to as measures of *objective social isolation*. Thus, someone who lives alone, has few social ties, participates in few social activities, and/or has minimal contact with others would be considered objectively socially isolated.

Rather than asking how many people comprise a person's social network, we could also ask about how satisfied the person is with their network by asking them how connected they feel. We could ask them things like "How often do you feel alone?" or "How often do you feel that your relationships with others are not meaningful?" (Russell, 1996). These subjective feelings of social disconnection, or loneliness, are measures of perceived or *subjective social isolation*. Loneliness is distress resulting from the discrepancy between what an individual wants out of social relationships and what he or she is receiving. For example, people who say they often lack companionship, feel isolated from others, and feel there is no one they can turn to would be considered lonely.

Although these variables are related, they are separable and distinct. For example, it is possible to imagine someone who has only one confidant (and thus may appear socially isolated on objective measures) but feels perfectly content and socially satiated. Conversely, someone may be surrounded by people and participate in numerous social activities but still feel isolated or unsatisfied with these social relationships and thus feel lonely. While it is important to note that social isolation can be measured in numerous ways, this chapter covers the effects of isolation on health regardless of the way it was measured.

Impact of Social Isolation on Health

Starting with Berkman and Syme (1979) and House, Robbins, and Metzner (1982) and continuing through today, decades of work have documented the impact of social isolation on morbidity and mortality. In fact, social isolation is a mortality risk factor comparable with traditional risk factors such as smoking or obesity (see Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015). Interestingly, a recent meta-analysis also found that there was no difference between objective and subjective measures of social isolation in terms of predicting mortality risk (see Holt-Lunstad et al., 2015), suggesting that social isolation is a powerful predictor of mortality regardless of how it is measured.

In addition to mortality, social isolation is related to lower levels of self-rated physical health, risk of cardiovascular disease, elevated blood pressure, risk of poor outcomes post-stroke, metabolic syndrome, increased frequency of doctor visits, and functional decline and disability (see Cacioppo, Grippo, London, Goossens, & Cacioppo, 2015).

There are also many associations between social isolation, in particular subjective social isolation or loneliness, and mental health. Loneliness is associated with depressive symptoms, suicidal ideation, social anxiety, substance abuse, eating disorders, and psychosis. Loneliness is also related to poorer cognitive performance, cognitive decline, and increased risk of Alzheimer's disease (see Hawkey & Cacioppo, 2010).

Potential Mechanisms

Overall, social isolation is associated with a wide array of mental and physical health issues, as well as mortality. Many mechanisms have been proposed to explain the relationship between social isolation and adverse health outcomes. We briefly outline some of the theorized

behavioral, psychological, and biological mechanisms below. Although we discuss these mechanisms separately for ease of presentation, in reality one mechanism is unlikely to fully explain the complex relationships between social isolation and health. These proposed pathways, along with other mechanisms not mentioned here, likely interact with and build off each other.

Behavioral

Health behaviors have been discussed as potential pathways through which social isolation can impact health (see Hawkey & Cacioppo, 2010; Thoits, 2011). For example, friends and family can help individuals start and maintain healthy habits, and they can also provide meaning in life, which can have a positive impact on health behaviors. Socially isolated individuals may thus be at risk for poor health outcomes in part because they are lacking a significant source of modeling of good health behaviors and a sense of purpose related to having close relationships.

Additionally, loneliness may impair executive control and self-regulation (see Hawkey & Cacioppo, 2010), which could have a direct impact on health behaviors such as substance use, physical activity, and eating behavior. Indeed, social isolation is related to smoking and drinking more (Broman, 1993), and there also appears to be a relationship between loneliness and alcohol abuse (Åkerlind & Hörnquist, 1992). In addition to substance use, loneliness is related to obesity. Lonely individuals are more likely to be overweight than non-lonely individuals (Lauder, Mummery, Jones, & Caperchione, 2006). Furthermore, feeling disconnected can also impair self-control over eating behavior (Baumeister, DeWall, Ciarocco, & Twenge, 2005), and loneliness is predictive of reductions in physical activity (Hawkey, Thisted, & Cacioppo, 2009).

Sleep is another behavior that may be a potential mechanism through which social isolation can impact health. Loneliness is related to poor sleep quality throughout the lifespan, and it is also associated with fatigue and low energy (Hawkey, Preacher, & Cacioppo, 2010). Given the importance of sleep for health and mortality, this may be another behavior that may partially explain the social isolation–health association.

Psychological

Many potential psychological mediators of the social ties–health link have also been proposed (see Hawkey & Cacioppo, 2010; Thoits, 2011). One psychological mechanism that has been proposed is maladaptive social cognition. That is, lonely individuals may have certain social cognitive biases, such as generally seeing the world as a more socially threatening place, which may set off a vicious, self-feeding cycle of psychobiological reactions that may ultimately contribute to poor health outcomes. Socially isolated individuals may also lack the sense of purpose or meaning in life that is provided by social connection, which can lead to impairments in psychological well-being and physical health. Those who are isolated may also lack socially supportive others who may “buffer” the impact of stressful life events on well-being and health (Cohen & Wills, 1985).

Besides these mechanisms, some other proposed psychological mediators explaining the impact of social isolation on health include positive affect, social influence, self-efficacy, and self-esteem (see Thoits, 2011). However, some have found that existing models of psychological mechanisms linking social ties to health are generally not supported by evidence, indicating that there is still room for further research to better understand the psychological pathways that may underlie the social isolation–health link.

Biological

Numerous biological pathways have also been proposed to explain the effect of social isolation on health (see Hawkley & Cacioppo, 2010). For example, lonely individuals have immune system impairments (Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985), suggesting that immune alterations may be one pathway through which loneliness operates to damage health.

Inflammation, the body's first line of defense against infection or injury, is one potential immunological process through which loneliness may exert its effects. Inflammatory processes are implicated in a wide array of physical and mental health conditions, as well as mortality (Emerging Risk Factors Collaboration, 2010). Lonely individuals have increased pro-inflammatory reactivity (Jaremka et al., 2013), as well as upregulated pro-inflammatory gene expression (Cole et al., 2007). This heightened inflammatory activity in lonely individuals indicates that inflammation may be mediating some of the effects of social isolation on health. Interestingly, inflammation can also lead to feelings of social disconnection (Eisenberger, Inagaki, Mashal, & Irwin, 2010; Moieni et al., 2015), suggesting that inflammation and loneliness may engage in a dangerous, mutually reinforcing cycle.

Related to inflammation, loneliness may also operate through neuroendocrine pathways. For example, lonely individuals have impairments related to the hypothalamus–pituitary–adrenal (HPA) axis, including increased levels of cortisol (Adam, Hawkley, Kudielka, & Cacioppo, 2006). Chronic social isolation may also lead to glucocorticoid insensitivity (see Hawkley & Cacioppo, 2010), inhibiting the ability of cortisol to have an anti-inflammatory effect.

Finally, neural mechanisms have also been proposed to mediate the social isolation–health link. A set of neural regions which are involved in threat detection, including the amygdala, dorsal anterior cingulate cortex (dACC), anterior insula, and periaqueductal gray, are thought to play a role in the relationship between social isolation and health (Eisenberger & Cole, 2012). These regions may serve as a neural alarm system to detect social threat (e.g., social isolation, social exclusion), and in turn, activation of these regions may have an impact on HPA and sympathetic nervous system activity, ultimately impacting health. For example, social exclusion or rejection activates the dACC and anterior insula (Eisenberger, 2015; Eisenberger, Lieberman, & Williams, 2003). Activation of these neural regions is related to both heightened inflammation, which is involved in many physical and health disorders, and cortisol, a hormone involved in the body's stress response.

While the existing evidence lends support to these particular biological mechanisms, more work is needed to flesh out the details of these pathways, as well as how they may interact with behavioral and psychological processes, to impact health.

Interventions

Given the influence of social isolation on health, it is not surprising that multiple types of interventions targeted at reducing social isolation have been conducted. Interventions have included offering social skills training, social cognitive training, mind–body interventions, opportunities for social contact or activities, or social support programs. The mode of delivery has ranged from group interventions to one-on-one interventions, from in person to online, and lasting from a few weeks to multiple years. There have been multiple reviews on this topic (e.g., Dickens, Richards, Greaves, & Campbell, 2011; Masi, Chen, Hawkley, & Cacioppo, 2010), so we will highlight a few examples of effective interventions that have been tested using randomized trials.

One type of intervention that has shown positive effects on social isolation are those promoting social support and social activities. In one study, older adults who were randomly assigned to an intervention intended to increase peer support and social activity showed decreases in social isolation (Routasalo, Tilvis, Kautiainen, & Pitkala, 2009); those in the intervention group became more socially active and found new friends significantly more often than the control group. However, the intervention did not lead to decreases in loneliness or increases in social network size. Other interventions have examined the effect of animal-assisted therapy to increase socialization and decrease loneliness (Banks & Banks, 2002; Banks, Willoughby, & Banks, 2008). Participants who were randomly assigned to the animal-assisted therapy group (i.e., regular visits from a dog) showed decreases in loneliness (Banks & Banks, 2002).

Another type of intervention that has shown promise for reducing loneliness is social cognitive training (Masi et al., 2010). For example, one study examined the effect of “reminiscence therapy” (vs. a wait-list control) on loneliness in nursing home patients (Chiang et al., 2010). Over the course of 8 weeks, those in the therapy condition participated in activities such as discussing how to be more aware and expressive of feelings, identifying positive social relationships, and recalling family history and life stories; those in this condition showed improvements in loneliness. Although these interventions have shown promise in reducing feelings of isolation, additional studies examining the effect of this type of intervention, particularly in different samples (e.g., isolated adolescents), are still needed.

Creswell et al. (2012) took yet another approach, examining the effect of a mindfulness-based stress reduction (MBSR) training on loneliness in older adults. They found that an 8-week MBSR program, compared with a wait-list control, led to reductions in loneliness, as well as reductions in pro-inflammatory gene expression. This finding suggests that other mind–body interventions may be successful in reducing loneliness, as well as biological mechanisms such as inflammation that may be driving some of the health effects associated with social isolation.

Indeed, there is certainly room to develop additional targeted interventions addressing social isolation based on these kinds of successful interventions. Some have suggested that interventions that incorporate some sort of social cognitive training are likely to be the most effective (Masi et al., 2010), while others have found that interventions with a social activity or social support component, and delivered at a group level (compared with one-on-one), may be the most effective (Dickens et al., 2011). Still others have suggested tailoring new interventions for particular developmental stages and thus focusing interventions on aspects of social isolation that are most important across different times of the lifespan (Qualter et al., 2015). Overall, it appears that testing new evidence-based interventions, particularly using randomized trials, is an important area for future research in this field.

Conclusion

In summary, social isolation has detrimental effects on a wide range of physical and mental health problems, as well as mortality. Numerous mechanisms have been proposed for the relationship between social isolation and health, but future work is needed to more fully flesh out these pathways. Finally, some interventions aimed at decreasing social isolation have been successful. However, given that social isolation can have such a powerful influence on our physical health and well-being, developing new low-cost, effective, and easy-to-implement interventions is an important area for further research that could ultimately have significant public health implications.

Author Biographies

Mona Moieni is a postdoctoral scholar in the Department of Psychology at the University of California, Los Angeles (UCLA). Her research focuses on the relationships between biological and social psychological processes, as well as how these relationships may be relevant for physical and mental health.

Naomi I. Eisenberger is an associate professor of psychology at the University of California, Los Angeles (UCLA). She specializes in social neurocognitive aspects of emotion and behavior and how these processes relate to health-relevant biomarkers.

References

- Adam, E. K., Hawkey, L. C., Kudielka, B. M., & Cacioppo, J. T. (2006). Day-to-day dynamics of experience–cortisol associations in a population-based sample of older adults. *Proceedings of the National Academy of Sciences*, 103(45), 17058–17063. doi:10.1073/pnas.0605053103
- Åkerlind, I., & Hörnquist, J. O. (1992). Loneliness and alcohol abuse: A review of evidences of an interplay. *Social Science & Medicine*, 34(4), 405–414. doi:10.1016/0277-9536(92)90300-F
- Banks, M. R., & Banks, W. A. (2002). The effects of animal-assisted therapy on loneliness in an elderly population in long-term care facilities. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 57(7), M428–M432. doi:10.1093/gerona/57.7.M428
- Banks, M. R., Willoughby, L. M., & Banks, W. A. (2008). Animal-assisted therapy and loneliness in nursing homes: Use of robotic versus living dogs. *Journal of the American Medical Directors Association*, 9(3), 173–177. doi:10.1016/j.jamda.2007.11.007
- Baumeister, R. F., DeWall, C. N., Ciarocco, N. J., & Twenge, J. M. (2005). Social exclusion impairs self-regulation. *Journal of Personality and Social Psychology*, 88(4), 589. doi:10.1037/0022-3514.88.4.589
- Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, 117(3), 497. doi:10.1037/0033-2909.117.3.497
- Berkman, L. F., & Syme, S. L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology*, 109(2), 186–204.
- Broman, C. L. (1993). Social relationships and health-related behavior. *Journal of Behavioral Medicine*, 16(4), 335–350. doi:10.1007/BF00844776
- Cacioppo, S., Grippo, A. J., London, S., Goossens, L., & Cacioppo, J. T. (2015). Loneliness clinical import and interventions. *Perspectives on Psychological Science*, 10(2), 238–249. doi:10.1177/1745691615570616
- Chiang, K.-J., Chu, H., Chang, H.-J., Chung, M.-H., Chen, C.-H., Chiou, H.-Y., & Chou, K.-R. (2010). The effects of reminiscence therapy on psychological well-being, depression, and loneliness among the institutionalized aged. *International Journal of Geriatric Psychiatry*, 25(4), 380. doi:10.1002/gps.2350
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98(2), 310. doi:10.1037/0033-2909.98.2.310
- Cole, S. W., Hawkey, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in human leukocytes. *Genome Biology*, 8(9), R189. doi:10.1186/gb-2007-8-9-r189
- Creswell, J. D., Irwin, M. R., Burkland, L. J., Lieberman, M. D., Arevalo, J. M., Ma, J., ... Cole, S. W. (2012). Mindfulness-based stress reduction training reduces loneliness and pro-inflammatory gene expression in older adults: A small randomized controlled trial. *Brain, Behavior, and Immunity*, 26(7), 1095–1101. doi:10.1016/j.bbi.2012.07.006

- Dickens, A. P., Richards, S. H., Greaves, C. J., & Campbell, J. L. (2011). Interventions targeting social isolation in older people: A systematic review. *BMC Public Health*, 11(1), 647. doi:10.1186/1471-2458-11-647
- Eisenberger, N. I. (2015). Meta-analytic evidence for the role of the anterior cingulate cortex in social pain. *Social Cognitive and Affective Neuroscience*, 10(1), 1–2. doi:10.1093/scan/nsu120
- Eisenberger, N. I., & Cole, S. W. (2012). Social neuroscience and health: Neurophysiological mechanisms linking social ties with physical health. *Nature Neuroscience*, 15(5), 669–674. doi:10.1038/nn.3086
- Eisenberger, N. I., Inagaki, T. K., Mashal, N. M., & Irwin, M. R. (2010). Inflammation and social experience: An inflammatory challenge induces feelings of social disconnection in addition to depressed mood. *Brain, Behavior, and Immunity*, 24(4), 558–563. doi:10.1016/j.bbi.2009.12.009
- Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, 302(5643), 290–292. doi:10.1126/science.1089134
- Emerging Risk Factors Collaboration. (2010). C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: An individual participant meta-analysis. *The Lancet*, 375(9709), 132–140. doi:10.1016/S0140-6736(09)61717-7
- Glaser, R., Kiecolt-Glaser, J. K., Speicher, C. E., & Holliday, J. E. (1985). Stress, loneliness, and changes in herpesvirus latency. *Journal of Behavioral Medicine*, 8(3), 249–260. doi:10.1007/BF00870312
- Hawkey, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine*, 40(2), 218–227. doi:10.1007/s12160-010-9210-8
- Hawkey, L. C., Preacher, K. J., & Cacioppo, J. T. (2010). Loneliness impairs daytime functioning but not sleep duration. *Health Psychology*, 29(2), 124. doi:10.1037/a0018646
- Hawkey, L. C., Thisted, R. A., & Cacioppo, J. T. (2009). Loneliness predicts reduced physical activity: Cross-sectional & longitudinal analyses. *Health Psychology*, 28(3), 354. doi:10.1037/a0014400
- Holt-Lunstad, J., Smith, T. B., Baker, M., Harris, T., & Stephenson, D. (2015). Loneliness and social isolation as risk factors for mortality a meta-analytic review. *Perspectives on Psychological Science*, 10(2), 227–237. doi:10.1177/1745691614568352
- House, J. S., Robbins, C., & Metzner, H. L. (1982). The association of social relationships and activities with mortality: Prospective evidence from the Tecumseh Community Health Study. *American Journal of Epidemiology*, 116(1), 123–140.
- Jaremka, L. M., Fagundes, C. P., Peng, J., Bennett, J. M., Glaser, R., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2013). Loneliness promotes inflammation during acute stress. *Psychological Science*, 24(7), 1089–1097. doi:10.1177/0956797612464059
- Lauder, W., Mummary, K., Jones, M., & Caperchione, C. (2006). A comparison of health behaviours in lonely and non-lonely populations. *Psychology, Health & Medicine*, 11(2), 233–245. doi:10.1080/13548500500266607
- Masi, C. M., Chen, H.-Y., Hawkey, L. C., & Cacioppo, J. T. (2010). A meta-analysis of interventions to reduce loneliness. *Personality and Social Psychology Review*. doi:10.1177/1088868310377394
- Moieni, M., Irwin, M. R., Jevtic, I., Olmstead, R., Breen, E. C., & Eisenberger, N. I. (2015). Sex differences in depressive and socioemotional responses to an inflammatory challenge: Implications for sex differences in depression. *Neuropsychopharmacology*, 40(7), 1709–1716. doi:10.1038/npp.2015.17
- Qualter, P., Vanhalst, J., Harris, R., Van Roekel, E., Lodder, G., Bangee, M., ... Verhagen, M. (2015). Loneliness across the life span. *Perspectives on Psychological Science*, 10(2), 250–264. doi:10.1177/1745691615568999
- Routasalo, P. E., Tilvis, R. S., Kautiainen, H., & Pitkala, K. H. (2009). Effects of psychosocial group rehabilitation on social functioning, loneliness and well-being of lonely, older people: Randomized controlled trial. *Journal of Advanced Nursing*, 65(2), 297–305. doi:10.1111/j.1365-2648.2008.04837.x
- Russell, D. W. (1996). UCLA loneliness scale (version 3): Reliability, validity, and factor structure. *Journal of Personality Assessment*, 66(1), 20–40. doi:10.1207/s15327752jpa6601_2

Thoits, P. A. (2011). Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior*, 52(2), 145–161. doi:10.1177/0022146510395592

Suggested Reading

Cacioppo, S., Grippo, A. J., London, S., Goossens, L., & Cacioppo, J. T. (2015). Loneliness clinical import and interventions. *Perspectives on Psychological Science*, 10(2), 238–249. doi:10.1177/1745691615570616

Eisenberger, N. I., & Cole, S. W. (2012). Social neuroscience and health: Neurophysiological mechanisms linking social ties with physical health. *Nature Neuroscience*, 15(5), 669–674. doi:10.1038/nn.3086

Hawkey, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine*, 40(2), 218–227. doi:10.1007/s12160-010-9210-8

Holt-Lunstad, J., Smith, T. B., Baker, M., Harris, T., & Stephenson, D. (2015). Loneliness and social isolation as risk factors for mortality a meta-analytic review. *Perspectives on Psychological Science*, 10(2), 227–237. doi:10.1177/1745691614568352

Thoits, P. A. (2011). Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior*, 52(2), 145–161. doi:10.1177/0022146510395592

Social Support and Health

Marci E. J. Gleason and Jerica X. Bornstein

*Department of Human Development and Family Sciences, University of
Texas at Austin, Austin, TX, USA*

Social support has been consistently shown to be beneficial for both physiological and psychological health. A meta-analysis of 148 studies investigating the influence of social relationships on mortality found that social support was highly protective (Holt-Lunstad, Smith, & Layton, 2010). Those who reported low levels of social support had an approximately 50% increase in mortality risk compared with those high in social support. These findings investigated individuals across the lifespan (ages ranged from 6 to 92) and were robust regardless of the sex or initial health status of the participants. However, as with most findings, the effect of social support on health is complicated and can range from very positive to neutral and even become negative depending on how social support is measured.

Social support has been measured in a myriad of ways and studied in various disciplines across multiple decades. Perhaps the earliest work that identified a type of social support as beneficial to health was Durkheim's (1897/1951) report that suicide risk was reduced among those who were socially integrated. He argued that individuals who were more embedded in social networks were more tied into society and were therefore less likely to kill themselves. He cited the higher suicide rate among single compared with married individuals as one example of the power of social ties. In line with this research, many studies of social support have focused on the size of individuals' social networks and the presence and number of close friends and family members in an individual's life, often referred to as "close ties." The size of an individual's social network is determined by a count of all the people that an individual typically interacts with and can include everyone from a spouse to the mailman. Although social network size has been linked to beneficial health outcomes, those linkages become stronger when one refines this measurement to include information on the strength of the ties between the focal individual and their social network (Holt-Lunstad et al., 2010). Researchers examining "close ties" gather information on the number of people in an individual's social network and will often measure the frequency of interaction with network members, as well as the depth and closeness of those relationships. Importantly, the quality of an individual's

relationships is a key enhancer of the gains associated with network ties: having close friends is more protective than having many friends.

The use of network ties as a proxy for social support has become less prevalent in the research literature and replaced with the idea that social support is composed of specific support behaviors, such as giving advice, listening, and providing monetary aid. These behaviors could be ones that have occurred within an individual's social relationship in the past or that the individual believes would be available to them in the future. This characterization of social support is often referred to as *perceived social support* or the *availability of social support*. Individuals who report high levels of perceived support, regardless of the number of actual social ties individuals have, report better mental and physical health.

Thus the benefits of social support and more specifically perceived social support are well documented, and several mechanisms to explain the association between social support and health have been posited. The dominant explanation is the stress-buffering hypothesis that contends that social support protects recipients from the negative effects of stress by helping the recipient cope more effectively with that stress—essentially providing extra resources for coping through practical means, such as lending money, or emotional means, by listening or offering comfort. Although numerous studies have shown that the effect of stress on health is buffered by perceived support, few studies have demonstrated that this buffering is the only or even the primary way in which perceived support is protective. In other words, it is not only that those who are high in perceived support seem to be less affected by the stress in their lives but also that regardless of stress level, those high in perceived support report better mental and physical health.

Such findings have led others to argue that support, specifically perceived support, reflects an individual difference variable or personality trait. Levels of perceived support are not strongly related to the number of specific support events individuals report receiving, suggesting that people differ in the amount they believe people will be there for them regardless of their actual support experiences. However, studies have failed to find evidence that perceived support operates as a personality characteristic. Instead, research on the source of perceived support has demonstrated that specific relationships create a sense of supportiveness, rather than certain individuals having a tendency to view others as supportive. This idea is explored in relational regulation theory (RRT) that posits that the extent to which someone perceives support is dependent on interactions with individuals that are specific to that particular recipient (Lakey & Orehek, 2011). A sense of perceived support arises when an individual has access to supportive relationships, and that sense of perceived support will not exist in the absence of those specific relationships. Unlike a personality trait that would be fairly consistent across time, RRT predicts that one's sense of perceived support will vary depending on one's current social context and relationships.

These findings suggest that regardless of how social support promotes positive health and how the perception of social support is formed, increasing social support in individuals' lives should lead to both mental and physical health benefits. However, there is a clear and established paradox in the social support literature. The perception of social support is positive, but when individual instances of support are investigated, receiving support is often associated with *negative* outcomes including emotional distress, poorer physical health, and increased mortality risk. This link between single instances of social support or enacted support and negative outcomes has been demonstrated in observational, experimental, and correlational studies. Work in the helping behavior literature corroborates this finding; being helped is associated with decreased self-esteem and increased depressed mood in recipients. Moreover, these negative outcomes are more pronounced when help is received from a close other such as a relationship partner.

Social support researchers have suggested many reasons for why enacted support is often associated with negative outcomes. Perhaps the explanation that has received the most attention both in the social support and helping behavior literature is that the receipt of support may undermine the recipient's sense of efficacy and/or control. Receiving support itself may signal to the recipient that the provider perceives them to be struggling or even incapable of coping with the stressor, therefore exacerbating the recipient's anxiety rather than alleviating it (Bolger & Amarel, 2007). Despite this general tendency for an instance of social support to be ineffective, research on enacted social support has identified specific types of support, referred to as skillful support, that are often associated with positive outcomes. Four types of skillful support—matched support, invisible support, supportive reciprocity, and capitalization—have been well documented in the literature, and we briefly describe these below.

Stressors can be categorized as controllable or uncontrollable, and this distinction is important for determining what type of enacted support is likely to promote positive health outcomes for the recipient. Controllable stressors are likely to benefit from informational support. For example, an individual who is trying to obtain a new passport is likely to find it helpful when a friend who recently renewed a passport tells them the various steps needed to obtain one. Alternatively, uncontrollable stressors, such as the stress associated with waiting to find out whether one has been promoted, are more likely to be alleviated by a friend providing a distraction or reassurance that the recipient is worthy of the promotion. Unfortunately, studies of support matching suggest that people struggle to identify the types of support their relationship partners actually desire. This results in high levels of unmatched support (Cutrona, 1996), rendering those support attempts ineffective and even detrimental to the receiver.

Overly visible or directive support has also been linked to negative outcomes, perhaps because these types of overt support decrease recipients' sense of control and increase their feelings of indebtedness to their providers as well as draw attention to the struggle an individual is undergoing. For instance, in an experimental study, participants were asked to give a difficult and impromptu speech. Those individuals who reported that they were given advice from a confederate during this task reported higher levels of anxiety than those who heard the same advice, but did not think it was directed at them (Bolger & Amarel, 2007). This latter type of support, known as invisible support, has been shown to ameliorate daily anxiety, increase goal attainment, and increase feelings of self-efficacy in the recipient.

Findings from studies examining equity and reciprocity in support, as well as ones examining the effects of provision on the provider, indicate that the costs of receiving support can also be lessened when there is an opportunity to reciprocate by providing support to one's partner. Interestingly, the provision of social support has generally been found to be highly beneficial to the provider and has been associated with positive outcomes. Further, whereas most of the literature addressing partner support focuses on support provided in response to stressors or hardships, recent research has demonstrated the importance of examining support provided in response to positive events or uplifts. Sharing positive experiences in the hope of getting a receptive, supportive audience is called a capitalization attempt. When the support that follows such attempts (i.e., capitalization support) is both active and positive, it yields strong personal and relational benefits (Gable, Gosnell, Maisel, & Strachman, 2012).

The field continues to explore other ways in which support can be both received and perceived positively. One promising approach is that of affectionate and physical touch in adult close relationships, which has been shown to help promote high quality relationships, physical well-being, and psychological outcomes in adulthood (Jakubiak & Feeney, 2016). Recent research has also suggested that the link between actual interactions and perceived support may come from common everyday conversation rather than actual support attempts. Perhaps

it is those we can speak comfortably and freely with about everyday matters, regardless of whether those individuals have actually provided support, that create a sense of perceived support in individuals (Lahey & Orehek, 2011).

The majority of the current social support literature has focused on in person interactions. However, the ways with which we communicate are changing rapidly, and many individuals now communicate with their closest social ties in part or even largely through email, text, and social networks. Physical proximity to our social networks may become both less common and potentially less important as online communication provides a new avenue through which to receive support. In addition, social networking platforms allow individuals to routinely request and receive support not only from current social network members but also from people they don't know outside of on online communication. Such communication has the potential to be positive: research has shown that those who engage in more supportive interactions on social networking sites have greater levels of positive affect (Oh, Ozkaya, & LaRose, 2014). However similar to earlier work on social networks, the quality of online support interactions matters. As society's communication habits and norms shift, so will the experience of social support. Whether the current shift to online communication creates an opportunity for individuals to gain access to better and more frequent supportive interactions remains to be seen. However, it is clear that social support researchers will have to address the implications of the online world on mental and physical well-being.

Social support has been repeatedly and strongly linked to positive health outcomes, but exactly why social support is beneficial is still being explored. In particular, the paradox that while perceived support is almost universally positive, enacted support is often negative has complicated the field's understanding of social support processes. Researchers continue to explore new mechanisms and types of social support and continue to gain new insights into the importance of social ties to individuals' health outcomes.

Author Biographies

Marci E. J. Gleason is an associate professor at the University of Texas at Austin in the Department of Human Development and Family Sciences. She received her PhD in social psychology at New York University. Her research focuses on how individuals cope with life transitions and stressors in the context of their social relationships.

Jerica X. Bornstein is a graduate student at the University of Texas at Austin in the Department of Human Development and Family Sciences. She is particularly interested in how various types of support exchanges within close relationships may help or hinder health behavior change and health outcomes.

References

- Bolger, N., & Amarel, D. (2007). Effects of social support visibility on adjustment to stress: Experimental evidence. *Journal of Personality and Social Psychology*, 92, 458–475. doi:10.1037/0022-3514.92.3.458
- Cutrona, C. E. (1996). *Social support in couples: Marriage as a resource in times of stress*. Thousand Oaks, CA: Sage Publications, Inc.
- Durkheim, E. (1951). *Suicide: A study in sociology* (JA Spaulding & G. Simpson, trans.). Glencoe, IL: Free Press. (Original work published 1897).

- Gable, S. L., Gosnell, C. L., Maisel, N. C., & Strachman, A. (2012). Safely testing the alarm: Close others' responses to personal positive events. *Journal of Personality and Social Psychology*, 103, 963–981. doi:10.1037/a0029488
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7, e1000316.
- Jakubiak, B. K., & Feeney, B. C. (2016). Affectionate touch to promote relational, psychological, and physical well-being in adulthood: A theoretical model and review of the research. *Personality and Social Psychology Review*, 21(3), 228–252. doi:10.1177/1088868316650307
- Lakey, B., & Orehek, E. (2011). Relational regulation theory: A new approach to explain the link between perceived social support and mental health. *Psychological Review*, 118, 482–495. doi:10.1037/a0023477
- Oh, H. J., Ozkaya, E., & LaRose, R. (2014). How does online social networking enhance life satisfaction? The relationships among online supportive interaction, affect, perceived social support, sense of community, and life satisfaction. *Computers in Human Behavior*, 30, 69–78.

Suggested Reading

- Gleason, M. E. J., & Iida, M. (2015). Social support. In M. Mikulincer, P. R. Shaver, J. A. Simpson, & J. F. Dovidio (Eds.), *APA handbook of personality and social psychology, Volume 3: Interpersonal relations* (pp. 351–370). Washington, DC: American Psychological Association.
- Kiecolt-Glaser, J. K., & Wilson, S. J. (2017). Lovesick: How couples' relationships influence health. *Annual Review of Clinical Psychology*, 13(1), 421–443.
- Uchino, B. N. (2009). Understanding the links between social support and physical health: A lifespan perspective with emphasis on the separability of perceived and received support. *Perspectives on Psychological Science*, 4, 236–255. doi:10.1111/j.1745-6924.2009.01122

Spirituality/Religiosity and Health

Kevin S. Masters and Stephanie A. Hooker

*Psychology Department, University of Colorado at Denver –
Anschutz Medical Campus, Aurora, CO, USA*

Religiosity and spirituality (R/S) are important to individuals worldwide. It is estimated that 80% of people identify with a religious group and many who are not members of a religious faith still hold to some religious or spiritual teaching. About 60% of Americans report that they regularly feel a deep sense of “spiritual peace and well-being” (Pew Research Center for Religion in Public Life, 2014).

An important issue that confronts empirical investigators is defining religiosity and spirituality and distinguishing between them. This has proven to be a difficult task and one that continues to evolve. Closely related is the problem of measurement: How is one to measure constructs that prove so elusive to consensual definition? Kapuscinski and Masters (2010) recently discussed these issues in detail and provided historical context. They also reviewed measures of spirituality. Though some individuals find their spirituality and religiosity to be inextricably intertwined, the more recent trend is to view them as related, or overlapping, but differentiated concepts. Both share a sense of being concerned with understanding and experiencing the sacred or the ultimate. However, religiosity undertakes this search within the context of an institution or group, whereas spirituality is viewed as a more personal journey, which may or may not take place within an organized religion. Other connotative differences found in the literature include the relative importance of codes of conduct (higher in religion) and the issue of externality (religion) versus internality (spirituality). There also seems to be an inclination among some social scientists to view religion as more likely “bad” and spirituality as more likely “good.” Scholars in the area, however, warn against this use, noting that most of the research to date has been done on religious constructs, and both religiosity and spirituality are complex and multidimensional.

Individuals who have a sense of R/S often believe that their spirituality influences their health as a result of the peace or well-being that their faith affords, religiously based behavioral practices (e.g., refraining from smoking, moderate use of alcohol, vegetarianism based on religious beliefs), or divine intervention. There is reasonably strong empirical evidence, some of it at the population level and other evidence from meta-analyses, generally suggesting a modest positive relationship between religiosity and health with the suggestion, not yet confirmed, of a beneficial effect of religiosity on health. The most studied variable, with the most consistently positive

findings, is religious service attendance. Attendance has been found, after controlling for relevant confounds, to predict a number of beneficial outcomes including decreased mortality (Chida, Steptoe, & Powell, 2009). Research on health and spirituality per se is only beginning. (Spiritually relevant practices such as meditation or yoga have their own bodies of literature.)

We refer those interested in comprehensive reviews of the empirical literature investigating the relationships between R/S and various health outcomes to Koenig, King, and Carson (2012). Park et al. (2017) provide a more recent review with specific emphasis on cardiovascular disease, cancer, and substance abuse. As important as these general findings are, however, the question posed to contemporary investigators is this: What aspects of R/S are related to what aspects of health for what types of people? A sophisticated literature at this level of specificity does not yet exist but is clearly an important direction for future work. One aspect of work aimed at greater specificity and understanding, currently underway, concerns investigation of mechanisms or processes that may account for relationships between aspects of R/S and health indicators. We now turn our attention to this literature.

Mechanisms Linking R/S to Health

R/S likely influences health through a number of different mechanisms, and given the multidimensional nature of R/S, different facets of R/S may influence these mechanisms differently. Three primary mechanisms discussed by empirical investigators include social support, health behaviors, and psychological processes. We will review the literature in these areas but openly acknowledge that there are likely other plausible mechanisms.

Social Support

One of the primary hypothesized mechanisms linking R/S to health is social support. Ample evidence has shown that social support is beneficial for a number of health outcomes. This mechanism is primarily found in the context of a religious community and, to this point, has not been viewed as a strong mechanism linking spirituality to health. For instance, those who regularly attend religious services have larger social networks, are more likely to stay in contact with other people, report greater perceived social support, are more likely to feel valued by their social group, tend to believe their religious congregations are more cohesive, and are more likely to stay married.

Interestingly, religious support has also been shown to be related to another hypothesized mediator of the relationship between R/S and health: health behavior. That is, individuals who feel more support from their congregation are more likely to engage in health-promoting behaviors such as better diet and increased physical activity. However, those who do not perceive this support or have negative interactions with fellow parishioners may engage in less healthy behavioral practices. Though social support may be important in terms of understanding the relationship between R/S and health, it does not account for a large portion of the variance between the two. Consequently, other factors have also been hypothesized to mediate the R/S and health relationship.

Health Behaviors

As noted previously, many religious teachings have health implications; therefore, health behaviors have been hypothesized as a mechanism linking R/S to health. Certainly, religious

leaders have great potential for influencing the healthy behaviors of their congregations. Given the multiple pathways by which R/S could influence health-promoting and health-compromising behaviors, it is not surprising that there are many studies linking R/S to health behaviors. Religious service attendance is an often-studied variable that is related to a number of healthy behaviors and less engagement in health-compromising behaviors. There is, however, also evidence that religious importance is associated with greater use of preventive healthcare services, including mammograms, flu shots, cholesterol screening, and prostate cancer screening (Benamins & Brown, 2004). Additionally, R/S variables, including religious involvement, religious service attendance, personal spirituality, and religious community support, are associated with a healthy lifestyle or engaging in many different health-relevant behaviors (Hooker, Masters, & Carey, 2014). In general, positive R/S variables are related to increased engagement in health-promoting behaviors and reduced likelihood of engaging in health-compromising behaviors, whereas negative R/S variables are related to reduced engagement in health-promoting behaviors and increased engagement in health-compromising behaviors.

In chronic illness patient populations, R/S may be related to treatment adherence. One study of patients with congestive heart failure (CHF) demonstrated that religious commitment predicts greater adherence to CHF-specific behaviors over 6 months even when controlling for initial levels of adherence (Park, Moehl, Fenster, Suresh, & Bliss, 2008). In cancer survivors, daily spiritual experiences and spiritual well-being are related to health-promoting behaviors, including eating five or more servings of fruits and vegetables a day, engaging in moderate to vigorous physical exercise, adhering to doctors' orders, and making overall positive changes in behavior (e.g., avoiding alcohol, cigarettes, and sun exposure, using sunscreen and sun-protective clothing; Park, Edmondson, Hale-Smith, & Blank, 2009). Conversely, religious struggles are related to less adherence to doctor's advice and less medication adherence (Park, Edmondson, Hale-Smith, & Blank, 2009). These studies suggest that R/S variables also influence health behaviors in the context of a chronic illness.

Psychological Processes

In addition to social support and health behaviors, a variety of psychological processes, including stress reduction and coping, reduced depressive symptoms, and enhanced self-regulation, have been proposed to mediate the relationship between R/S and health.

R/S may be a buffer against the effects of stressful life events. Religious coping, in particular, has been studied as a protective factor for coping with life stressors. Religious coping behaviors may be thought of as positive (e.g., religious purification or forgiveness, collaborative religious coping) and negative strategies (e.g., spiritual discontent, viewing God as punishing) that individuals use to mitigate the effects of stress. One meta-analysis has shown that positive religious coping behaviors are positively related to better psychological adjustment (e.g., optimism, happiness, hope, life satisfaction) and negatively related to negative psychological adjustment (e.g., depression, anxiety, burden, distress), whereas negative religious coping behaviors are positively related to negative psychological adjustment (Ano & Vasconcelles, 2005). Moreover, negative religious coping is related to greater perceived stress and to less use of problem-focused coping strategies, which are related to less perceived stress. Recent experimental studies have begun to examine the effects of religious behaviors, such as prayer, on the effects of cardiovascular reactivity to stress. In an experimental study in our lab, we found that devotional prayer reduced stress reactivity to a provocative religious interview in a group of self-identified Christians; however, more work is needed to understand the pathways between religious behavior and stress reduction.

Another psychological process linking R/S and health may be a reduction in depressive symptoms. R/S involvement is shown to be positively related to mental health. One very large meta-analysis of 147 studies shows that R/S variables are negatively correlated with depressive symptoms (Smith, McCullough, & Poll, 2003). Interestingly, prayer has been shown to be related to fewer depressive symptoms and less distress in patients postcoronary artery bypass graft (Ai et al., 2006). Conversely, religious struggles and negative religious coping are related to increases in depressive symptoms in patients with congestive heart failure (Park, Brooks, & Sussman, 2009). In general, positive R/S variables are protective against depressive symptoms, whereas negative R/S variables are related to greater depressive symptoms.

A new and particularly interesting hypothesis is that R/S may be related to health through enhanced self-regulation. McCullough and Willoughby (2009) offer a number of testable hypotheses in this area that are substantive and potentially important in guiding future research. Deciphering temporal precedence and cause and effect relationships will likely prove challenging. It may be that individuals who are better self-regulated are more likely to engage in regular behaviors (such as attending services and engaging in healthy behaviors) but are also better at controlling their emotions and coping with stress. However, it is also likely that the doctrines, messages, and practices of religious faiths reinforce and normalize self-regulatory behavior and provide social support for its maintenance. More work is needed in this area, but we find this mechanism to be a particularly exciting advancement of this field.

Special Topics in R/S and Health Research

Distant Intercessory Prayer

A topic that, perhaps surprisingly, has garnered significant attention in both the popular and scientific press is the possibility that distant intercessory prayer (i.e., prayer said on another's behalf and without the intended beneficiary present) can bring about healing or relief of symptoms. There is no question that religious individuals, and many nonreligious individuals, are more prone to pray both for themselves and others when there is an immediate health concern, and those prayers are often for healing or relief of disabling symptoms. The topic of prayer and health is important and worthy of investigation on its own. The focus of this section of our entry, however, is on the potential effects of distant intercessory prayer.

In a typical study on this topic, investigators recruit "intercessors" who will pray for a group of patients. In almost all studies, the patients did not know whether they were being prayed for or not and in some cases did not know that they were even in a research study. Some early reports suggested beneficial effects for those who were prayed for versus a control group that did not receive prayer, but a careful analysis of these studies suggests that failure to consider multiple comparisons and other concerns render the results less compelling. Subsequently, Masters, Spielmans, and Goodson (2006) conducted a meta-analysis and concluded that there was no evidence to suggest any beneficial effects for those who received prayer versus non-prayed-for controls. A subsequent Cochrane review (Roberts, Ahmed, & Davison, 2009) reached a similar conclusion, and both reviews recommended that further research in this area not be supported largely because of a serious methodological impediment that cannot be overcome. Regardless of whatever one may think about distant intercessory prayer per se, there is simply no way to conduct a meaningful controlled investigation because there is no practical (or ethical) means to ensure that the control group does not receive prayers of its own from family, friends, clergy, or congregations, and there is no plausible

rationale to suggest that the prayers of unknown intercessors would somehow have greater efficacy than the prayers of those who know the patient. In this regard, it is interesting to note that in one of these studies, one of three experimental groups knew that they were receiving prayer from study intercessors. This group demonstrated a higher incidence of complications than did two other groups who did not know whether they were receiving prayer (one group was, the other was not).

Spirituality in Cancer Patients and Survivors

Research in R/S and health, and in particular spirituality and health, has been embraced within the cancer literature, and there has been a surge of findings in this area in recent years. Indeed, one analysis of the literature shows that the number of abstracts regarding R/S and cancer have increased more than 2.5 times over previous decades (Salsman, Fitchett, Merluzzi, Sherman, & Park, 2015). A series of recent meta-analyses (Jim et al., 2015; Salsman, Pustejovsky, et al., 2015; Sherman et al., 2015) show that across 44,000 cancer patients, R/S dimensions are positively related to physical, mental, and social health. The strongest relationships are seen in affective dimensions of R/S, or the emotional experience of R/S including “a sense of transcendence, meaning, purpose, or connection larger than oneself” (Salsman, Fitchett, et al., 2015, p. 3). There are small associations for cognitive (e.g., R/S beliefs, religious orientation, R/S problem solving) and other dimensions of R/S (e.g., composite indexes, social support, religious affiliation) in relation to all three dimensions of health. Behavioral factors, including R/S coping and public and private R/S activities, demonstrated a small, positive relationship with social health but were not significantly related to physical or mental health. This literature could be greatly enhanced with studies that are more rigorous in their design (e.g., less reliance on cross-sectional data) and attend in greater detail to the possible moderators and mediators of these relationships (Park et al., 2017).

Future Directions

The topic of R/S and health has experienced significant growth over the past two decades, resulting in greater understanding of the relationships and implications of what the next steps should be. Clearly, much more needs to be done. We offer suggestions for future research in this area and refer interested readers to Park et al. (2017) for a more detailed discussion.

As noted above, future work that aims for greater specificity is needed, for example, to specify the particular aspects of R/S that are beneficial, the particular components of health most likely to be associated with R/S, the relevant theoretical mechanisms, and the particular groups of people most likely to benefit. This work must consider both the socioeconomic and cultural context in which R/S takes place, as well as the aspects of R/S that themselves constitute cultural variables. This kind of detailed work can only be properly conducted as part of an ongoing program of research wherein the relationships between R/S and health are the focus of investigations.

Though it is not always possible, depending on what aspects of R/S and health are being studied, greater use of experimental studies is encouraged. Nearly all of the existing work on R/S and health is observational. To some degree, observational work will always play a prominent role in this area because it is simply not possible to randomly assign individuals to, for example, certain religious beliefs or groups. However, some aspects of religious practice (e.g., scripture reading or other private religious practices) are amenable to randomized controlled

study when carried out in sensitive ways among members of proper groups (i.e., those willing to engage in the particular practice).

Greater use of large-scale longitudinal research designs is strongly indicated. Though these studies are not capable of determining causality, they may be able to establish temporal precedence, an important factor in many areas of R/S and health research and a preliminary step toward drawing causal conclusions. Further, longitudinal designs, particularly those that are carried out over the long term, have the potential to shed light on developmental trends in relationships between R/S and health.

Finally, we recommend that investigators partner with theologians or become more familiar with theological constructs themselves, as these constructs pertain to the particular groups that are the focus of study. In this way, a deeper understanding of the workings of religion and health is likely to develop, one that may ultimately have practical implications for clergy and their congregants.

Author Biographies

Kevin S. Masters, PhD, Professor of Psychology, Program Director, Clinical Health Psychology, University of Colorado Denver, Denver, Colorado, USA. Dr. Masters' research investigates religious and spiritual factors and processes as they relate to health, in particular cardiovascular responses to psychological stress and healthy behavior change. Recently he has begun investigations of meaning in daily life and its possible effects on health variables. Dr. Masters is past president of the Society for Health Psychology.

Stephanie A. Hooker, MS, MPH, Clinical Health Psychology PhD Candidate, University of Colorado Denver, Denver, Colorado, USA. Ms. Hooker's research investigates positive psychosocial predictors of health behavior change and maintenance. She is interested in translating her basic research findings to develop and evaluate theory-driven behavioral interventions for obesity and cardiovascular disease prevention and treatment. Her recent research has focused on daily meaning in relation to physical activity.

References

- Ai, A. L., Peterson, C., Tice, T. N., Rodgers, W., Seymour, E. M., & Bolling, S. F. (2006). Differential effects of faith-based coping on physical and mental fatigue in middle-aged and older cardiac patients. *The International Journal of Psychiatry in Medicine*, 36, 351–365. doi:10.2190/88CC-W73K-0TM4-JX3J
- Ano, G. G., & Vasconcelles, E. B. (2005). Religious coping and psychological adjustment to stress: A meta-analysis. *Journal of Clinical Psychology*, 61, 461–480. doi:10.1002/jclp.20049
- Benjamins, M. R., & Brown, C. (2004). Religion and preventative health care utilization among the elderly. *Social Science & Medicine*, 58, 109–118. doi:10.1016/s/0277-9536(03)00152-7
- Chida, Y., Steptoe, A., & Powell, L. H. (2009). Religiosity/spirituality and mortality. A systematic quantitative review. *Psychotherapy and Psychosomatics*, 78, 81–90. doi:10.1159/000190791
- Hooker, S. A., Masters, K. S., & Carey, K. B. (2014). Multidimensional assessment of religiousness/spirituality and health behaviors in college students. *The International Journal for the Psychology of Religion*, 24, 228–240. doi:10.1080/10408619.2013.808870
- Jim, H. S. L., Pustejovsky, J. E., Park, C. L., Danhauer, S. C., Sherman, A. C., Fitchett, G., ... Salsman, J. M. (2015). Religion, spirituality, and physical health in cancer patients: A meta-analysis. *Cancer*. doi:10.1002/cncr.29353

- Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality*, 2, 191–205. doi:10.1037/a0020498
- Koenig, H. G., King, D., & Carson, V. B. (2012). *Handbook of religion and health* (2nd ed.). New York, NY: Oxford University Press.
- Masters, K. S., Spielmans, G. I., & Goodson, J. T. (2006). Are there demonstrable effects of distant intercessory prayer? A meta-analytic review. *Annals of Behavioral Medicine*, 32, 337–342. doi:10.1207/s15324796abm3201_3
- McCullough, M. E., & Willoughby, B. L. B. (2009). Religion, self-regulation, and self-control: Associations, explanations, and implications. *Psychological Bulletin*, 135, 69–93. doi:10.1037/a0014213
- Park, C. L., Brooks, M. A., & Sussman, J. (2009). Dimensions of religion and spirituality in psychological adjustment in older adults living with congestive heart failure. In A. Ai & M. Ardel (Eds.), *The role of faith in the well-being of older adults: Linking theories with evidence in an interdisciplinary inquiry* (pp. 112–134). Hauppauge, NY: Nova Science Publishers.
- Park, C. L., Edmondson, D., Hale-Smith, A., & Blank, T. O. (2009). Religiousness/spirituality and health behaviors in younger adult cancer survivors: Does faith promote a healthier lifestyle? *Journal of Behavioral Medicine*, 32, 582–591. doi:10.1007/s10865-009-9223-6
- Park, C. L., Masters, K. S., Salsman, J. M., Wachholtz, A., Clements, A. D., Salmoirago-Blotcher, E., ... Wischenka, D. M. (2017). Advancing our understanding of religion and spirituality in the context of behavioral medicine. *Journal of Behavioral Medicine*. doi:10.1007/s10865-016-9755-5
- Park, C. L., Moehl, B., Fenster, J. R., Suresh, D. P., & Bliss, D. (2008). Religiousness and treatment adherence in congestive heart failure patients. *Journal of Religion, Spirituality & Aging*, 20, 249–266. doi:10.1080/15528030802232270
- Pew Research Center for Religion in Public Life. (2014). *Religious landscape study*. Washington, DC: Pew Research Center. <http://www.pewforum.org/religious-landscape-study/>
- Roberts, L., Ahmed, I., & Davison, A. (2009). Intercessory prayer for the alleviation of ill health. *Cochrane Database of Systematic Reviews*, (2), CD000368. doi:10.1002/14651858.CD000368.pub3
- Salsman, J. M., Fitchett, G., Merluzzi, T. V., Sherman, A. C., & Park, C. L. (2015). Religion, spirituality, and health outcomes in cancer: A case for a meta-analytic review. *Cancer*. doi:10.1002/cncr.29349
- Salsman, J. M., Pustejovsky, J. E., Jim, H. S. L., Munoz, A. R., Merluzzi, T. V., George, L., ... Fitchett, G. (2015). A meta-analytic approach to examining the correlation between religion/spirituality and mental health in cancer. *Cancer*. doi:10.1002/cncr.29350
- Sherman, A. C., Merluzzi, T. V., Pustejovsky, J. E., Park, C. L., George, L., Fitchett, G., ... Salsman, J. M. (2015). A meta-analytic review of religious or spiritual involvement and social health among cancer patients. *Cancer*. doi:10.1002/cncr.29352
- Smith, T. B., McCullough, M. E., & Poll, J. (2003). Religiousness and depression: Evidence for a main effect and the moderating influence of stressful life events. *Psychological Bulletin*, 129, 614–636.

Further Readings

- Ai, A. L., Huang, B., Bjorck, J., & Appel, H. B. (2013). Religious attendance and major depression among Asian Americans from a national database: The mediation of social support. *Psychology of Religion and Spirituality*, 5, 78–89. doi:10.1037/a0030625
- Carter, E. C., McCullough, M. E., & Carver, C. S. (2012). The mediating role of monitoring in the association of religion with self-control. *Social Psychological and Personality Science*, 3, 691–697. doi:10.1177/1948550612438925
- Ellison, C. G., & Hummer, R. A. (Eds.) (2010). *Religion, families, and health: Population-based research in the United States*. New Brunswick, NJ: Rutgers University Press.

- Miller, W. R., & Thoresen, C. E. (2003). Spirituality, religion, and health. An emerging research field. *American Psychologist*, 58, 24–35. doi:10.1037/0003-066x.58.1.24
- Oman, D., & Thoresen, C. E. (2002). Does religion cause health? Differing interpretations and diverse meanings. *Journal of Health Psychology*, 7, 365–380. doi:10.1177/135910539900400301

Stress and Resilience in Pregnancy

Alyssa C. D. Cheadle¹, Isabel F. Ramos²,
and Christine Dunkel Schetter²

¹*Psychology Department, Hope College, Holland, MI, USA*

²*Department of Psychology, University of California, Los Angeles, CA, USA*

Pregnancy is the period during which offspring develop in their mother's womb. In humans, a full-term pregnancy lasts 40 weeks and is divided into three trimesters. The first trimester begins at conception and goes through 12 weeks, the second trimester is 13–28 weeks, and the third trimester is 29 weeks through the birth of the baby. The normative physiological changes in pregnancy are well established, and most pregnancies result in a healthy infant born at full term, of normal weight. However, there is substantial variability in pregnancy outcomes. Infants born prior to 37 weeks of gestation are preterm or premature, and those born weighing less than 2500 g (5 lb, 8 oz) are low birthweight. These unfavorable birth outcomes occur for 8 and 12% of pregnancies worldwide. These birth outcomes are consequential with respect to infant mortality and developmental problems in offspring and incur high emotional and economic costs for families (Butler & Behrman, 2007). The etiology of adverse birth outcomes is not well determined. However, the life experiences of women during pregnancy have been clearly implicated. Today, scientists across disciplines are working together to increase our understanding of biopsychosocial and sociocultural influences on pregnancy and birth.

Pregnancy is stereotyped as a happy time and for many, it is. The image of a mother and her partner preparing to welcome a child they want is prevalent, but it does not reflect that many pregnant women's lives are affected by socioeconomic disadvantages, life stress, and emotional distress. During pregnancy, women may face a variety of major stressors, like the death of a family member and legal problems including with their own or family members' immigration status, relationship conflict, and financial hardship. The impact on pregnancy and birth of these surprisingly common stressors cannot be underestimated. For example, most of the studies on major life events including major catastrophes such as earthquakes show an effect on preterm birth. Additionally, in recent years, stress research has broadened its focus to include the reverse—resilience in the face of chronic stress—defined here as the ability to withstand and

cope with ongoing or repeated demands (Schetter & Dolbier, 2011). Resilience resources that a woman has may benefit her emotional welfare, coping, and the outcomes of her pregnancy.

Mood Disorders

Affective disorders are more prevalent during pregnancy and postpartum than other times in women's lives. Prenatal and postpartum affective disorders and symptoms are noteworthy because they can have potentially deleterious effects on women, infants, and families. Depression and anxiety during pregnancy have been implicated in poor birth outcomes, namely, preterm birth and low birthweight (Schetter & Tanner, 2012). Pregnancy and the postpartum period are marked by increased risk of negative mood states including depressive and anxiety disorders, partly related to the extensive physiological changes that can adversely affect mood and often due to changes in life circumstances. Additionally, when women discontinue medications for mood disorders in pregnancy to protect their fetus from risk, their mood may become a risk factor.

Pregnancy Anxiety

Up to one in four women experience prenatal anxiety, though prevalence depends on the type of anxiety symptoms and disorders studied (Schetter & Tanner, 2012). *Pregnancy anxiety* refers to specific fears or worries about a current pregnancy, including about the health of one's baby, labor, delivery, and parenthood. These pregnancy-specific worries are associated with increased risk of preterm birth. Elevated levels of pregnancy anxiety also predict infant and child developmental outcomes, especially poorer cognitive and motor performance, and more negative temperament in infancy (Blair, Glynn, Sandman, & Davis, 2011). Little research links prenatal anxiety to low birthweight (Accortt, Cheadle, & Schetter, 2015).

Prenatal and Postpartum Depression

Conservative estimates of rates of postpartum depression (PPD) range from 7 to 13% (Rich-Edwards et al., 2006), making this type of depression as prevalent as major depressive disorder among nonpregnant women. A recent systematic review found prenatal depression to be consistently associated with increased risk of low birthweight possibly as a result of adverse health behaviors that affect growth. The overall evidence on preterm birth is less conclusive with some studies suggesting a link and most suggesting no link (Accortt et al., 2015). PPD is also consequential for women. Women who experience postpartum affective disorders are more likely to have difficulty returning to prepregnancy levels of general well-being and employment functioning and to experience relationship stress (Abrams & Curran, 2007). PPD is associated with increased risk of poor infant–parent attachment and low rates of breastfeeding that can lead to greater risk of cognitive, psychological, and behavioral problems in childhood and beyond (Paul, Downs, Schaefer, Beiler, & Weisman, 2013). It is important for women and health providers to pay attention to depressive symptoms preceding and following birth—even if they are not clinically significant.

Health Disparities

Socioeconomic status (SES) is a clear risk factor for preterm birth and low birthweight. Low SES can lead to life conditions such as unemployment and crowding, which in turn predict poor

birth outcomes including low birthweight (Borders, Grobman, Amsden, & Holl, 2007). SES also works in combination with other risks, such as race/ethnicity, for adverse birth outcomes.

Ethnic/racial disparities are evident in the numbers and types of stressors and the emotional experiences of pregnancy and birth outcomes. Regarding PPD, the majority of evidence suggests that African American and Latina women experience higher rates than non-Hispanic White women (e.g., Rich-Edwards et al., 2006). This disparity persists even when SES is taken into account (e.g., McLennan, Kotelchuck, & Cho, 2001). In addition, African American women have higher rates of poor birth outcomes with rates nearly twice as high as those of other groups, even after controlling for SES. Perceived racism and discrimination during pregnancy and over a mother's lifetime may contribute to this disparity (e.g., Parker-Dominguez, Schetter, Mancuso, Rini, & Hobel, 2005). Importantly, these effects are independent of other stressors.

Mexican American women, or those born in the United States of Mexican descent, have higher levels of perceived social stress, have more exposure to chronic stressors, and may be more likely to be depressed than Mexican immigrant women. However, pregnant Mexican immigrant women have been found to experience higher levels of pregnancy-related anxiety (Fleuriet & Sunil, 2014). Finally, all Latinas (foreign born and US born) report feeling more pregnancy anxiety than European American women living in California (Campos et al., 2008).

Close Relationships

Partners, family, and friends are common sources of support in life especially during pregnancy. Social support refers to perceived available and received, or enacted, support, both of which can have an impact on the emotional experiences and outcomes of pregnancy. Support may be material, task, emotional, or informational (i.e., advice), and it may be effective or ineffective (Rini, Schetter, Hobel, Glynn, & Sandman, 2006). Generally speaking, greater prenatal support appears to predict better birth outcomes including better fetal growth and higher birthweight (e.g., Hedegaard, Henriksen, Secher, Hatch, & Sabroe, 1996).

Social support may also moderate, or buffer, the negative influences of stressors on the emotional experience and outcomes of pregnancy. Some support for this exists. For instance, in a study of pregnant women, social support was associated with higher birthweight, but this effect existed only for those women who reported experiencing high numbers of stressful life events (Collins, Schetter, Lobel, & Scrimshaw, 1993).

Though relationships can be sources of support, relationships can also be sources of stress in general and for pregnant women specifically. When a woman has few social connections or support from family and friends is inadequate, poorer birth outcomes are more likely including shorter gestational length, smaller size at birth, and preterm birth.

Perhaps the most important relationship during pregnancy is that between the mother and the baby's father. Fathers who provide material and task assistance and who are perceived as emotionally supportive can be key to a healthier pregnancy. On the flip side, negative interactions and, in the extreme, intimate partner violence can negatively impact a mother's health and that of her infant (e.g., depressive symptoms; Urquia, O'Campo, Heaman, Janssen, & Thiessen, 2011).

Health Behaviors

Smoking during pregnancy is associated with increased risk of giving birth to an infant of low birthweight and with premature delivery (Butler & Behrman, 2007). Low maternal education, low

income, and non-Hispanic White ethnicity as well as stress are predictors of smoking during pregnancy (Mathews, 2001). The mechanisms by which unhealthy behaviors during pregnancy explain the influence of stress on adverse birth outcomes remain largely unknown. Nonetheless, there is evidence that expecting mothers who experience high levels of pregnancy-specific stress, e.g., concerns about medical care, physical symptoms, and bodily changes during pregnancy, smoke cigarettes more often during pregnancy and are more likely to deliver a low birthweight infant (Lobel, 2008). Regular and moderately intense physical activity throughout pregnancy is associated with optimal pregnancy and delivery outcomes (Clapp, Kim, Burciu, & Lopez, 2000). For example, women who begin exercising regularly early in pregnancy and continue to delivery have given birth to higher birthweight infants compared with women who did not exercise regularly.

Sociocultural Influences

Researchers have explored the extent to which an expectant mother's cultural background positively influences the support she receives throughout her pregnancy and even affects her pregnancy health and birth outcomes. Of particular interest is *familism*, a cultural value that is high among Latinos and highlights the importance of emotionally positive, supportive family relationships (Freeberg & Stein, 1996). In a sample of foreign-born Latina, US-born Latina, and European American pregnant women, familism was positively associated with social support and negatively associated with stress and pregnancy anxiety (Campos et al., 2008). Interestingly, for foreign-born Latinas only (not those born in the United States), social support predicted higher infant birthweight. Thus, sociocultural factors as they influence close relationships figure prominently in both maternal mental health and healthy outcomes such as birthweight.

Religiousness and Spirituality

Pregnancy and childbirth are times in the lives of women and families when religiousness and spirituality are especially salient. The birth of a child often occasions discussions of religious issues including religious rituals surrounding birth and parental decision making about religious rearing of children. A small literature shows that religious participation and spiritual behaviors and experiences are associated with lower PPD. Recently, work by our group with African American women found that both religiosity and spirituality independently predicted favorable trajectories of depressive symptoms over the 6 months after the birth of their children (Cheadle et al., 2015). These studies and others indicate that religiousness and spirituality have implications for better emotional experiences of pregnancy.

Conclusions

In summary, pregnancy and birth outcomes are impacted by many complex factors in the lives of mothers at the individual, family, and sociocultural levels that can be sources of stress and of resilience. During pregnancy, women are at greater risk of mood disorders and experiencing pregnancy-related anxiety and depressive symptoms with consequences for birth outcomes and, ultimately, for the well-being of children and families. Low SES and its sequelae also have extremely adverse and sometimes compounding effects on birth and developmental outcomes. There are also ethnic/racial disparities both in terms of mental health during pregnancy and

birth outcomes with African American and Latina women experiencing worse mental health and poorer birth outcomes, whereas cultural factors such as familism and collectivism are associated with better mental health during and after pregnancy. In general, resilience resources may enable poor and lower educated women to function well despite low SES. Women's close relationships can also have positive influences on pregnancy and birth outcomes; however, unsupportive or abusive relationships are clearly harmful. Some health behaviors such as smoking, use of other substances, and unhealthy diet are associated with poorer birth outcomes, whereas positive health behaviors like moderate exercise are associated with better outcomes.

The physiological changes of pregnancy are relatively well established and normative, yet knowledge on psychological and sociocultural factors and how they interact with biological processes to impact mental and physical health during and after pregnancy and birth outcomes is underdeveloped. Past research clearly demonstrates that stress and resilience matter to the experience and health of a pregnancy, yet there is much we still can learn and use to help women and their families including how to have a healthy preconception period. Future research should focus on the physiological impacts of stress on mothers, pregnancy experiences, and infants and the interaction of these effects with resilience factors.

Author Biographies

Alyssa C. D. Cheadle, PhD, is an assistant professor in the Psychology Department at Hope College in Holland, Michigan. She received her PhD in Psychology from the University of California, Los Angeles, in 2016. She conducts research on religiousness, spirituality, and mental and physical health and mechanisms underlying the effects. Her previous research focused on these topics in pregnant and postpartum women, and she has studied the health effects of forgiveness.

Isabel F. Ramos, MA, is a doctoral student in Health Psychology at the University of California, Los Angeles. She received her BA in Psychology from UC Riverside. Isabel now investigates stress and anxiety in pregnant women of diverse ethnicities. Her research examines ethnic and racial disparities in maternal health and cultural factors that influence perinatal biopsychosocial processes. Her work has shown that pregnancy anxiety predicts the timing of delivery in Latina, White, and Black women.

Christine Dunkel Schetter, PhD, is a professor of psychology and psychiatry at the University of California, Los Angeles. She received her PhD from Northwestern University and completed postdoctoral training at UC Berkeley with Richard Lazarus. Her research expertise is in stress, coping, and social support in a variety of health and mental health contexts with primary focus on stress processes in pregnancy. She is proud of her many lab members, past and present, including Dr. Cheadle and Ms. Ramos.

References

- Abrams, L. S., & Curran, L. (2007). Not just a middle-class affliction: Crafting a social work research agenda on postpartum depression. *Health & Social Work, 32*(4), 289–296. doi:10.1093/hsw/32.4.289
- Accortt, E. E., Cheadle, A. C., & Schetter, C. D. (2015). Prenatal depression and adverse birth outcomes: An updated systematic review. *Maternal and Child Health Journal, 19*(6), 1306–1337. doi:10.1007/s10995-014-1637-2

- Blair, M. M., Glynn, L. M., Sandman, C. A., & Davis, E. P. (2011). Prenatal maternal anxiety and early childhood temperament. *Stress, 14*(6), 644–651. doi:0.3109/10253890.2011.594121
- Borders, A. E. B., Grobman, W. A., Amsden, L. B., & Holl, J. L. (2007). Chronic stress and low birth weight neonates in a low-income population of women. *Obstetrics & Gynecology, 109*(2, Part 1), 331–338. doi:10.1097/01.AOG.0000250535.97920.b5
- Butler, A. S., & Behrman, R. E. (Eds.) (2007). *Preterm birth: Causes, consequences, and prevention*. National Academies Press. doi:10.17226/11622
- Campos, B., Schetter, C. D., Abdou, C. M., Hobel, C. J., Glynn, L. M., & Sandman, C. A. (2008). Familialism, social support, and stress: Positive implications for pregnant Latinas. *Cultural Diversity and Ethnic Minority Psychology, 14*(2), 155. doi:10.1037/1099-9809.14.2.155
- Cheadle, A. C. D., Schetter, C. D., Gaines Lanzi, R., Reed Vance, M., Sahadeo, L. S., Shalowitz, M. U., ... CCHN, T. (2015). Spiritual and religious resources in African American women: Protection from depressive symptoms after childbirth. *Clinical Psychological Science, 3*(2), 283–291. doi:10.1177/2167702614531581
- Clapp, J. F., Kim, H., Burciu, B., & Lopez, B. (2000). Beginning regular exercise in early pregnancy: Effect on fetoplacental growth. *American Journal of Obstetrics and Gynecology, 183*(6), 1484–1488. doi:10.1067/mob.2000.107096
- Collins, N. L., Schetter, C. D., Lobel, M., & Scrimshaw, S. C. (1993). Social support in pregnancy: psychosocial correlates of birth outcomes and postpartum depression. *Journal of Personality and Social Psychology, 65*(6), 1243. doi:10.1037/0022-3514.65.6.1243
- Fleuriet, K. J., & Sunil, T. S. (2014). Perceived social stress, pregnancy-related anxiety, depression and subjective social status among pregnant Mexican and Mexican American women in south Texas. *Journal of Health Care for the Poor and Underserved, 25*(2), 546–561. doi:10.1353/hpu.2014.0092
- Freeberg, A. L., & Stein, C. H. (1996). Felt obligation towards parents in Mexican-American and Anglo-American young adults. *Journal of Social and Personal Relationships, 13*(3), 457–471. doi:10.1177/0265407596133009
- Hedegaard, M., Henriksen, T. B., Secher, N. J., Hatch, M. C., & Sabroe, S. (1996). Do stressful life events affect duration of gestation and risk of preterm delivery? *Epidemiology, 7*(4), 339–345. doi:10.1097/00001648-199607000-00001
- Lobel, M., Hamilton, J. G., & Cannella, D. T. (2008). Psychosocial perspectives on pregnancy: Prenatal maternal stress and coping. *Social and Personality Psychology Compass, 2*(4), 1600–1623. doi:10.1111/j.1751-9004.2008.00119.x
- Mathews, T. J. (2001). Smoking during pregnancy in the 1990s. *National Vital Statistics Report, 49*(7), 1–14. doi:10.1037/e558942006-001
- McLennan, J. D., Kotelchuck, M., & Cho, H. (2001). Prevalence, persistence, and correlates of depressive symptoms in a national sample of mothers of toddlers. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(11), 1316–1323. doi:10.1097/00004583-200111000-00012
- Parker-Dominguez, T., Schetter, C. D., Mancuso, R., Rini, C. M., & Hobel, C. (2005). Stress in African American pregnancies: testing the roles of various stress concepts in prediction of birth outcomes. *Annals of Behavioral Medicine, 29*(1), 12–21. doi:10.1207/s15324796abm2901_3
- Paul, I. M., Downs, D. S., Schaefer, E. W., Beiler, J. S., & Weisman, C. S. (2013). Postpartum anxiety and maternal-infant health outcomes. *Pediatrics, 131*(4), e1218–e1224. doi:10.1542/peds.2012-2147
- Rich-Edwards, J. W., Kleinman, K., Abrams, A., Harlow, B. L., McLaughlin, T. J., Joffe, H., & Gillman, M. W. (2006). Sociodemographic predictors of antenatal and postpartum depressive symptoms among women in a medical group practice. *Journal of Epidemiology and Community Health, 60*(3), 221–227. doi:10.1136/jech.2005.039370
- Rini, C., Schetter, C. D., Hobel, C. J., Glynn, L. M., & Sandman, C. A. (2006). Effective social support: Antecedents and consequences of partner support during pregnancy. *Personal Relationships, 13*(2), 207–229. doi:10.1111/j.1475-6811.2006.00114.x
- Schetter, C. D., & Dolbier, C. (2011). Resilience in the context of chronic stress and health in adults. *Social and Personality Psychology Compass, 5*(9), 634–652. doi:10.1111/j.1751-9004.2011.00379.x

- Schetter, C. D., & Tanner, L. (2012). Anxiety, depression and stress in pregnancy: implications for mothers, children, research, and practice. *Current Opinion in Psychiatry*, 25(2), 141–148.
- Urquia, M. L., O'Campo, P. J., Heaman, M. I., Janssen, P. A., & Thiessen, K. R. (2011). Experiences of violence before and during pregnancy and adverse pregnancy outcomes: An analysis of the Canadian Maternity Experiences Survey. *BMC Pregnancy and Childbirth*, 11(1), 1–9. doi:10.1186/1471-2393-11-42

Suggested Reading

- Giscombé, C. L., & Lobel, M. (2005). Explaining disproportionately high rates of adverse birth outcomes among African Americans: the impact of stress, racism, and related factors in pregnancy. *Psychological Bulletin*, 131(5), 662–683. doi:10.1037/0033-2909.131.5.662
- Lobel, M., Hamilton, J. G., & Cannella, D. T. (2008). Psychosocial perspectives on pregnancy: prenatal maternal stress and coping. *Social and Personality Psychology Compass*, 2(4), 1600–1623. doi:10.1111/j.1751-9004.2008.00119.x
- Schetter, C. D. (2009). Stress processes in pregnancy and preterm birth. *Current Directions in Psychological Science*, 18(4), 205–209. doi:10.1111/j.1467-8721.2009.01637.x
- Schetter, C. D. (2011). Psychological science on pregnancy: Stress processes, biopsychosocial models, and emerging research issues. *Annual Review of Psychology*, 62, 531–558. doi:10.1146/annurev.psych.031809.130727
- Schetter, C. D., & Lobel, M. (2010). Pregnancy and birth: A multilevel analysis of stress and birth weight. In T. A. Revenson, A. Baum, & J. Singer (Eds.), *Handbook of Health Psychology* (pp. 43–463). London: Psychology Press. doi:10.4324/9780203804100

Subjective Health Norms

Allecia E. Reid¹, Molly B. Hodgkins², Carly A. Taylor³,
and Alexandra A. Belzer¹

¹*Colby College, Waterville, ME, USA*

²*Department of Psychological and Brain Sciences, University of Maine, Orono, ME, USA*

³*Boston Medical Center, Boston, MA, USA*

Psychologists, sociologists, and others interested in human behavior have long recognized that the attitudes and behaviors of others bear influence on our own attitudes and behaviors. These social norms reflect common standards for behavior, set by and for members of a social group. Although a number of types of social norms have been examined, a large body of research has specifically focused on Cialdini, Reno, and Kallgren (1990) distinction between descriptive and injunctive norms. Descriptive and injunctive norms capture the distinction between the *is* and the *ought* of behavior. That is, while descriptive norms reflect perceptions of how most others behave in a given situation, injunctive norms capture perceptions of what is commonly approved or disapproved by most others. For example, a descriptive norm might reflect that most individuals in an environment smoke cigarettes. However, the corresponding injunctive norm might reflect that most individuals disapprove of smoking due to its negative health effects.

Much research has also examined subjective norms, a construct proposed as a determinant of intentions to engage in a behavior in the theories of reasoned action and planned behavior. Subjective norms differ from the broader conceptualization of injunctive norms in that they assess whether specific valued individuals (e.g., family members, one's doctor) approve of a behavior, rather than whether a generalized group of others approve of the behavior. The present entry focuses on the broader conceptualization of injunctive norm, incorporating research that has assessed subjective norms where appropriate.

Descriptive and injunctive norms have distinct motivational underpinnings and therefore are likely to operate under different circumstances (Cialdini & Trost, 1998; Jacobson, Mortensen, & Cialdini, 2011). Descriptive norms are most likely to motivate behavior when accurate decision making is of importance and are particularly influential in novel situations or when the appropriate course of action is unclear. Injunctive norms, on the other hand, exist to support the formation and maintenance of social bonds with important groups or individuals.

As a result, injunctive norms motivate behavior primarily through the threat of social disapproval for inappropriate behavior or the promise of approval for conformity. Conformity to injunctive norms is therefore most likely when social approval concerns are heightened.

Questions have been raised about the distinctiveness of descriptive and injunctive norms. Indeed, the manner in which others behave is often consistent with what is approved of by most others. However, confirmatory factor analyses examining descriptive and injunctive norms for calcium consumption, exercise, and eating behaviors, among others, support that the two are distinct, unitary constructs. Further, as demonstrated in the smoking example above, descriptive and injunctive norms can convey opposing information for the same behavior. This contradiction is often evident in public service announcements and health interventions that depict unhealthy behaviors as regrettably common, conveying a favorable descriptive norm, but widely disapproved, conveying an unfavorable injunctive norm.

Influences of Norms on Health Behaviors

Previous research has examined whether descriptive and injunctive norms predict intentions or behavior with respect to health risk, health protective, and health screening behaviors.

Health Risk Behaviors

In the context of health risk behaviors, perceptions of norms play particularly prominent roles in use of cigarettes, alcohol, and other drugs among adolescents and young adults. Research has documented associations of descriptive norms with increases in substance use up to 2 years later. Supporting the view that descriptive norms are particularly influential in novel situations, preexisting perceptions of descriptive norms influence alcohol use across transitions into new situations, such as matriculating to college and studying abroad. The literature is mixed regarding the influence of injunctive norms on alcohol and drug use relative to descriptive norms. Prospective research has demonstrated roles for both norms in alcohol and cigarette use, only an influence of descriptive norms on alcohol use, and only an influence of injunctive norms on alcohol use. Although much of the health risk literature focuses on adolescents and young adults, norms also influence adults' behaviors. For example, descriptive but not injunctive norms longitudinally predict sharing needles and other drug equipment among middle-aged injection drug users.

The trend toward finding associations between substance use outcomes and descriptive but not injunctive norms may reflect that these behaviors often occur with peers in social settings, a situation that likely enhances the salience and therefore the impact of descriptive norms relative to injunctive norms (Cialdini et al., 1990). Moreover, recognizing and conforming to others' behaviors requires less cognitive processing and potentially engages a faster motivational system than considering others' approval of the behavior (Cialdini, 2003; Jacobson et al., 2011). As a result, descriptive norms may generally more strongly influence behaviors that occur primarily in social contexts.

Health Protective Behaviors

Descriptive and injunctive norms also both play roles in health protective behaviors. This literature, drawing heavily on the theories of reasoned action and planned behavior, has demonstrated that the influence of norms on behavior is fully mediated by intentions. As a result,

cross-sectional examinations of the relationships between norms and intentions are common, with longitudinal examination of the influence of intentions on behavior.

Descriptive and injunctive norms have demonstrated associations with intentions and occasionally behavior for a range of health protective behaviors, including sun protection, fruit and vegetable consumption, diet, and physical activity. Meta-analysis also supports cross-sectional and longitudinal associations of both norms with condom use. Both norms also relate to intentions for receiving vaccinations to protect against human papillomavirus and swine flu, with injunctive norms predicting later uptake of the HPV vaccine. Among the limited studies that have simultaneously examined both descriptive and injunctive norms, the balance indicates that descriptive norms are more strongly associated with intentions to engage in healthy eating, calcium consumption, exercise, and other health behaviors. The salience of others' behavior may explain the stronger influence of descriptive norms on outcomes that are publicly visible. Ceiling effects may also mitigate the role of injunctive norms in this domain, with individuals perceiving little, if any, disapproval for protecting one's health by eating healthily, exercising, etc.

Screening Behaviors

Injunctive and descriptive norms are also both related to screening behaviors. However, research has found that injunctive norms bear stronger influence on intentions to obtain a mammogram, a colonoscopy, and prostate cancer screening than descriptive norms. Likewise, only injunctive norms predict intentions to undergo screening for sexually transmitted infections or uptake of prostate or colorectal cancer screening. Of course, alternative findings exist. Nonetheless, the stronger influence of injunctive norms on screenings may reflect lack of confidence in perceptions of the extent to which others engage in these private behaviors. Indeed, lack of confidence in norm perceptions has been found to weaken the impact of norms. On the other hand, we may infer that health screenings are widely approved of by others, as conveyed by public service announcements encouraging monitoring one's health. The types of behaviors for which injunctive versus descriptive norms are likely to influence intentions and behavior require more careful scrutiny in future research.

Using Norms to Change Behavior

Whether norms can be used to change behavior, which typically requires prolonged effort and self-regulation, is ultimately of interest. Social norms theory (Perkins & Berkowitz, 1986) forms the basis of most research that uses social norms to promote healthier behaviors. From the perspective of this theory, perceptual biases and reliance on heuristics lead to a general tendency to overestimate the extent to which others engage in and approve of risky behaviors but underestimate others' engagement in and approval of protective behaviors. For example, young adults overestimate the level of sexual activity among peers and underestimate peers' condom use. Misperceptions have also been documented with respect to alcohol, tobacco, marijuana, and other drug use. Women also incorrectly believe that other women are thinner than they themselves are and that men find overly thin women attractive.

Given the existence of these misperceptions, the social norms approach proposes that making individuals aware of the true norms in their environment should recalibrate their perceptions of the norms, producing positive changes in behavior. These norm reeducation approaches have primarily been applied to health risk and health protective behaviors. Most studies have

taken either a social norms marketing or personalized normative feedback approach, with fewer studies utilizing alternative methods.

The social norms marketing approach simply presents participants with information about the norms for behavior among peers, without addressing participants' own behavior or misperceptions of the norm. For example, this type of message might state, "More than 90 percent of the time, people in this building use the stairs instead of the elevator," communicating a descriptive norm that favors staircase over elevator use (Burger & Shelton, 2011). Social norms marketing messages are often delivered via advertisements placed in key locations but may also be delivered individually. Descriptive social norms marketing has demonstrated efficacy in increasing occupational physical activity, fruit consumption, salad consumption, and interest in chemotherapy. This approach has also demonstrated efficacy in improving alcohol use, intervening with friends who may be drinking too much, and intentions for condom use. Although descriptive social norms marketing has been applied to encouraging cancer screening, its efficacy in that domain remains unclear. Importantly, social norms marketing has been shown to alter perceptions of the descriptive norms, and in turn, changes in descriptive norms fully mediate or account for intervention-induced change in behavior.

The few studies that have used social norms marketing to address injunctive norms have documented mixed efficacy. Thus, additional research is needed to clarify whether this is a viable means for altering injunctive norms. It should be noted that the studies described above differ from mass media marketing campaigns in that they either delivered interventions to participants individually or displayed messages in conspicuous locations and also observed participants' behaviors in the same setting. Mass media efforts that employ normative information can be efficacious (Perkins, Linkenbach, Lewis, & Neighbors, 2010) but may also prove ineffective (Wechsler et al., 2003) because the target audience may not sufficiently process these messages.

Personalized normative feedback, a complementary approach for altering normative perceptions, typically compares three pieces of information: (a) the individual's perceptions of the norms in his or her environment (e.g., perceptions of the extent of alcohol use among peers), (b) the actual descriptive norm in the environment (e.g., actual level of alcohol use among peers derived from surveys), and (c) the individual's own behavior. A comparative efficacy trial found social norms marketing and personalized normative feedback to be similarly efficacious (Neighbors et al., 2011). However, the use of individually tailored content in personalized feedback should hypothetically enhance the self-relevance of the message, thereby encouraging both greater depth of processing and better maintenance of behavior change over time.

As with the social norms marketing approach, personalized normative feedback studies have primarily focused on descriptive norm information. This type of feedback has been utilized both individually and in a group setting as a stand-alone intervention to reduce college students' alcohol use. It is also the primary active ingredient through which multicomponent interventions reduce college students' alcohol use. Personalized descriptive norm feedback was also recently shown to reduce alcohol-induced risky sexual behavior. Further, personalized descriptive norm feedback, whether in single or multiple component interventions, is efficacious because it alters perceptions of the descriptive norms, which in turn drive behavior change. Personalized injunctive norm feedback has received little attention but has been shown to improve women's sun-protective behaviors. Future research might examine whether personalized injunctive norm feedback reliably alters behavior.

The large literature examining the use of personalized normative feedback in the context of college student drinking provides insight into conditions that maximize its impact (Reid &

Carey, 2015). Specifically, although normative information is highly portable and can be delivered via the web or mail, normative feedback is in fact less effective when delivered remotely, rather than by a counselor or computer in person. Remotely delivered interventions suffer because recipients often engage in other activities (e.g., texting, listening to music) while completing the intervention (Lewis & Neighbors, 2015). In addition, it is important to choose the optimal referent group to serve as the basis for the normative message. In general, normative data referencing peers in the same environment is more effective than norms derived from national data. Gender differences in alcohol consumption necessitate providing gender-specific norms, but the utility of this specification likely varies by behavior. It is possible, however, that a referent group might be too specific. LaBrie et al. (2013) found that students held more accurate perceptions of the norms for students of the same gender, ethnicity, and Greek status, relative to norms for “the typical student.” Given that there was little to correct, feedback on the gender–ethnicity–Greek specific norms was associated with *decreased* likelihood of behavior change. It is therefore important to identify the optimal referent group for each behavior of interest.

As noted above, alternative methods exist for altering perceptions of norms in service of behavior change. One unique approach centers on labeling undesirable behaviors as normative among members of an out-group. Berger and Rand (2008) provided undergraduates with information that high-fat food consumption and heavy alcohol use were common among out-groups with which they did not want to be associated. This increased selection of healthy foods and reduced alcohol use among message recipients as a means to avoid being incorrectly labeled as members of these undesirable groups. A second alternative method focuses on aspirational peers and demonstrates that norms have shifted over time. Jackson and Aiken (2006) improved sun protection among young women through photographs demonstrating that tanned skin was common in the past but was no longer popular among present day actresses. This approach might be extended to address other observable behaviors. A third alternative approach involves facilitating structured interactions with peers in group settings such that healthy norms are elicited and supported. Participating in intervention sessions with peers has been shown to alter perceptions of norms for healthy eating among firefighters, safe injection practices among injection drug users, and thin-ideal endorsement and dieting among women with eating disorders (e.g., Ranby et al., 2011). Mediation analyses supported that the effects of these peer-centered interventions on behavior change were explained by changes in normative perceptions.

Moderators of Norm-Based Interventions

Previous research examining individual differences that may moderate the effect of norms on behavior change have fallen into three main categories: level of identification with the referent group, sensitivity to external pressures, and extent of discrepancy between perceptions and the true norms. Consistent with social identity theory (Tajfel & Turner, 1979), research has found descriptive norm interventions to be particularly efficacious for individuals who identify most strongly with the referent group. Similarly, norm interventions show increased efficacy among individuals who are more sensitive to social pressures because they fear being negatively evaluated, drink alcohol primarily for social reasons, or seek out cues in the environment to determine how to behave. Finally, consistent with social norms theory, the more individuals misperceive the norms in their environment, the more likely they are to change their behavior after having their perceptions corrected. Given these moderators, researchers should be

attentive when selecting a referent group and might maximize discrepancy by selecting cognitions for feedback that evidence the largest discrepancies at the individual level, rather than at the group level. Additional research is needed to determine whether social norms interventions can be enhanced for those less sensitive to social forces or if an attitudinal appeal is more appropriate for these individuals.

Future Directions and Conclusions

As indicated above, few studies have utilized injunctive norms to change behavior. While additional research is needed on injunctive norms, normative information is likely to be most influential when descriptive and injunctive norms correspond with one another (Cialdini, 2003). Accordingly, research that compares a combined norms condition against separate descriptive and injunctive norms conditions may find support for this contention, as in previous research on sun protection.

An additional consideration is whether altering normative perceptions affects other psychosocial constructs en route to behavior change. Reid and Aiken (2013) demonstrated that the effect of injunctive norm feedback was mediated by not only change in injunctive norms but also change in attitudes. Exploring whether additional psychological constructs transmit the effects of descriptive and injunctive norms will aid in identifying conditions that are likely to maximize their impact.

In sum, correlational and experimental research support that social norms influence health behaviors. Though unresolved issues remain, research confirms descriptive and injunctive norms are indeed strong motivators of behavior that have much to offer in both characterizing and improving health behaviors.

Author Biographies

Allecia E. Reid is an assistant professor of psychology at Colby College. Her research examines the influences of social norms and peer social networks on health behaviors. She studies mediators and moderators of the influences of these social factors, the use of social norms information for motivating healthier behavior, and techniques for protecting individuals against the negative influence of peers.

Molly B. Hodgkins completed a bachelor's degree in psychology from Colby College in 2015. She is currently pursuing a master of social work at the University of Maine. Molly's research has primarily focused on how feelings of social belonging influence well-being in college students.

Carly A. Taylor completed a bachelor's degree in psychology from Colby College in 2015. She is currently a patient navigator at Boston Medical Center. Carly's research interests include developing interventions to reduce stigma associated with mental illness. Consistent with her goal of becoming a medical doctor, she is also interested in improving doctor-patient interactions as a means for improving patient outcomes.

Alexandra A. Belzer is an undergraduate at Colby College, where she is a research assistant in Dr. Reid's lab. Alexandra is broadly interested in the connection between psychological factors and health outcomes. Her research interests focus on understanding and reducing the negative influence of social norms on health behaviors.

References

- Berger, J., & Rand, L. (2008). Shifting signals to help health: Using identity signaling to reduce risky health behaviors. *Journal of Consumer Research*, 35, 509–518.
- Burger, J. M., & Shelton, M. (2011). Changing everyday health behaviors through descriptive norm manipulations. *Social Influence*, 6, 69–77.
- Cialdini, R. B. (2003). Crafting normative messages to protect the environment. *Current Directions in Psychological Science*, 12, 105–109.
- Cialdini, R. B., Reno, R. R., & Kallgren, C. A. (1990). A focus theory of normative conduct: Recycling the concept of norms to reduce littering in public places. *Journal of Personality and Social Psychology*, 58, 1015–1026.
- Cialdini, R. B., & Trost, M. R. (1998). Social influence: Social norms, conformity and compliance. In D. T. Gilbert, S. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., pp. 151–192). New York, NY: McGraw-Hill.
- Jackson, K. M., & Aiken, L. S. (2006). Evaluation of a multicomponent appearance-based sun-protective intervention for young women: Uncovering the mechanisms of program efficacy. *Health Psychology*, 25, 34.
- Jacobson, R. P., Mortensen, C. R., & Cialdini, R. B. (2011). Bodies obliged and unbound: Differentiated response tendencies for injunctive and descriptive social norms. *Journal of Personality and Social Psychology*, 100, 433–448.
- LaBrie, J. W., Lewis, M. A., Atkins, D. C., Neighbors, C., Zheng, C., Kenney, S. R., ... Hummer, J. F. (2013). RCT of web-based personalized normative feedback for college drinking prevention: Are typical student norms good enough? *Journal of Consulting and Clinical Psychology*, 81, 1074–1086. doi:10.1037/a0034087
- Lewis, M. A., & Neighbors, C. (2015). An examination of college student activities and attentiveness during a web-delivered personalized normative feedback intervention. *Psychology of Addictive Behaviors*, 29, 162–167. doi:10.1037/adb0000003
- Neighbors, C., Jensen, M., Tidwell, J., Walter, T., Fossos, N., & Lewis, M. A. (2011). Social-norms interventions for light and nondrinking students. *Group Processes & Intergroup Relations*, 14, 651–669.
- Perkins, H. W., & Berkowitz, A. D. (1986). Perceiving the community norms of alcohol use among students: Some research implications for campus alcohol education programming. *Substance Use & Misuse*, 21, 961–976.
- Perkins, H. W., Linkenbach, J. W., Lewis, M. A., & Neighbors, C. (2010). Effectiveness of social norms media marketing in reducing drinking and driving: A statewide campaign. *Addictive Behaviors*, 35, 866–874.
- Ranby, K. W., MacKinnon, D. P., Fairchild, A. J., Elliot, D. L., Kuehl, K. S., & Goldberg, L. (2011). The PHLAME (Promoting Healthy Lifestyles: Alternative Models' Effects) firefighter study: Testing mediating mechanisms. *Journal of Occupational Health Psychology*, 16, 501–513.
- Reid, A. E., & Aiken, L. S. (2013). Correcting injunctive norm misperceptions motivates behavior change: A randomized controlled sun protection intervention. *Health Psychology*, 32, 551–560. doi:10.1037/a0028140
- Reid, A. E., & Carey, K. B. (2015). Interventions to reduce college student drinking: State of the evidence for mechanisms of behavior change. *Clinical Psychology Review*, 40, 213–224.
- Tajfel, H., & Turner, J. C. (1979). An integrative theory of intergroup conflict. In W. G. Austin & S. Worchel (Eds.), *The social psychology of intergroup relations* (pp. 33–47). Monterey, CA: Brooks-Cole.
- Wechsler, H., Nelson, T. F., Lee, J. E., Seibring, M., Lewis, C., & Keeling, R. P. (2003). Perception and reality: A national evaluation of social norms marketing interventions to reduce college students' heavy alcohol use. *Journal of Studies on Alcohol and Drugs*, 64, 484–494.

Suggested Reading

- Cialdini, R. B., & Trost, M. R. (1998). Social influence: Social norms, conformity and compliance. In D. T. Gilbert, S. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., pp. 151–192). New York, NY: McGraw-Hill.
- Neighbors, C., Larimer, M. E., & Lewis, M. A. (2004). Targeting misperceptions of descriptive drinking norms: Efficacy of a computer-delivered personalized normative feedback intervention. *Journal of Consulting and Clinical Psychology*, 72, 434–447.
- Schultz, P. W., Tabanico, J., & Rendón, T. (2008). Normative beliefs as agents of influence: Basic processes and real-world applications. In R. Prislin & W. Crano (Eds.), *Attitudes and attitude change* (pp. 385–409). New York, NY: Psychology Press.

Temptation

David A. Kalkstein¹ and Kentaro Fujita²

¹*Department of Psychology, New York University, New York, NY, USA*

²*Department of Psychology, The Ohio State University, Columbus, OH, USA*

Temptations are stimuli in one's immediate environment that prompt thoughts, feelings, and behavior that are contrary to one's goals and values. Temptations rest at the heart of self-control dilemmas—decisions that require choosing between smaller-immediate and larger-distal outcomes (e.g., Mischel, Shoda, & Rodriguez, 1989). Motivationally, self-control can be understood as a conflict between competing motivations—a desire for something now versus a desire for some other outcome of greater magnitude (Fujita, 2011). The ability to secure some small outcome immediately may tempt people to forgo outcomes that are more valuable yet not immediately available. Dieters, for example, might be tempted to abandon their weight-loss efforts when presented with an opportunity to eat chocolate cake now. Substance abusers may struggle to advance their desire to be sober when their drug of choice is freely available. The end of a long, hard day in the office may tempt the sedentary to watch TV rather than work out at the gym. Successful self-control requires overcoming temptations, advancing the pursuit of larger-distal ends over these smaller-immediate outcomes. The omnipresence of temptations in one's social environment poses one of the greatest challenges to achieving and maintaining personal health. Although people may value health, securing and maintaining this end requires overcoming numerous temptations—that is, self-control.

It is important to observe that temptations are subjective in nature. What constitutes a temptation depends on idiosyncratic motivations, values, goals, and desires, which vary from person to person. What represents a temptation for one person may not necessarily represent a temptation for another. First, people must find temptations appealing in some way. Chocolate cake may represent a temptation for someone who likes chocolate. This same object, however, would not represent a temptation for someone who dislikes the taste of chocolate. Second, even when the immediate outcome is desirable in some way, it is only a temptation to the extent that it competes with or undermines some more valuable end. For example, although the allure of cake may represent a temptation for someone concerned with weight loss, it would not represent a temptation for someone unconcerned about their weight. Without this

conflict, rewarding stimuli are simply rewarding stimuli, not temptations. What makes a stimulus a temptation is that it idiomatically makes people “want their cake and eat it too”—it evokes a situation in which two competing desires are activated, only one of which can be satisfied. Critically, of these two competing desires, one can be satisfied immediately yet is of lesser value, whereas the other cannot be satisfied immediately yet is of greater value. In short, for a stimulus to be considered a temptation, it must *both* appeal to immediate desires and impede progress toward more distal but more valued ends.

Self-control is not generally viewed as a problem of knowledge. Self-control failure is defined by acting on a temptation despite knowing in some way that doing so is detrimental to securing more valuable outcomes. To take an extreme example, suppose some dieters erroneously believe that eating donuts will lead to weight loss. It would be inappropriate to label these donuts as temptations for these individuals, just as it would be inappropriate to label their behavior as a self-control failure, since they did not know that eating donuts would harm their weight-loss goals (in this example, the dieters actually thought that eating these donuts would help advance those valued goals). Objects are not temptations if individuals acting on them are not aware implicitly or explicitly that acting on them is detrimental to their broader objectives.

A final, often overlooked, aspect of temptations that is important to consider is that temptations come in the form of both positive *and* negative incentives. One can be tempted by a desire to obtain some smaller yet immediate rewards at the expense of larger yet not immediately available rewards. Examples of this type of temptation include the enjoyment of consuming tasty yet unhealthy food, the highs substance abusers experience from taking harmful drugs, or the hedonic pleasure of engaging in risky sexual behaviors. One might also, however, be tempted by a desire to avoid some smaller yet immediate cost. Examples of this type of temptation include wanting to avoid the discomfort associated with strenuous exercise, the inconvenience and anxiety associated with diagnostic medical tests, and the negative minor side effects of drug regimens. In short, temptations can correspond to desires to obtain proximal rewards or to avoid proximal costs, as both forms have the potential to conflict with and derail more valued goals and objectives.

Deleterious Effects of Temptations

Much of the work done on self-control in the field of psychology has focused on the deleterious impacts of temptations on people’s goal-directed behavior (Kelley, Wagner, & Heatherton, 2015). Generally, this work tends to argue that exposure to temptations leads to self-control failure because it triggers cravings and urges to act on immediately gratifying motivations. For example, tobacco users and drug addicts report the strongest urges to use drugs during, or immediately following, exposure to temptation cues such as drug paraphernalia, pictures of drugs, or *in vivo* presentations of drugs or tobacco (see Carter & Tiffany, 1999).

This research argues that these cravings and impulses influence thoughts, attitudes, and behaviors in the direction of favoring indulgence in the temptation. Evidence for this idea comes from the finding that exposing dieters to tempting food cues (e.g., the smell of fresh baked pizza) leads dieters to report stronger cravings to indulge, which subsequently predicts greater consumption of unhealthy foods (for review, see Kelley et al., 2015). At the neural level, researchers have shown that increases in activity in regions of the brain that process rewards (the nucleus accumbens) in response to temptation cues predict future indulgence in those temptations (Kelley et al., 2015). Indeed, a great deal of research across a variety of health domains has documented cases in which exposure to a temptation cue increases the likelihood of indulgence in that temptation and hence self-control failure.

Underlying much of this work is an implicit (or sometimes explicit) assumption that exposure to temptations automatically activates positive thoughts, feelings, and behavioral tendencies that promote indulgence (e.g., Baumeister & Heatherton, 1996; Fujita, 2011). These automatic impulses influence behavior unless people inhibit or override them. This inhibition process, however, is believed to be effortful, requiring sufficient cognitive and motivational resources. It follows from these premises that any burden on these resources, such as cognitive load or distraction, reduces the likelihood that people will successfully inhibit these impulses, thus leading to an increased likelihood of indulging in temptation.

One especially influential line of research has proposed that this effortful inhibition process not only requires having sufficient cognitive resources but also depletes a motivational resource, much like a battery or muscle (Baumeister & Heatherton, 1996). Proponents of this limited resource model of self-control suggest that all acts of self-control draw on the same limited supply of energy and that an initial exertion of self-control will result in a period of depletion and thus a temporarily diminished capacity to inhibit further automatic impulses that favor temptations. For example, Vohs and Heatherton (2000) showed that dieters who restrained from eating popcorn in an initial task subsequently ate more ice cream on a following task. Similarly, Hofmann, Rauch, and Gawronski (2007) showed that after regulating their emotional responses during an initial movie viewing task, participants' candy consumption was predicted by their automatic attitudes toward candy (which tended to be positive) rather than their dietary restraint standards. These findings support the idea that when people lack the requisite energy to engage in effortful inhibition and are thus "depleted," their behavior is more susceptible to automatic temptation impulses and self-control failure.

Overcoming Temptations

Despite the abundance of research demonstrating the deleterious consequences of exposure to temptations, people also possess a remarkable array of psychological mechanisms and behavioral strategies at their disposal for dealing with temptations.

Motivation to Overcome Temptations

The question of how to overcome and resist temptations is fundamentally a question about how to maintain the motivation to behave in line with valued ends when confronted with the opportunity to indulge in more immediate rewards. The more motivated people are to achieve these more distal ends, the more likely they will be to initiate, engage, and persist in coping behaviors and strategies to deal with and overcome temptations (Trope & Fishbach, 2000). The role of motivation is particularly evident in situations that research suggests people are most vulnerable to temptations—when their cognitive and motivational resources are taxed. For example, providing monetary incentives to increase people's motivation to resist temptation has been shown to improve people's self-control even following periods of depletion (for review, see Masicampo, Martin, & Anderson, 2014). The nature of people's motivation also appears to play an important role in self-control. Research suggests that when people are intrinsically rather than extrinsically motivated—when they are pursuing an end that they are personally relevant and important—they are less susceptible to depletion (see Masicampo et al., 2014).

Research, moreover, indicates that people's beliefs about themselves can impact their motivation to exert the necessary effort to overcome temptations. Motivational theories suggest

that bolstering people's beliefs that they are indeed capable of resisting temptation and procuring more desirable end states (i.e., self-efficacy; Bandura, 1977) should increase their self-control motivation and help them resist temptations. Indeed, it has been shown that increasing people's self-efficacy beliefs about their ability to quit smoking and their ability to lose weight results in greater smoking reduction and more weight loss (for review, see Strecher, DeVellis, Becker, & Rosenstock, 1986).

Beyond self-efficacy, research also points to the important role of people's beliefs about the nature of self-control in their successful resistance to temptations. Research indicates that some endorse a limited view of self-control—a belief that one's ability to resist temptations is finite and can be depleted—whereas others do not (Job, Dweck, & Walton, 2010). Endorsing the view that self-control is limited appears to lead people to give in to temptation more readily, whereas endorsing a more unlimited view appears to motivate more sustained effort to overcome temptation. For example, those who endorse a limited versus unlimited view of self-control tend to eat less healthily, achieve poorer grades, and show less persistence in the face of difficulty (Job, Walton, Bernecker, & Dweck, 2015). Research, moreover, indicates that these beliefs can be changed, suggesting the possibility of leveraging these beliefs about the nature of self-control as a potential point of intervention (Job et al., 2010).

Cognitive Habits

Given sufficient motivation, research suggests that people can develop what might be termed “cognitive habits” to enhance resistance to temptation. In other words, although self-control is often believed to be effortful and involve intensive use of psychological resources, research suggests that people can automate their psychological responses to temptations and be successful in self-control without such resource-intensive effort. Those highly committed to dieting goals, for example, evidence a cognitive readiness to evaluate tempting foods negatively (Fishbach & Shah, 2006). These automatically activated attitudes induce a readiness to resist temptations without requiring effortful deliberation and control.

Beyond evaluations and attitudes, research suggests that sufficiently motivated individuals also develop cognitive associations that bias their thoughts in favor of valued ends rather than more immediate smaller rewards. Self-control should be enhanced to the extent that thoughts about temptations cue thoughts about overriding goals (e.g., “cake” cues “weight loss”), whereas thoughts about those overriding goals does not reciprocally cue thoughts about temptations (e.g., “weight loss” does not cue “cake”). This asymmetric pattern of activation should in turn bias more frequent thoughts about overriding goals rather than temptations. Indeed, research suggests that those committed to their goals are those most likely to display these adaptive asymmetric temptation–goal associations (Fishbach, Friedman, & Kruglanski, 2003). Importantly, people display these associations even under suboptimal processing conditions, such as states of cognitive load—suggesting that these processes may enhance self-control efficiently and without requiring the intensive effort that is commonly assumed necessary for resisting temptations.

It is often assumed that “cognitive habits” to overcome temptations require repeated practice over time, much like a learned skill. However, research suggests that these habits can develop relatively quickly through what is referred to as “implementation intentions” (Gollwitzer & Sheeran, 2006). Implementation intentions are simple “if-then” action plans that link anticipated situations or cues with goal-directed behavior (e.g., “If I’m bored and want a snack, then I will eat an apple!”). They are designed to automate goal-directed behavior such that when a given cue is encountered (e.g., “I am bored”), people will automatically react

with their stated intention (e.g., “eat an apple”). Research has shown that these simple plans can increase health behaviors such as cancer screenings, exercise and physical activity, and dietary restraint and appears to do so by promoting self-control in the face of temptations and under suboptimal processing conditions such as resource depletion (for a review of findings, see Gollwitzer & Sheeran, 2006).

Prospective Control

The most effective strategies that one can employ to overcome temptation may be those involving prospective control. Prospective control seeks to combat the allure of immediate temptations by developing strategies to overcome them prior to exposure. Classic research on self-control has shown that decisions that are made for the future and in the absence of temptations tend to be more in line with people’s valued goals and best interests (Ainslie, 1975). Prospective control facilitates the overcoming of temptation by encouraging people to make decisions to pursue their more valued ends and committing to these decisions in advance of encountering temptations.

Prospective control strategies can be divided into two classes: strategies that seek to regulate the availability of temptations and strategies that seek proactively to regulate responses to temptations. Regulating the availability of temptations involves modifying behavior or selecting situations that make it less likely that one will be encountering temptations and thus less likely that they will feel impulses or desires to indulge the temptations. For example, a dieter who takes an alternative route to work in the morning to avoid the donut shop is utilizing prospective control. In this example, the dieter is purposefully avoiding the sight and smell of donuts that may serve as cues that trigger cravings to eat the donut. By reducing the availability of temptations and avoiding temptation cues, the dieter decreases the likelihood of breaking his diet by eating a donut. Other examples of regulating the availability of temptations and exposure to temptation cues would be a recovering alcoholic staying at home on New Year’s Eve instead of going out to the bars, or a past drug addict refraining from hanging out with drug-using friends. Avoiding temptations from the outset is one of the most effective strategies that people can employ to decrease the likelihood of succumbing to temptation (Mischel et al., 1989). Individuals cannot give in to temptations upon which they never have the opportunity to act.

A second prospective self-control strategy that people can employ to overcome temptation is to regulate potential responses to temptations and precommit oneself to behavior in line with more valued ends. A typical precommitment strategy involves manipulating the contingencies and outcomes associated with acting on the temptation through means such as self-imposed penalties and success-contingent rewards (also referred to as “side bets”; Ainslie, 1975). In one demonstration of self-imposed punishment, Trope and Fishbach (2000) presented people with a medical screening opportunity that promised to provide accurate and useful health information but entailed uncomfortable procedures. To protect their health goals from the temptation of averting physical discomfort, participants agreed to pay higher monetary fines for failing to meet their screening appointment. People also will structure self-control decisions to render rewards contingent on success. In another study by Trope and Fishbach, participants preferred to delay receiving a monetary reward until after (rather than before) they completed the medical screening. By self-imposing penalties and making rewards contingent on success, these strategies served to precommit people to a course of action that served their more valued health goals.

Implementation intentions, which were discussed earlier, can be viewed as a form of prospective control in that they are designed to regulate responses to temptations proactively by

specifying action plans prior to exposure. So while self-imposed penalties and success-contingent rewards regulate responses by changing the cost–benefit contingencies of acting on temptation, implementation intentions regulate responses by automating responses to be in line with more valued ends. As reviewed earlier, research suggests that implementation intentions are indeed an effective means of countering negative influence of temptations, particularly in the health domain (e.g., Gollwitzer & Sheeran, 2006).

The Role of Construal

Another major factor that influences people’s motivations to overcome or indulge in temptations is their construal of the temptation. It is a truism of modern psychology that people’s understanding and experience of the world is subjective in nature. The same object or situation can be construed and experienced by different people in different ways or by the same person in different ways depending on the context and that person’s motivations. For example, consider a candy bar. A candy bar can be construed as “tasty snack” or as a “fattening overindulgence.” Clearly, these different construals have different evaluative connotations and suggest divergent responses to the same target. Construing the candy bar as a “tasty snack” promotes consumption and may promote self-control failure among those concerned about weight loss. By contrast, construing the candy bar as a “fattening overindulgence” promotes restraint and may promote self-control success among those concerned about weight loss. In this way, how people construe temptations may impact their self-control decisions.

Research suggests that changing people’s construals of temptations is a particularly effective strategy for dealing with and overcoming temptations. The less people incorporate the alluring and rewarding features of the temptation into their construals of it, the less likely people are to want and pursue the temptation (Mischel et al., 1989). Similarly, the more people think about the temptation in terms of the more valued ends that it threatens to undermine, the less appealing it is and the less likely they are to succumb to it (Fujita & Carnevale, 2012).

However, one of the major challenges of self-control is that the immediacy of temptations available in the here and now often prompts people to construe them in concrete ways, leading them to be viewed as an isolated opportunity for visceral, tangible, and immediate reward. Such concrete thought tends to lead people to act on these more proximal motivations and indulge in the temptation. Research suggests that key to overcoming this tendency is adopting a more abstract construal (Fujita & Carnevale, 2012). Abstract construals extract the central, stable, and enduring aspects of a target or situation. Consider a choice between a candy bar and an apple. Whereas a concrete construal might focus deliberations on the pros and cons of “this candy” vs. “that apple,” a more abstract construal focuses decision-making processes instead on the more essential, global features of the choice—namely, “hedonism” vs. “health.” In general, whereas concrete construals of temptations tend to highlight their rewarding properties, abstract construals relate temptations to a person’s more valued goals and motives. These construals in turn change the experience of temptations by changing what the temptations mean to people. Abstract relative to concrete construals help individuals transcend the pull of the immediate situation to see the bigger picture and thus to evaluate, plan, and act in accordance with more global, superordinate goals and motives. Extensive research has shown that abstract construals help people resist alluring temptations and enhance self-control (Fujita & Carnevale, 2012).

Of note, research suggests that implementation of many of the psychological mechanisms and behavioral strategies discussed earlier (i.e., cognitive habits, prospective control) may be contingent on more abstract rather than concrete construals. Abstract rather than concrete

construals of temptation, for example, lead people to associate temptations with negativity, which in turn promotes self-control (Fujita & Han, 2009). Similarly, asymmetric temptation–goal associations are more evident when people adopt more abstract versus concrete construals of temptations (Fujita & Sasota, 2011). Moreover, abstract rather than concrete construals promote the use of prospective self-control (Fujita & Roberts, 2010). Thus, people’s construal of temptations appears to represent a key variable in promoting resistance to temptations.

Importance

The ubiquity of temptations in people’s environments makes maintaining and sustaining personal health and well-being goals challenging. Although temptations can undermine health goals, people have at their disposal a wide variety of psychological tools to combat them. Indeed, research suggests that those better able to resist temptations are healthier, happier, and more well adjusted (e.g., Mischel et al., 1989). This link would thus suggest that understanding how people react to temptations and how they successfully overcome them is key to promoting and supporting health behavior change.

Author Biographies

David A. Kalkstein is a doctoral candidate in social psychology at New York University. David’s research focuses primarily on how cognitive processes of abstraction aid in self-regulation, self-control, and social exchange.

Kentaro Fujita is an associate professor of psychology at the Ohio State University. His research focuses on why people so often make decisions and behave in ways that they know are contrary to their goals and values. To understand these self-control failures, Dr. Fujita’s research draws from a number of areas in psychology, including motivation, cognition, self-regulation, and judgment and decision making.

References

- Ainslie, G. (1975). Specious reward: A behavioral theory of impulsiveness and impulse control. *Psychological Bulletin*, 82, 463–496. <http://doi.org/10.1037/h0076860>
- Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84, 191–215. <http://doi.org/10.1037/0033-295X.84.2.191>
- Baumeister, R. F., & Heatherton, T. F. (1996). Self-regulation failure: An overview. *Psychological Inquiry*, 7, 1–15. http://doi.org/10.1207/s15327965pli0701_1
- Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction*, 94, 327–340. <http://doi.org/10.1046/j.1360-0443.1999.9433273.x>
- Fishbach, A., Friedman, R. S., & Kruglanski, A. W. (2003). Leading us not into temptation: Momentary allurements elicit overriding goal activation. *Journal of Personality and Social Psychology*, 84, 296–309.
- Fishbach, A., & Shah, J. Y. (2006). Self-control in action: Implicit dispositions toward goals and away from temptations. *Journal of Personality and Social Psychology*, 90, 820–832. <http://doi.org/10.1037/0022-3514.90.5.820>
- Fujita, K. (2011). On conceptualizing self-control as more than the effortful inhibition of impulses. *Personality and Social Psychology Review*, 15, 352–366. <http://doi.org/10.1177/1088868311411165>

- Fujita, K., & Carnevale, J. J. (2012). Transcending temptation through abstraction: The role of construal level in self-control. *Current Directions in Psychological Science*, 21, 248–252. <http://doi.org/10.1177/0963721412449169>
- Fujita, K., & Han, H. A. (2009). Moving beyond deliberative control of impulses: The effect of construal levels on evaluative associations in self-control conflicts. *Psychological Science*, 20, 799–804. <http://doi.org/10.1111/j.1467-9280.2009.02372.x>
- Fujita, K., & Roberts, J. C. (2010). Promoting prospective self-control through abstraction. *Journal of Experimental Social Psychology*, 46, 1049–1054. <http://doi.org/10.1016/j.jesp.2010.05.013>
- Fujita, K., & Sasota, J. A. (2011). The effects of construal levels on asymmetric temptation-goal cognitive associations. *Social Cognition*, 29, 125–146. <http://doi.org/10.1521/soco.2011.29.2.124>
- Gollwitzer, P. M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. *Advances in Experimental Social Psychology*, 38, 69–119. [http://doi.org/10.1016/S0065-2601\(06\)38002-1](http://doi.org/10.1016/S0065-2601(06)38002-1)
- Hofmann, W., Rauch, W., & Gawronski, B. (2007). And deplete us not into temptation: Automatic attitudes, dietary restraint, and self-regulatory resources as determinants of eating behavior. *Journal of Experimental Social Psychology*, 43, 497–504. <http://doi.org/10.1016/j.jesp.2006.05.004>
- Job, V., Dweck, C. S., & Walton, G. M. (2010). Ego depletion—Is it all in your head? Implicit theories about willpower affect self-regulation. *Psychological Science*, 21, 1686–1693. <http://doi.org/10.1177/0956797610384745>
- Job, V., Walton, G. M., Bernecker, K., & Dweck, C. S. (2015). Implicit theories about willpower predict self-regulation and grades in everyday life. *Journal of Personality and Social Psychology*, 108, 637–647. <http://doi.org/10.1037/pspp0000014>
- Kelley, W. M., Wagner, D. D., & Heatherton, T. F. (2015). In search of a human self-regulation system. *Annual Review of Neuroscience*, 38, 389–411. <http://doi.org/10.1146/annurev-neuro-071013-014243>
- Masicampo, E. J., Martin, S. R., & Anderson, R. A. (2014). Understanding and overcoming self-control depletion. *Social and Personality Psychology Compass*, 8, 638–649. <http://doi.org/10.1111/spc3.12139>
- Mischel, W., Shoda, Y., & Rodriguez, M. I. (1989). Delay of gratification in children. *Science*, 244, 933–938. <http://doi.org/10.1126/science.2658056>
- Strecher, V. J., DeVellis, B. M., Becker, M. H., & Rosenstock, I. M. (1986). The role of self-efficacy in achieving health behavior change. *Health Education & Behavior*, 13(1), 73–92. <http://doi.org/10.1177/109019818601300108>
- Trope, Y., & Fishbach, A. (2000). Counteractive self-control in overcoming temptation. *Journal of Personality and Social Psychology*, 79, 493–506. <http://doi.org/10.1037/0022-3514.79.4.493>
- Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: A resource-depletion approach. *Psychological Science*, 11, 249–254. <http://doi.org/10.1111/1467-9280.00250>

Suggested Reading

- Fujita, K. (2011). On conceptualizing self-control as more than the effortful inhibition of impulses. *Personality and Social Psychology Review*, 15, 352–366. <http://dx.doi.org/10.1177/1088868311411165>
- Mann, T., de Ridder, D., & Fujita, K. (2013). Self-regulation of health behavior: Social psychological approaches to goal setting and goal striving. *Health Psychology*, 32, 487–498. <http://dx.doi.org/10.1037/a0028533>
- Mischel, W. (2014). *The marshmallow test: Mastering self-control*. New York: Little, Brown.
- Mischel, W., Shoda, Y., & Rodriguez, M. I. (1989). Delay of gratification in children. *Science*, 244, 933–938. <http://dx.doi.org/10.1126/science.2658056>

Terror Management Health Model

Patrick Boyd and Jamie L. Goldenberg

Department of Psychology, University of South Florida, Tampa, FL, USA

The terror management health model (TMHM) builds on terror management theory (TMT), a social psychology theory concerning how people manage the psychological awareness of their own mortality, to offer unique insights into how concerns about mortality affect decision making with respect to health. The model assumes that in the context of health decision making, thoughts of death are likely to be activated; before the TMHM was introduced, no theory in health psychology had been offered to account for the unique defensive responses associated with the awareness of mortality.

Origins of the TMHM

In the early 1970s, cultural anthropologist Ernest Becker (e.g., 1973) was arguing that cultures provide belief systems that enable individuals to derive meaning and a sense of personal value in the face of existential uncertainty, brought about by the awareness of death. Then, in the late 1980s, a team of social psychologists (Greenberg, Pyszczynski, & Solomon, 1986) extended Becker's theorizing and developed TMT, providing an empirical framework for Becker's ideas. The first of two primary hypotheses proposed by this research team was that if culture functions to help individuals cope with death, cultural investments (or derogation of cultural belief systems that threaten one's own) should increase when death is salient. The second hypothesis concerned self-esteem striving: because self-esteem is derived from the cultural system within which one is embedded, attempts to bolster self-esteem by living up to cultural standards should also increase in response to the awareness of death.

After a decade of research supporting TMT, the theory underwent some conceptual fine-tuning. The researchers observed that worldview defense and self-esteem striving occurred reliably when individuals were reminded of their mortality and then distracted from it, or when death was subliminally primed. A dual-defense model was proposed (Pyszczynski, Greenberg, & Solomon, 1999) to explain the time course of worldview defense and

self-esteem striving, and critically, specifying that symbolic defenses concerning meaning and self-esteem are most strongly manifested when thoughts of death are activated outside of focal awareness. Specifically, the dual-defense model explains that when death thoughts are in focal awareness, individuals focus their cognitive resources on suppressing death thoughts. After suppression, worldview defense and self-esteem striving occur to ameliorate the residue of unconscious death thoughts.

Overview of the TMHM

Jamie Goldenberg and Jamie Arndt (2008) developed the TMHM to extend TMT to the health domain—a seemingly obvious but unexplored application of TMT. The TMHM begins with the assumption that health conditions have varying potential to make people think about death. The model then provides a formal framework for explaining, predicting, and ultimately intervening in behavioral health outcomes as a function of the accessibility of death thoughts.

Integrating the insights from the dual-defense model of TMT, the TMHM specifies that when mortality concerns are conscious, health decisions will be guided by the *proximal* motivational goal of removing death-related thoughts from focal attention by reducing perceived vulnerability to the health threat. This process can entail efforts to better one's health, but perceptions of vulnerability can also be reduced through less productive means, such as denial. In contrast, when mortality concerns are active but outside of focal attention, health relevant decisions are guided by the *distal* motivational goals of bolstering self-esteem and maintaining one's symbolic conception of self. Again, the implications can be both good and bad for health (e.g., sun protection or sun tanning)—but here motivations are focused on the value and meaning of the self.

Proximal TMHM Responses

To examine responses to conscious death thoughts with experimental methods, participants are typically asked to contemplate their mortality, usually with open-ended questions (e.g., “Jot down, as specifically as you can, what you think will happen to *you* as you physically die and once you are physically dead”), and immediately thereafter health responses are assessed. Perhaps not surprisingly, under such conditions people increase their intentions to exercise (Arndt, Schimel, & Goldenberg, 2003) and protect their skin from the sun (Routledge, Arndt, & Goldenberg, 2004) and also deny their vulnerability to health risk factors (Greenberg, Arndt, Simon, Pyszczynski, & Solomon, 2000).

Moreover, supporting the assumption that such responses reflect efforts to reduce perceived vulnerability to the health threat, variables relevant to coping with the health threats have been shown to moderate health outcomes when thoughts of death are conscious. This has been demonstrated by measuring perceived vulnerability (Arndt, Cook, Goldenberg, & Cox, 2007), response efficacy (Cooper, Goldenberg, & Arndt, 2010), active coping strategies (Arndt, Routledge, & Goldenberg, 2006), and health optimism (Arndt et al., 2006; Cooper et al., 2010). These studies converge to indicate that when individuals maintain optimism about their health, perceive a health response as effective, or approach health situations with active coping strategies, they respond to conscious reminders of death with health promotion. For example, Cooper et al. (2010) found that when death was in conscious awareness, participants high in health optimism responded with increased intentions to screen for cancer, whereas individuals low in this resource decreased their intentions. Moreover, these moderating effects

were not found when death thoughts were allowed to recede from consciousness, nor were they observed in a health domain unrelated to death (i.e., cavity protection), further supporting the assumption that these defenses are aimed at managing death-related thoughts.

Distal TMHM Responses

When mortality concerns are active but outside of focal attention, the TMHM predicts that health decisions will be guided by the implications of the behavior for meaning and self-esteem rather than by the relevance for health. This research has examined the moderating potential of worldview beliefs (e.g., the effects of religious fundamentalism on prayer as a medical substitute; Vess, Arndt, Cox, Routledge, & Goldenberg, 2009) and the potential for physical inspection of the body (e.g., breast self-exams) to undermine symbolic value (e.g., Goldenberg, Arndt, Hart, & Routledge, 2008).

The lion's share of research, however, has focused on esteem contingencies. For example, in contrast to the immediate increase in sun protection intentions when death thoughts were conscious, Routledge et al. (2004) found that the women in their study (who had previously indicated being tan as relevant to their self-esteem) increased their intentions to tan when a delay followed the mortality salience prime and thoughts of death were presumably no longer in consciousness. Likewise, Arndt et al. (2003) not only found increased intentions to exercise immediately following the mortality reminder, but after a delay, participants who derived self-esteem from exercising also increased their intentions to exercise, whereas those low in fitness-contingent self-esteem did not. Similarly, Hansen, Winzeler, and Topolinski (2010) first identified individuals who derived self-esteem from smoking and then showed that they had more positive responses toward questions assessing smoking attitudes after viewing anti-smoking packaging that reminded them of their mortality. In another series of studies, women restricted their consumption of a healthy but fattening food when mortality concerns were activated, a behavioral decision presumably driven by esteem concerns rather than health (men's eating was unaffected; Goldenberg, Arndt, Hart, & Brown, 2005).

Interventional Potential of the TMHM

The TMHM framework informs what health outcomes can be expected as a function of the consciousness of death thoughts, and it also provides a framework for intervening in such outcomes. One interventional direction is to use conscious death-related thoughts to bolster the influence of more conventional health cognitions. For example, Cooper, Goldenberg, and Arndt (2014) found that presenting beach patrons with a communication that highlighted risk of death from cancer and simultaneously framed sun protection as effective resulted in increased sun protection intentions compared with when sun protection was framed as ineffective. This relationship was not observed in the non-death condition, or when a delay followed the cancer prime.

Given the finding that responses to nonconscious death thoughts depend on esteem contingencies and cultural beliefs, a complementary and more extensive wave of research targets the malleability of these bases so as to affect more productive health outcomes. In the domain of tanning, for example, Cox et al. (2009) found that participants given a mortality prime followed by a delay, who also read a fashion column touting "bronze is beautiful," increased tanning intentions, whereas participants primed with "pale is pretty" decreased intentions to tan under the same conditions. These findings were replicated among beach patrons in South

Florida who, in response to “pale is pretty” and nonconscious death thought activation, indicated that they would prefer sample lotions with higher SPFs.

Along similar lines, when thoughts of mortality are accessible but not conscious, smokers have been shown to be more persuaded by health communications highlighting the social disadvantages of being a smoker (Arndt et al., 2009), and women are more persuaded by feedback targeting appearance rather than health consequences of tanning (Morris, Cooper, Goldenberg, Arndt, & Gibbons, 2014). People are also more willing to get a flu shot when it is endorsed by a cultural icon (e.g., a celebrity) than when it is endorsed by a doctor, but only when death thoughts are outside of conscious awareness (McCabe, Vail, Arndt, & Goldenberg, 2014). This pattern is reversed when death thoughts are conscious, with participants showing greater willingness to get a flu shot when a doctor endorses it. In a naturalistic setting, supermarket shoppers were found to make healthier purchases when they were exposed to a questionnaire that nonconsciously primed mortality and then asked them to visualize a prototypical healthy eater (McCabe et al., 2015). These studies indicate that targeting social norms and bases of self-esteem can be effective routes to influencing health decisions and, importantly, that these motivations are most influential when death thoughts are activated but not conscious.

Implications and Conclusion

Integrating insights about how people manage existential concerns into a model of health decision making, the TMHM connects previously unconsidered factors influencing health decision making. Research in health psychology has identified important health cognitions (e.g., perceived vulnerability) and concerns about esteem and social norms, but until the TMHM was developed, the impact of mortality awareness on health had not been investigated. The TMHM offers a framework for understanding how conscious and nonconscious death thoughts interact with these other variables and identifies when health risk and health promotion outcomes will occur.

In addition, the TMHM offers insights that explain why some health promotion efforts fail. Consider the tactic of fear appeals. Though people may very well heed a cancer warning and put down their cigarette or lather on sunscreen, the TMHM predicts that these effects may be short lived. Once death thoughts have been suppressed, self-esteem concerns become paramount, and smoking or tanning efforts may increase to the extent that a person derives self-esteem from looking cool as a smoker or appearing bronzed and beautiful.

Since the inception of the TMHM in 2008, a growing body of support has accumulated. In addition, there is evidence documenting the effectiveness of an interventional approach informed by the TMHM. Going forward it will be important to investigate the durability of TMHM effects, as well as to continue to apply interventions utilizing the TMHM in naturalistic settings.

Author Biographies

Patrick Boyd received a BA in psychology from the University of Southern California in 2007 and an MA in social psychology from San Francisco State University in 2012. He began pursuing his PhD at the University of South Florida in 2013. His research generally focuses on terror management theory, and within this paradigm he has explored how self-worth that is contingent upon being healthful can globally predict behaviors in a variety of health contexts.

Jamie L. Goldenberg is a professor of psychology at the University of South Florida. Her area of specialization is social psychology with a focus on health behavior and women's health in particular. She is the developer of the terror management health model (TMHM) along with coauthor Arndt. Research on the TMHM has been funded for 10 years by the *National Cancer Institute (NCI)* and has resulted in dozens of publications, including a manuscript in *Psychological Review* depicting the model.

References

- Arndt, J., Cook, A., Goldenberg, J. L., & Cox, C. R. (2007). Cancer and the threat of death: The cognitive dynamics of death thought suppression and its impact on behavioral health intentions. *Journal of Personality and Social Psychology*, 92, 12–29. doi:10.1037/0022-3514.92.1.12
- Arndt, J., Cox, C. R., Goldenberg, J. L., Vess, M., Routledge, C., Cooper, D. P., & Cohen, F. (2009). Blowing in the (social) wind: Implications of extrinsic esteem contingencies for terror management and health. *Journal of Personality and Social Psychology*, 96, 1191–1205. doi:10.1037/a0015182
- Arndt, J., Routledge, C., & Goldenberg, J. L. (2006). Predicting proximal health responses to reminders of death: The influence of coping style and health optimism. *Psychology and Health*, 21, 593–614. doi:10.1080/14768320500537662
- Arndt, J., Schimel, J., & Goldenberg, J. L. (2003). Death can be good for your health: Fitness intentions as a proximal and distal defense against mortality salience. *Journal of Applied Social Psychology*, 33, 1726–1746. doi:10.1111/j.1559-1816.2003.tb01972.x
- Becker, E. (1973). *The denial of death*. New York, NY: Free Press.
- Cooper, D. P., Goldenberg, J. L., & Arndt, J. (2010). Examining the terror management health model: The interactive effect of conscious death thought and health-coping variables on decisions in potentially fatal health domains. *Personality and Social Psychology Bulletin*, 36, 937–946. doi:10.1177/0146167210370694
- Cooper, D. P., Goldenberg, J. L., & Arndt, J. (2014). Perceived efficacy, conscious fear of death and intentions to tan: Not all fear appeals are created equal. *British Journal of Health Psychology*, 19, 1–15. doi:10.1111/bjhp.12019
- Cox, C. R., Cooper, D. P., Vess, M., Arndt, J., Goldenberg, J. L., & Routledge, C. (2009). Bronze is beautiful but pale can be pretty: The effects of appearance standards and mortality salience on sun-tanning outcomes. *Health Psychology*, 28, 746–752. doi:10.1037/a0016388
- Goldenberg, J. L., & Arndt, J. (2008). The implications of death for health: A terror management health model for behavioral health promotion. *Psychological Review*, 115, 1032–1053. doi:10.1037/a0013326
- Goldenberg, J. L., Arndt, J., Hart, J., & Brown, M. (2005). Dying to be thin: The effects of mortality salience and body mass index on restricted eating among women. *Personality and Social Psychology Bulletin*, 31, 1400–1412. doi:10.1177/0146167205277207
- Goldenberg, J. L., Arndt, J., Hart, J., & Routledge, C. (2008). Uncovering an existential barrier to breast self-exam behaviors. *Journal of Experimental Social Psychology*, 44, 260–274. doi:10.1016/j.jesp.2007.05.002
- Greenberg, J., Arndt, J., Simon, L., Pyszczynski, T., & Solomon, S. (2000). Proximal and distal defenses in response to reminders of one's mortality: Evidence of a temporal sequence. *Personality and Social Psychology Bulletin*, 26, 91–99. doi:10.1177/0146167200261009
- Greenberg, J., Pyszczynski, T., & Solomon, S. (1986). The causes and consequences of a need for self-esteem: A terror management theory. In R. F. Baumeister (Ed.), *Public self and private self* (pp. 189–212). New York, NY: Springer-Verlag.
- Hansen, J., Winzeler, S., & Topolinski, S. (2010). When the death makes you smoke: A terror management perspective on the effectiveness of cigarette on-pack warnings. *Journal of Experimental Social Psychology*, 46, 226–228. doi:10.1016/j.jesp.2009.09.007

- McCabe, S., Arndt, J., Goldenberg, J. L., Vess, M., Vail, K. I., Gibbons, F. X., & Rogers, R. (2015). The effect of visualizing healthy eaters and mortality reminders on nutritious grocery purchases: An integrative terror management and prototype willingness analysis. *Health Psychology, 34*(3), 279–282. doi:10.1037/hea0000154
- McCabe, S., Vail, K. E. I. I., Arndt, J., & Goldenberg, J. L. (2014). Hails from the crypt: A terror management health model investigation of health and celebrity endorsements. *Personality and Social Psychology Bulletin, 40*, 289–300. doi:10.1177/0146167213510745
- Morris, K. L., Cooper, D. P., Goldenberg, J. L., Arndt, J., & Gibbons, F. X. (2014). Improving the efficacy of appearance-based sun exposure intervention with the terror management health model. *Psychology and Health, 29*, 1245–1264. doi:10.1080/08870446.2014.922184
- Pyszczynski, T., Greenberg, J., & Solomon, S. (1999). A dual-process model of defense against conscious and unconscious death-related thoughts: An extension of terror management theory. *Psychological Review, 106*, 835–845. doi:10.1037/0033-295X.106.4.835
- Routledge, C., Arndt, J., & Goldenberg, J. L. (2004). A time to tan: Proximal and distal effects of mortality salience on sun exposure intentions. *Personality and Social Psychology Bulletin, 30*, 1347–1358. doi:10.1177/0146167204264056
- Vess, M., Arndt, J., Cox, C. R., Routledge, C., & Goldenberg, J. L. (2009). Exploring the existential function of religion: The effect of religious fundamentalism and mortality salience on faith-based medical refusals. *Journal of Personality and Social Psychology, 97*, 334–350. doi:10.1037/a0015545

Suggested Reading

- Goldenberg, J. L., & Arndt, J. (2008). The implications of death for health: A terror management health model for behavioral health promotion. *Psychological Review, 115*, 1032–1053. doi:10.1037/a0013326
- Pyszczynski, T., Greenberg, J., & Solomon, S. (1999). A dual-process model of defense against conscious and unconscious death-related thoughts: An extension of terror management theory. *Psychological Review, 106*, 835–845. doi:10.1037/0033-295X.106.4.835
- Routledge, C., Arndt, J., & Goldenberg, J. L. (2004). A time to tan: Proximal and distal effects of mortality salience on sun exposure intentions. *Personality and Social Psychology Bulletin, 30*, 1347–1358. doi:10.1177/0146167204264056

The Role of Persuasion in Health-Related Attitude and Behavior Change

Colin A. Zestcott and Jeff Stone

Psychology Department, University of Arizona, Tucson, AZ, USA

Persuasion processes play a central role in the public's health. Most health professionals, from doctors and nurses in a clinic to public health officials, rely on persuasion to prevent disease and illness and to gain compliance with health recommendations. Achieving these public health goals often requires getting people to change their health-related attitudes and behaviors. The goal of this entry is to review the major techniques for persuading health attitude and behavior change and the psychological processes that determine how and when people make changes.

For the purposes of this entry, we define *persuasion* as any attempt by an external source, or by the individual, to change a health-related attitude, behavioral intention, or behavior. Research on persuasion and health tends to target two types of behavioral outcomes: changing behavioral intentions, defined as a person's deliberate plan to adopt a specific health behavior, and changing the health behavior directly. Thus, the present entry will cover both types of behavioral outcomes. In addition, while many persuasive strategies are designed to change an individual's behavior, theories in psychology such as the theory of planned behavior illuminate the importance of understanding how attitudes guide future intentions and behavior. Thus, focusing solely on behavior as a main outcome of persuasion techniques in a health context may fail to provide a thorough understanding of the underlying mechanisms, such as the importance of attitude change, when evaluating the effectiveness of health behavior interventions.

In line with the broader literature, persuasion processes that target health attitude change tend to define *attitudes* as an evaluation of an object. According to Eagly and Chaiken (1993), an attitude is "a psychological tendency that is expressed by evaluating a particular entity with some degree of favor or disfavor" (p. 1). However, contemporary research in psychology also delineates between explicit, or directly measured, attitudes and implicit, or indirectly measured, attitudes. While explicitly measured attitudes are primarily based on self-report, most implicit attitudes are measured via reaction time tasks. There remains considerable debate about the psychological nature of implicit attitudes and the reliability and validity of measures

like the Implicit Association Test. Nevertheless, because research on the role of implicit attitudes in health has greatly expanded in the past decade, we believe this entry would be incomplete if it did not also examine implicit attitude change.

Finally, the literature suggests that the source of a persuasive message can stem from external or internal sources. External sources for persuasive messages can include mass media public health campaigns, warning labels on cigarette packs, or a doctor's advice to get more exercise. In contrast, the individual may serve as the source of persuasion in that individuals may think or act in ways that motivate change in health-related attitudes or behavior (Aronson, 1999). This entry will address external versus internal sources of persuasion separately.

Persuasion to Change Explicit Health-Related Attitudes and Behaviors

In the early 1950s, the experimental examination of persuasion began to focus on the ways in which external message sources, message recipients, and the content of the message contributed to attitude change. Many of the early studies examined communication strategies designed to change health-related attitudes and behaviors.

Role of Fear Appeals in Persuasion

The formal study of health persuasion in psychology began with research on fear to motivate health-related attitude and behavior change. When facing a fear appeal, individuals are exposed to a fear-inducing message that is perceived as a serious threat to one's health or well-being. Based on drive model theorizing (Hovland, Janis, & Kelly, 1953), the unpleasant emotional state of experiencing fear motivates individuals to alleviate or eliminate the source of the threat. Meta-analyses covering a range of health issues such as exercise, sunscreen use, and cigarette smoking suggest that fear appeals can change an individual's attitudes, behavioral intentions, and behaviors to be more favorable toward the health topic (e.g. Witte & Allen, 2000). According to the drive model, fear appeals effectively reduce defensiveness and reactance to a message if an individual is provided with information that moves them toward the intended outcome. Consequently, fear appeals are most effective at changing health-related attitudes and behavior when recipients believe the threat of the message is severe and relevant (i.e., recipients believe they are vulnerable), the message provides an effective solution to reduce or eliminate the threat, and recipients perceive that they possess efficacy to mitigate the threat (Witte & Allen, 2000).

The Elaboration Likelihood Model

While early health persuasion techniques focused on the role that motivation can play in changing health attitudes and behavior, subsequent research took a broader approach to the concept of motivation and at the same time explored the possibility that people could be persuaded to change their health attitudes and behaviors when their motivation was low. The development of a "dual-process" approach to persuasion led to novel predictions regarding the factors that affect the persistence of health attitude and behavior change.

One of the most influential dual-process theories of persuasion is the elaboration likelihood model (ELM; Petty & Cacioppo, 1986). Based on the cognitive response approach to attitude change, the ELM stipulates that persuasion occurs along a continuum of elaboration or thinking. On the high end of the elaboration continuum, often referred to as the "central route,"

persuasion occurs when people generate relatively many positive thoughts after thinking carefully about the content of the message. In order to think carefully about the message, people must be motivated and have the ability to think about the information in the communication. In addition, in order to assure that a highly thoughtful audience generates positive responses to the message, the content of the message must contain strong arguments; otherwise, the audience will counterargue and dismiss the information. Individual difference variables, such as increased involvement with an issue and increased need for cognition, are also associated with more central route processing (Petty & Cacioppo, 1986). Attitude change that follows from careful scrutiny of a strong message tends to endure over time and is more highly predictive of subsequent behavior, which is an important outcome when the goal is to alter future health outcomes.

Research in the ELM tradition also shows that people need not think carefully about the message in order for a message to be persuasive. On the lower end of the elaboration continuum, where people are paying attention but not scrutinizing the arguments in the message, factors that work on the periphery of the message can cause attitude change. The “peripheral route” to persuasion occurs when people either do not care about the message content or care but are unable to process the information. Persuasion in the absence of much thinking occurs when the message and its delivery include simple cues (e.g. “he has a nice smile”; “she is really funny”) or heuristics (e.g. “he graduated from Harvard”; “she is a doctor”) that lead to a positive conclusion about the message without considering the arguments. Whereas new attitudes formed through the peripheral route do not last long nor predict future behavior, such outcomes may not be relevant if the goal is to achieve a momentary attitude and behavior shift, like when trying to sign someone up for a new health club membership.

The principles stipulated by the ELM are useful for understanding multiple processes by which health attitudes and behavior change. For example, research on AIDS prevention by Bakker (1999) shows that adolescents high in need for cognition report more positive attitudes toward using condoms in the future if they are exposed to a clear and concise written brochure compared with a humorous cartoon brochure with the same information. However, this pattern reversed for those low in need for cognition such that their knowledge and behavioral intentions to use condoms increased when viewing the cartoon brochure rather than the written brochure.

The ELM also provides an explanation for why health campaigns show greater success when they tailor and target their messages to specific audience characteristics. Tailoring uses information about an individual to determine the content and delivery that will gain the most attention. Targeting refers to customizing messages for broader population subgroups that share certain characteristics. Consistent with the message-matching application of the ELM, research shows that tailored messages increase positive thoughts and personal connections about weight-loss information (Kreuter, Bull, Clark, & Oswald, 1999).

Self-Affirmation

One limitation to using threat, such as fear appeals, to motivate health behavior change is that in some situations, people cope with the threat by denying that the health issue applies to them and avoiding the issue altogether. One way to reduce denial and avoidance responses is by providing a self-affirming buffer. According to self-affirmation theory, individuals can bolster the positive integrity of the self-system by reflecting upon their most cherished positive values, actions, or attributes (Steele, 1988). Consequently, affirming values boosts the psychological resources needed to manage a threat and leads to a separation (i.e. a buffer) of the threat from the self.

Studies on self-affirmation and health show that participants who self-affirm, typically in the form of reflecting on important values, are more likely to change their health-related attitudes and behavior (see Jessop & Harris, this volume). For example, females who suntan and self-affirm report more positive attitudes toward sunscreen and are more likely to acquire a sunscreen sample than those who do not self-affirm after reading a pamphlet about the risks of sun exposure (Jessop, Simmons, & Sparks, 2009). Other studies show that individuals who self-affirm are more likely to accept threatening health risk information and are less likely to derogate a message than those who do not self-affirm (Jessop et al., 2009).

Persuasion to Change Implicit Health-Related Attitudes

Research over the past two decades in psychology has examined the ways in which implicit, or introspectively inaccessible constructs, can be measured to assess topics such as prejudice, stereotyping, and self-esteem. While most research in the health domain assesses explicit attitudes, other research suggests that self-presentational concerns may cause a dissociation between an individual's implicit and explicit attitudes (e.g. Fazio & Dunton, 1997). For example, an individual who enjoys suntanning may express positive explicit attitudes toward sunscreen to others yet still hold negative implicit attitudes toward sunscreen. Consequently, implicit attitudes may be a better predictor of health-related attitudes compared with explicit attitudes.

The most common way to examine implicit attitudes an individual may hold is through indirect measures that assess reaction times and categorization biases. Using reaction time procedures, research shows that implicit attitudes may be more predictive of behavior than explicit attitudes. For example, researched by Sherman, Chassin, Presson, Seo, and Macy (2009) shows that implicit attitudes toward cigarette smoking are more predictive of later smoking than explicit attitudes.

If implicit attitudes are more predictive of health behavior than explicit attitudes, it is important to determine the processes by which implicit attitudes can change. For example, persuasive campaigns that describe the negative health effects of drugs like marijuana and tobacco induce more negative implicit attitudes toward the drugs by creating stronger associations between drug-related images and negative words (Czyzewska & Ginsburg, 2007). The context in which information is presented may also affect implicit attitudes. Sherman, Rose, Koch, Presson, and Chassin (2003) show that individuals express more negative implicit attitudes toward smoking when the presentation highlights informational aspects of smoking (e.g., pictures of cigarette packs) compared with sensory aspects (e.g., a cigarette burning in an ashtray). Directly changing associative pairings can also affect implicit attitudes. For example, participants in an evaluative conditioning paradigm who paired images of unhealthy snack foods with aversive health consequences were subsequently faster to associate negative words with snack-related words and positive words with fruit-related words (Hollands, Prestwich, & Marteau, 2011).

Self-Persuasion to Change Health-Related Attitudes and Behavior

A parallel tradition in the study of persuasion in health focuses on the processes by which people convince themselves to change their health-related attitudes and behavior. According to Aronson (1999), self-persuasion occurs when individuals take a certain course of action and, as a result, change their health-related attitudes and future behaviors. Thus, the primary source

of influence to pursue health-related attitude or behavior change stems from the individual rather than an external source. Researchers interested in the health changes that people make for themselves have primarily focused on the role of cognitive dissonance and self-perception processes.

Cognitive Dissonance Theory

Cognitive dissonance theory stipulates that individuals feel an unpleasant state of tension when they possess two inconsistent cognitions, which motivates them to reduce or eliminate the inconsistency (Festinger, 1957). For example, when people who smoke cigarettes are exposed to information regarding the negative health effects of smoking, the inconsistency between their smoking behavior and knowledge of the health risks can induce a state of psychological discomfort, which they become motivated to reduce. Smokers may alleviate the inconsistency in a number of ways, such as changing the behavioral cognition (e.g., quitting smoking), changing their knowledge about the risks of smoking (e.g., challenging the research on the relationship between smoking and cancer), or by recruiting new cognitions that place the inconsistency in a larger framework of consistency (e.g., "I would quit, but then I will gain weight, and that is worse"). Thus, the key to successful intervention rests in channeling dissonance reduction toward the targeted health attitude or behavior change.

One way to use cognitive dissonance to motivate people to change their attitudes toward unhealthy behavior is to have them argue against performing the behavior that they wish to change. A well-established example of this approach involves women's perceptions of their bodies and the thin ideal imposed by many societies. Stice and colleagues (for a review, see Stice, Shaw, Becker, & Rohde, 2008) predict that women who voluntarily adopt an anti-thin position in a group should perceive inconsistency between their positive attitude toward the thin ideal and their present behavior of taking an anti-thin stance during the small group discussion. Several studies by Stice and colleagues show that following the intervention, women reported more positive body images, reduction in the thin-ideal internalization and body dissatisfaction, less negative affect, less bulimic symptoms, and less eating disorder risk factors compared with control conditions.

Another way to use dissonance to motivate change is through an act of hypocrisy. In the hypocrisy approach, individuals first advocate for a health behavior about which they hold a positive attitude. Dissonance occurs when they are then reminded of times they failed to perform the advocated behavior in the past. The inconsistency between their advocacy about the importance of the target health behavior, and their past failures to perform the target act, makes people aware that they have acted in an insincere fashion. In order to reduce the dissonance and restore their self-integrity, people become motivated to change their own health behavior so they can bring it into line with the good health advice they were willing to give others.

In studies designed to motivate sexually active college students to practice safer sex, individuals who videotaped a speech about the importance of using of condoms, and were then reminded of past failures to use condoms during sexual intercourse, were more likely to purchase condoms at the completion of the study, compared with control conditions (Stone, Aronson, Crain, Winslow, & Fried, 1994).

Self-Perception Processes

Whereas dissonance processes change attitudes and behavior by getting people to act in ways that causes psychological discomfort, it is also possible to promote more healthy attitudes and

behaviors by getting people to act in ways that induce them to draw a new conclusion about their own behavior. According to self-perception theory, when people do not already hold an attitude toward an object, they can infer their attitude through an examination of their own behavioral interactions with the object (Bem, 1972). People then develop new positive attitudes toward the topic, which can subsequently guide new behavior.

One way in which self-perception can cause health-related attitude and behavior change is through the foot-in-the-door (FITD) technique. The FITD predicts that when an individual agrees to a request that is easy to comply with, they are more likely to comply with a second, more difficult request (e.g., Freedman & Fraser, 1966). As a result of complying with the second request, people infer that their behavior must reflect a positive attitude toward the issue or object. In the domain of health, studies show that individuals who complete a questionnaire about organ donation express greater willingness to be an organ donor compared with those that do not complete the initial questionnaire (e.g. Carducci & Deuser, 1984).

Conclusion and Future Considerations

The research in this entry indicates that there are a number of mechanisms that effectively persuade people to adopt new attitudes and behaviors that support good health. However, it is important to note that few of the findings above show evidence for changes in health attitudes and behaviors that last beyond the immediate experimental context; with a few notable exceptions (e.g., Stice et al., 2008), most of the studies in the health persuasion literature focus more on questions about basic process than on the critical question of what is necessary to create lasting attitude and behavior change. Thus, at present, the utility of the health persuasion literature for use by public health officials, healthcare providers, and educational institutions is not well understood. The question of when and how health persuasion processes lead people to maintain new changes over time deserves more empirical attention.

Author Biographies

Colin A. Zestcott, MA, is a doctoral graduate student in the Department of Psychology at the University of Arizona. His research interests include stereotyping and prejudice, implicit attitudes, and the function of metaphor in social cognition.

Jeff Stone, PhD, is a professor of psychology in the College of Science, and professor of psychiatry in the College of Medicine, at the University of Arizona. Dr. Stone's research investigates mechanisms of health attitude and behavior change, prejudice and stereotyping, stereotype threat processes, and the reduction of intergroup bias. Dr. Stone's current NIH-funded research focuses on documenting and reducing the implicit bias processes that contribute directly to ethnic and racial disparities in health.

References

- Aronson, E. (1999). The power of self-persuasion. *American Psychologist*, 54, 875–884. doi:10.1037/h0088188
- Bakker, A. B. (1999). Persuasive communication about AIDS prevention: Need for cognition determines the impact of message format. *AIDS Education and Prevention*, 11, 150–162.

- Bem, D. J. (1972). Self-perception theory. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 6, pp. 1–62). New York, NY: Academic.
- Carducci, B. J., & Deuser, F. S. (1984). The foot-in-the door technique: Initial request and organ donation. *Basic and Applied Social Psychology*, 5, 75–81. doi:10.1207/s15324834basp0501_S
- Czyzewska, M., & Ginsburg, H. J. (2007). Explicit and implicit effects of anti-marijuana and anti-tobacco TV advertisements. *Addictive Behavior*, 32, 114–127. doi:10.1016/j.addbeh.2006.03.025
- Eagly, A. H., & Chaiken, S. (1993). *The psychology of attitudes*. Orlando, FL: Harcourt Brace Jovanovich College Publishers.
- Fazio, R. H., & Dunton, B. C. (1997). Categorization by race: The impact of automatic and controlled components of racial prejudice. *Journal of Experimental Social Psychology*, 33, 451–470. doi:10.1006/jesp.1997.1330
- Festinger, L. (1957). *A theory of cognitive dissonance*. Stanford, CA: Stanford University Press.
- Freedman, J. L., & Fraser, S. C. (1966). Compliance without pressure: The foot-in-the-door technique. *Journal of Personality and Social Psychology*, 4, 195–202. doi:10.1037/h0023552
- Hollands, G. J., Prestwich, A., & Marteau, T. M. (2011). Using aversive images to enhance healthy food choices and implicit attitudes: An experimental test of evaluative conditioning. *Health Psychology*, 30, 195–203. doi:10.1037/a0022261
- Hovland, C., Janis, I., & Kelly, H. (1953). *Communication and persuasion*. New Haven, CT: Yale University Press.
- Jessop, D. C., Simmonds, L. V., & Sparks, P. (2009). Motivational and behavioral consequences of self-affirmation interventions: A study of sunscreen use among women. *Psychology and Health*, 24, 529–544. doi:10.1080/08870440801930320
- Kreuter, M. W., Bull, F. C., Clark, E. M., & Oswald, D. L. (1999). Understanding how people process health information: A comparison of tailored and nontailored weight-loss materials. *Health Psychology*, 18, 487–494. doi:10.1037/0278-6133.18.5.487
- Petty, R. E., & Cacioppo, J. T. (1986). The elaboration likelihood model of persuasion. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 19, pp. 123–205). New York, NY: Academic Press.
- Sherman, S. J., Chassin, L., Presson, C., Seo, D., & Macy, J. (2009). The intergenerational transmission of implicit and explicit attitudes toward smoking: Predicting adolescent smoking initiation. *Journal of Experimental Social Psychology*, 45, 313–319. doi:10.1016/j.jesp.2008.09.012
- Sherman, S. J., Rose, J. S., Koch, K., Presson, C. C., & Chassin, L. (2003). Implicit and explicit attitudes toward cigarette smoking: The effects of context and motivation. *Journal of Social and Clinical Psychology*, 23, 13–39. doi:10.1521/jscp.22.1.13.22/66
- Steele, C. M. (1988). The psychology of self-affirmation: Sustaining the integrity of the self. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 21, pp. 261–302). New York, NY: Academic Press.
- Stice, E., Shaw, H., Becker, C. B., & Rohde, P. (2008). Dissonance-based interventions for the prevention of eating disorders: Using persuasion principles to promote health. *Prevention Science*, 9, 114–128. doi:10.1007/s11121-008-0093-x
- Stone, J., Aronson, E., Crain, A. L., Winslow, M. P., & Fried, C. B. (1994). Inducing hypocrisy as a means of encouraging young adults to use condoms. *Personality and Social Psychology Bulletin*, 20, 116–128. doi:10.1177/0146167294201012
- Witte, K., & Allen, M. (2000). A meta-analysis of fear appeals: Implications for effective public health campaigns. *Health Education and Behavior*, 27, 591–615. doi:10.1177/109019810002700506

Suggested Reading

- Mongeau, P. A. (2013). Fear appeals. In J. P. Dillard & L. Shen (Eds.), *The SAGE handbook of persuasion* (2nd ed., pp. 184–199). Thousand Oaks, CA: SAGE Publications.

- Petty, R. E., Barden, J., & Wheeler, S. C. (2009). The elaboration likelihood model of persuasion: Health promotions that yield sustained behavioral change. In R. J. DiClemente, R. A. Crosby, & M. Kegler (Eds.), *Emerging theories in health promotion practice and research* (2nd ed., pp. 185–214). San Francisco, CA: Jossey-Bass.
- Stone, J., & Focella, E. (2011). Hypocrisy, dissonance and the self-regulation processes that improve health. *Self and Identity*, 10, 295–303. doi:10.1080/15298868.2010.538550

The Theories of Reasoned Action and Planned Behavior

Christina Nisson and Allison Earl

Department of Psychology, University of Michigan, Ann Arbor, MI, USA

The ability to understand the fundamental causes of public health problems and develop interventions to address those problems is important to a range of researchers involved in the study of health psychology. Two of the most widely tested models of this nature are the theory of reasoned action (Ajzen & Fishbein, 2005; Fishbein & Ajzen, 1975) and the theory of planned behavior (Ajzen, 1991; Ajzen & Fishbein, 1980, 2005). The theory of reasoned action posits that behavior is a function of behavioral intentions that are, in turn, a function of attitudes and subjective norms. The theory of planned behavior took the components of the theory of reasoned action but added perceived behavioral control as an additional factor predicting both behavioral intentions and behavior. In recent years, these models have been collapsed under the umbrella of the reasoned action approach.

Description of the Models

Theory of Reasoned Action

Both the theory of reasoned action and the theory of planned behavior developed out of a theoretical tradition that considered attitudes as a major influence on human behavior (e.g., Smith, 1932; Stagner, 1942). However, other contradictory research emerged suggesting that the link between attitudes and behavior was tenuous at best (e.g., Corey, 1937; LaPiere, 1934), with some researchers even calling for abandonment of the attitude construct altogether (Wicker, 1969). However, Fishbein and Ajzen (1974) noted that the inconsistency between attitudes and behaviors could be improved by measuring attitudes and behaviors at the same level of specificity. Thus, rather than using global attitudes (e.g., attitudes toward condoms) to predict specific behaviors (e.g., condom use during the next sexual encounter), Fishbein and Ajzen (1975) posited that researchers should focus on the specific antecedents of

specific behaviors (e.g., attitudes toward condom use during the next sexual encounter predicting condom use during the next sexual encounter).

Furthermore, Fishbein and Ajzen (1975) posited that the link between attitudes and behavior might best be explained by an appeal to specific behavioral intentions. That is, attitudes about performing a behavior would predict behavioral intentions to enact the behavior, which would in turn predict behavior. In addition, because one may also take into account how others perceive one's actions, subjective norms about how to behave were also included as a predictor of behavioral intentions. Behavioral intentions were then identified as the best predictor of behavior (Fishbein & Ajzen, 1975). In this model, *attitudes toward the behavior* were defined as an aggregate of readily accessible or salient beliefs about the likely outcomes of performing the target behavior, whereas *subjective norms* were defined as the perceived social pressure to perform or not perform the target behavior and *behavioral intentions* were defined as the perceived likelihood of performing the target behavior.

Theory of Planned Behavior

One factor that may limit the translation of intentions to behavior is one's ability to enact the desired behavior. As such, the theory of planned behavior updated the theory of reasoned action to include a component of *perceived behavioral control*, which specifies one's perceived ability to enact the target behavior. In fact, perceived behavioral control was added to the model to extend its applicability beyond purely volitional behaviors. Prior to this addition, the model was relatively unsuccessful at predicting behaviors that were not mainly under volitional control. Thus, the theory of planned behavior proposed that the primary determinants of behavior are an individual's behavioral intention and perceived behavioral control.

As such, according to the theory of planned behavior, behavioral intentions are framed as the motivational component of the model, or one's conscious plan or decision to exert effort to perform the target behavior. Behavioral intentions are determined by *attitudes toward the behavior* (e.g., whether engaging in the behavior is evaluated to be positive or negative), *subjective norms* surrounding the behavior (e.g., beliefs about whether others think one should engage in the behavior), and *perceived behavioral control* (e.g., beliefs regarding how easy or difficult performing the behavior is likely to be). In this context, perceived behavioral control reflects both external factors (e.g., availability of time or money, social support) and internal factors (e.g., ability, skill information). In other words, low perceived behavioral control exists in situations in which performance of the target behavior is dependent upon a number of other factors, which may or may not be within an individual's control. For example, one may experience low perceived behavioral control for the target behavior of eating healthy if constraints such as time, affordability, access, and temptation are viewed as obstacles to engage in the behavior despite strong intentions. As a result, the greater the perceived behavioral control for a target behavior, the stronger the predictive power of behavioral intentions for that behavior.

Reasoned Action Approach

In recent years, the theory of reasoned action and the theory of planned behavior have fallen under the umbrella of the *reasoned action approach* (Ajzen & Albarracín, 2007; Fishbein & Ajzen, 2010). The reasoned action approach encompasses all of the components proposed by earlier models (e.g., attitudes toward the behavior, subjective norms, perceived behavioral control, and intentions) while also including additional factors such as *actual control*, defined

as skills, abilities, and environmental factors that influence one's ability to enact a target behavior. As such, the theory of reasoned action and the theory of planned behavior will be referred to jointly as the reasoned action approach throughout this entry.

Ability to Predict Health Intentions and Behaviors

A series of meta-analyses and reviews examining the application of the reasoned action approach to health behaviors have now been published, including ones focusing on multiple health domains (e.g., Armitage & Connor, 1999; Godin & Kok, 1996; McEachan, Conner, Taylor, & Lawton, 2011; Webb, Joseph, Yardley, & Michie, 2010) and ones focusing on specific behaviors (e.g., exercise; Hagger, Chatzisarantis, & Biddle, 2002; condom use; Albarracín, Johnson, Fishbein, & Muellerleile, 2001). Such reviews have shown the reasoned action approach to be a relatively successful predictor of health intentions and behavior, explaining 32–44% of the variance in intentions and 15–41% of the variance in behavior.

One important moderator of the predictive ability of the reasoned action approach is behavior type. For instance, the reasoned action approach appears to be particularly successful in the prediction of diet and exercise behaviors, as well as condom use. In a recent comprehensive meta-analysis, McEachan et al. (2011) found that the reasoned action approach was able to explain 21 and 24% of the variance in dietary and exercise behaviors, respectively. At the same time, the reasoned action approach appears to be less successful at explaining the variance in addictive and clinical screening/detection behaviors. The same meta-analysis revealed that the reasoned action approach was able to explain only 15 and 14% of the variance in such behaviors, respectively. It is not surprising that the reasoned action approach is better at predicting some behaviors than others. Looking at the categories in which the reasoned action approach is more versus less successful in predicting behavior, it follows that the model is less predictive of addictive and clinical screening behaviors, as these behaviors are likely to be low in perceived and actual behavior control, affected not only by personal motivation and desire but also other factors (e.g., biological aspects of addiction, access to treatment and health services, financial resources to engage in screening behaviors).

Along with behavior type, there are also two important methodological moderators to consider when examining the ability of the reasoned action approach to predict health behavior: length of follow-up and method of measurement (objective vs. self-report). The amount of time between measurement of reasoned action approach variables and assessment of behavior is an inherent limiting condition of the reasoned action approach. Ajzen and Fishbein have repeatedly stressed that the measurement of behavior should occur as close as possible to the measurement of the reasoned action approach variables, as the model is only able to predict behavior to the extent that the reasoned action approach variables remain consistent from the time of measurement to the time of assessment of behavior (Ajzen, 1985; Ajzen & Fishbein, 1980). Given that it is less likely for the reasoned action approach variables to remain stable as the length of follow-up increases, it is not surprising that the overall ability of the model to predict health behavior decreases over time (Albarracín et al., 2001; McEachan et al., 2011).¹ For dietary and exercise behaviors, McEachan et al. (2011) found that the reasoned action approach was only able to predict 18% (dietary) and 16% (exercise) of the variance in behavior when follow-up occurred more than 5 weeks after initial measurement, compared with 23% (dietary) and 32% (exercise) when follow-up was less than 5 weeks after initial measurement.

Like many models of human behavior, a long-standing concern regarding the ability of the reasoned action approach to predict health behavior, and behavior in general, is its

frequent reliance on self-reported measures of behavior. A primary concern regarding reliance on self-reported behavior is the vulnerability of self-report measures to social desirability bias, or the tendency to over-report desirable behavior and underreport undesirable behavior (Edwards, 1953; Schroder, Carey, & Vanable, 2003). This may be particularly problematic for the prediction of health behavior given the tendency for many health behaviors to be viewed as either desirable (e.g., healthy eating, exercise, condom use) or undesirable (e.g., drug and alcohol use). Given the prominent role of attitudes in the reasoned action approach, individuals' inclination to maintain attitudinal and behavioral consistency (Hessing, Elffers, & Weigel, 1988; Kiesler, 1971) is also of particular concern. Thus, the model may overstate the intention-behavior relation due to individual's desire to maintain consistency in their reported intentions and behaviors. Two large meta-analyses support this concern. Armitage and Conner (1999) found that reasoned action approach explained 31% of the variance in self-reported behavior but only 20% of the variance when behavior was directly observed. Specific to health behavior, McEachan et al. (2011) found that reasoned action approach variables explained 26% of the variance in self-reported physical activity but only 12% of the variance in objectively measured physical activity. Although the reasoned action approach is able to predict a significant amount of variance in behavior regardless of length of follow-up or method of measurement, it consistently shows greater efficacy in situations with short follow-up periods and self-reported measurement of behavior.

Interventions to Change Behavior

Although the reasoned action approach was originally presented as a tool to "understand" and "predict" behavior (Ajzen & Fishbein, 1980), there is growing interest in the theory's possible utility in designing behavioral interventions. Ajzen and Fishbein (2005) agree that successful modification of predictors specified by the reasoned action approach should lead to a corresponding change in behavior. McEachan et al. (2011) found encouraging evidence for the model's ability to identify important targets for interventions to change health behaviors. Although their meta-analysis of health behaviors found that past behavior exhibited the strongest correlation with current behavior (mean $\rho = 0.50$),² intention was also a strong predictor of behavior (mean $\rho = 0.43$) and remained so after controlling for past behavior. From a behavioral intervention perspective, intentions are more relevant than past behavior as they are susceptible to change, whereas past behavior is not. Thus, it is encouraging that intentions remain a strong predictor of behavior even when controlling for past behavior (McEachan et al., 2011).

In 2002, Hardeman and colleagues published a review of behavior change interventions using the reasoned action approach (Hardeman et al., 2002). The review identified 21 interventions targeting health-related behaviors, including smoking cessation, exercise, and testicular self-examination. Of the 21 interventions identified, only 10 actually used the reasoned action approach to develop the intervention; the remaining 11 interventions simply used the reasoned action approach for measurement and therefore should not be considered a valid assessment of the theory's ability to help change behavior. It is important to note that even the 10 theory-based interventions often focused on selected reasoned action approach components only. Furthermore, the descriptions of the interventions were limited, and it was often difficult to assess the specific manner in which the reasoned action approach informed the intervention design. Nonetheless, among the 10 theory-driven interventions, 4 (40%) reported

a positive change in behavioral intentions as a result of the intervention, 3 (30%) reported no difference in behavioral intentions, and 3 (30%) did not measure intentions. In terms of behavior change, three (30%) of the interventions reported a positive change in behavior as a result of the intervention, two (20%) reported no difference in behavior, and five (50%) did not measure behavior.

In the domain of condom use, Albarracín and colleagues (Albarracín et al., 2005) examined the efficacy of the reasoned action approach by examining the link between intervention components and change in model constructs, as well as the links with behavior change. In line with the reasoned action approach, attitudinal arguments about condom use did indeed change attitudes toward condom use, which in turn led to changes in condom use. In addition, self-management training led to changes in perceived behavioral control, which in turn facilitated change in condom use (Albarracín et al., 2005).

Although it may be argued that the mixed results of the Hardeman et al. (2002) review suggest that the reasoned action approach is far better suited to predict behavior than to help change it, it is important to remember that there were only 10 studies in which the reasoned action approach was used to develop the intervention, the interventions often focused only on selected reasoned action approach components, the descriptions of the interventions were limited, and evidence about mediation of effects by reasoned action approach components was rare. McEachan and colleagues' (2011) finding that behavioral intentions remain a significant predictor of behavior even when controlling for past behavior, combined with evidence indicating that a change in individuals' beliefs can bring about changes in attitudes, intentions, and behavior (Ajzen & Fishbein, 2005), lends support to the potential for the reasoned action approach to inform behavioral change interventions.

Author Biographies

Christina Nisson received her BA in psychology and economics from Cornell University in 2009 and her MS and PhD in social psychology from the University of Michigan in 2011 and 2014, respectively. Broadly, Christina is interested in applying social psychological research to health messaging and health behaviors. Her primary line of research examines the role of health message characteristics (e.g., approach vs. avoidance goals; action vs. inaction orientation) on message processing and healthy eating behaviors.

Allison Earl received bachelor's degrees in anthropology and psychology from the University of Florida and a PhD in psychology from the University of Illinois. She is an assistant professor of social psychology at the University of Michigan and director of the Health, Attitudes, and Influence Lab (HAILab). Dr. Earl's research program focuses on understanding what we pay attention to and why and how to best increase attention to health promotion programs, particularly for high-risk audiences.

Notes

- 1 Randall and Wolff (1994) conducted a meta-analysis of 98 studies and found no significant relationship between length of follow-up and the strength of the intention-behavior relationship. It is important to note, however, that this meta-analysis did not focus specifically on health behavior and only looked at the correlation between intentions and behavior, not the overall predictive power of the model.
- 2 McEachan et al. (2011) used mean true score correlations corrected for sampling and measurement error (mean ρ).

References

- Ajzen, I. (1985). From intention to actions: A theory of planned behavior. In J. Kuhl & J. Bechmann (Eds.), *Action control: From cognitions to behavior* (pp. 11–39). New York, NY: Springer-Verlag.
- Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, 50, 179–211.
- Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs, NJ: Prentice-Hall.
- Ajzen, I., & Fishbein, M. (2005). The influence of attitudes on behavior. In D. Albarracín, B. T. Johnson, & M. P. Zanna (Eds.), *The handbook of attitudes* (pp. 173–222). Mahwah, NJ: Lawrence Erlbaum Associates.
- Albarracín, D., Gillette, J., Earl, A., Glasman, L. R., Durantini, M. R., & Ho, M. H. (2005). A test of major assumptions about behavior change: A comprehensive look at HIV prevention interventions since the beginning of the epidemic. *Psychological Bulletin*, 131, 856–897.
- Albarracín, D., Johnson, B. T., Fishbein, M., & Muellerleile, P. A. (2001). Theories of reasoned action and planned behavior as models of condom use: A meta-analysis. *Psychological Bulletin*, 127, 142–161.
- Armitage, C. J., & Conner, M. (1999). Distinguishing perceptions of control from self-efficacy: Predicting consumption of a low-fat diet using the theory of planned behavior. *Journal of Applied Social Psychology*, 29, 72–90.
- Corey, S. M. (1937). Professed attitudes and actual behavior. *Journal of Educational Psychology*, 28, 271–280.
- Edwards, A. L. (1953). The relationship between the judged desirability of a trait and the probability that the trait will be endorsed. *Journal of Applied Psychology*, 37(2), 90–93.
- Fishbein, M., & Ajzen, I. (1974). Attitudes towards objects as predictors of single and multiple behavioral criteria. *Psychological Review*, 81, 59–74.
- Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention and behavior: An introduction to theory and research*. Reading, MA: Addison Wesley.
- Fishbein, M., & Ajzen, I. (2010). *Predicting and changing behavior: The reasoned action approach*. New York, NY: Taylor & Francis.
- Godin, G., & Kok, G. (1996). The theory of planned behavior: A review of its applications to health-related behaviors. *American Journal of Health Promotion*, 11, 87–98.
- Hagger, M. S., Chatzisarantis, N. L. D., & Biddle, S. J. H. (2002). A meta-analytic review of the theories of reasoned action and planned behavior in physical activity: Predictive validity and the contribution of additional variables. *Journal of Sport and Exercise Psychology*, 24, 3–32.
- Hardeman, W., Johnston, M., Johnston, D., Bonetti, D., Wareham, N., & Kinmonth, A. L. (2002). Application of the theory of planned behavior in behavior change interventions: A systematic review. *Psychology & Health*, 17, 123–158.
- Hessing, D. J., Elffers, H., & Weigel, R. H. (1988). Exploring the limits of self-reports and reasoned action: An investigation of the psychology of tax evasion behavior. *Journal of Personality and Social Psychology*, 54, 405–413.
- Kiesler, C. A. (1971). *The psychology of commitment: Experiments linking behavior to belief*. New York, NY: Academic Press.
- LaPiere, R. T. (1934). Attitudes vs. actions. *Social Forces*, 13, 230–237.
- McEachan, R. R. C., Conner, M., Taylor, N. J., & Lawton, R. J. (2011). Prospective prediction of health-related behaviours with the theory of planned behaviour: A meta-analysis. *Health Psychology Review*, 5(2), 97–144.
- Randall, D. M., & Wolff, J. A. (1994). The time interval in the intention-behaviour relationship: Meta-analysis. *British Journal of Social Psychology*, 33(4), 405–418.
- Schroder, K. E. E., Carey, M. P., & Vanable, P. A. (2003). Methodological challenges in research on sexual risk behavior: II. Accuracy of self-reports. *Annals of Behavioral Medicine*, 26, 104–123.

- Smith, H. N. (1932). A scale for measuring attitudes about prohibition. *Journal of Abnormal and Social Psychology*, 26, 429–437.
- Stagner, R. (1942). Some factors related to attitude toward war, 1938. *Journal of Social Psychology*, 16, 131–142.
- Webb, T. L., Joseph, J., Yardley, L., & Michie, S. (2010). Using the internet to promote health behavior change: A systematic review and meta-analysis of the impact of theoretical basis, use of behavior change techniques, and mode of delivery on efficacy. *Journal of Medical Internet Research*, 10, e4.
- Wicker, A. W. (1969). Attitudes versus actions: The relationship of verbal and overt behavioral responses to attitude objects. *Journal of Social Issues*, 25, 41–78.

Suggested Reading

- Ajzen, I., & Albarracín, D. (2007). Predicting and changing behavior: A reasoned action approach. In I. Ajzen, D. Albarracín, & R. Hornik (Eds.), *Prediction and change of health behavior: Applying the reasoned action approach* (pp. 3–21). Mahwah, NJ: Lawrence Erlbaum Associates.
- Albarracín, D., Johnson, B. T., Fishbein, M., & Muellerleile, P. A. (2001). Theories of reasoned action and planned behavior as models of condom use: A meta-analysis. *Psychological Bulletin*, 127, 142–161.
- Fishbein, M., & Ajzen, I. (2010). *Predicting and changing behavior: The reasoned action approach*. New York, NY: Taylor & Francis.
- McEachan, R. R. C., Conner, M., Taylor, N. J., & Lawton, R. J. (2011). Prospective prediction of health-related behaviours with the theory of planned behaviour: A meta-analysis. *Health Psychology Review*, 5(2), 97–144.

Thriving in Life

Margaret L. Kern¹ and Jessie Sun^{1,2}

¹*Center for Positive Psychology, Graduate School of Education,
University of Melbourne, Parkville, VIC, Australia*

²*Department of Psychology, University of California, Davis, CA, USA*

Introduction

People experience life in a myriad of ways. For some, life is an adventure. They are optimistic, socially engaged, physically healthy, and active participants. For others, life is a daily grind. Work consumes a large portion of time and is simply something to endure each day. And for others, life is a struggle, with physical or psychological disability making it a challenge to function in daily life.

Considerable individual and societal resources are spent addressing the needs of those afflicted by physical and mental illness and disability. Medicine has made great strides in restoring normal function for those who suffer. Research and practice within health psychology has successfully identified ways to alleviate distress, helping patients to enjoy the highest quality of life possible, within the constraints of physical or mental disability.

But what about the majority of people who are not distressed, but also not functioning as well as they could be? A growing body of literature suggests that individuals who proactively approach life experience lower rates of physical and mental disabilities. It is shortsighted to take action only when symptoms appear. Rather, a proactive approach that builds internal and external resources over time will allow as many people as possible to not only survive but to truly thrive throughout life.

Thriving Defined

Thriving can be defined as a state at a single point in time or as a trajectory over time. As a state, thriving refers to current success or prosperity. As a trajectory, thriving refers to successful progression toward a desired goal or outcome, despite obstacles that might occur along the

way. The definition of success, prosperity, and desirable outcomes depends on the field of study.

The traditional medical perspective defines success in terms of biological function. Thriving can be observed objectively based on organic characteristics. “Failure to thrive” describes infants and children who do not develop as expected and fail to gain and maintain weight. The cause may be physical or emotional, but early diagnosis is important before permanent damage to the child occurs. Failure to thrive is also used in gerontology to define progressive physical and cognitive decline. In contrast, thriving is a normal state of health with no detectable signs of disease.

The traditional psychological perspective similarly defines success in terms of the lack of disability and disease but focuses on mental and social health rather than physical conditions. It is more subjective in nature—an organic cause of suffering might not exist, but the individual fails to function well in society.

Over the past few decades, there has been growing interest in a positive psychology perspective, which similarly focuses on mental and social health but argues that success is more than the lack of disease and disability. Thriving involves high levels of positive emotions, satisfaction with one’s life, positive social relationships, and the presence of other positive constructs such as a sense of meaning, mastery, personal growth, engagement in life, and accomplishment.

From an economic perspective, success is defined in terms of financial outcomes, such as income, gross domestic product, or profit. A thriving business is growing and building financial capital. A thriving economy has low levels of unemployment and performs strongly in the international market.

In the 1940s, the World Health Organization defined health as a complete state of physical, mental, and social well-being, not only the absence of disease and disability. This perhaps best captures the health psychology perspective, which includes physical, mental, and social aspects. Thriving is defined in terms of high quality of life and active engagement in life, despite the possible presence of physical and/or mental disorder. The aim of thriving is to allow a person to live as long as possible with the least physical, psychological, and social disabilities possible, focusing on quality rather than quantity (i.e., length) of life alone. This provides benefit to both the individual (by reducing suffering) and society (by reducing economic burden on the system).

Gerontology distinguishes among pathological (where there is accelerated physical, cognitive, and functional decline), usual (general breakdown in the body over time due to cumulative internal and external pressures over time), and successful aging (lack of physical disease and disability, high cognitive functioning, and active engagement in life). Multiple domains—including length of life, biological health, mental health, cognitive function, social competence, productivity, personal control, and life satisfaction—form the foundation of one’s health (Friedman & Kern, 2014). Successful aging involves selecting which elements will optimize life, allowing some domains to compensate for others (Baltes & Baltes, 1990).

One’s definition of thriving impacts how it is assessed, the extent to which the construct is continuous versus dichotomous, unidimensional versus multidimensional, subjective versus objective, and whether it can be captured at a single time point or necessitates seeing the person’s trajectory across life as a whole.

The common thread across these definitions is that thriving involves success across one or more domains. The traditional medical and psychological models tend to take a diagnostic approach, such that one is either healthy or unhealthy. Other perspectives more directly capture the continuous nature of biopsychosocial functioning, such that there is a broad range of functioning even among the healthy. Thriving refers to the higher end of this spectrum.

For our purposes here, we define *thriving* as subjective and objective success across multiple domains (e.g., physical, mental, cognitive, social, functional, economic), and we use the term interchangeably with *flourishing*. The domains that matter most may vary for different individuals and at different periods of time, depending on one's personality and social context. From this perspective, thriving is not a single number, but involves feeling and functioning well across multiple valued areas of life.

This definition is sufficient if we are only interested in identifying who or how many people are thriving at any given point in time. We suggest that *thriving in life* goes a step further and involves not only success at a single point in time but also a consistent trajectory of wellness over time. Thriving in life is a dynamic phenomenon that depends on multiple influences that interact and accumulate over the course of years and decades. Sustained states of thriving are rarely the outcome of a one-time fix or short-term behaviors that are not maintained; rather, thriving in life depends on the cumulative effects of a constellation of repeated behaviors combined with supportive environments. Just as plants need a regular supply of water and sunlight to flourish, human thriving depends on a sustained combination of salutary supports.

Predictors of Thriving

Numerous factors predict one's level of thriving, including one's personality, habitual behaviors, social relationships, socioeconomic status (SES), the environment, and culture.

Individual Factors

Some of the most consistent predictors of thriving are one's personality characteristics. Meta-analyses spanning thousands of participants find that conscientiousness (the tendency to be self-controlled, hardworking, dependable, organized, and socially responsible) and intelligence predict length of life on par with well-established risk factors (such as smoking, alcohol consumption, physical inactivity, and unhealthy diet; Calvin et al., 2011; Kern & Friedman, 2008). Personality accounts for over 40% of the variance in subjective well-being, with extraversion (the tendency to be sociable, talkative, assertive, enthusiastic, and active) and low neuroticism (the tendency to be irritable, moody, and temperamental) being the strongest predictors (Steel, Schmidt, & Shultz, 2008).

Behavioral Factors

Thriving is impacted not only by who we are but also by what we do. Habitual behaviors cumulatively impact one's health and well-being over time. Protective behaviors include moderate physical activity, healthy diet and weight control, restful sleep, immunizations, safe driving, wearing sunscreen, and regular medical checkups, whereas risky behaviors include smoking, drug use, alcohol abuse, thrill-seeking activities, and unsafe sex. For instance, regular physical activity can prevent and assist in the management of chronic illnesses and disease (Pedersen & Saltin, 2006), moderate sleep duration relates to lower mortality risk (Cappuccio, D'Elia, Strazzullo, & Miller, 2010), and the combination of not smoking, physical activity, healthy diet, and moderate drinking can predict up to a 14-year difference in length of life (Khaw et al., 2008). Such behaviors are physically healthy and relate to higher levels of psychological well-being, less mental distress, and better cognitive function.

Daily Activities

A related factor involves how time is spent, including employment, voluntary, home, and leisure activities. The impact of work is most clearly seen in the case of involuntary unemployment, which relates to a greater number of health problems, lower psychological well-being, and greater psychological distress (McKee-Ryan, Song, Wanberg, & Kinicki, 2005). Similarly, one of the greatest risk periods for early death is within the first few years after retirement. Beyond being necessary for meeting basic needs, work can provide a sense of identity, connection to like-minded others, and a source of personal accomplishment.

While work provides physical, psychological, social, and economic benefits, too much work can also be problematic. Technological developments have blurred the boundaries between work and home in many occupations. Working beyond contractual hours has been related to increased risk of one or more health problems, greater distress, and perceived work–life imbalance (Arlinghaus & Nachreiner, 2014).

Social Factors

The need to belong is one of the most pervasive and powerful fundamental human needs (Baumeister & Leary, 1995). Humans are extremely sensitive to even the slightest hint of social exclusion. Peer rejection and victimization in childhood predicts poor adjustment and psychopathology later in life. Loneliness is one of the strongest risk factors for failure to thrive—including higher risk of mortality, physical disease, and depressive symptoms (Hawkey & Cacioppo, 2010).

Social relationships range in quality; in general, positive relationships are protective against poor health outcomes, whereas hostile and negative relationships increase risk. Perceived social support and lower levels of interpersonal conflict may buffer the negative health-related consequences of stress and relate to lower risk of infection, morbidity, and mortality.

It is commonly believed that social relationships are one of the most important contributors to psychological well-being. However, this is complicated by how thriving is defined and measured. If thriving is defined in part as good social function, then social relationships are a marker of thriving, not a predictor. Associations among both objective and subjective relationship variables and subjective well-being may also be confounded by shared method variance and underlying variables such as personality traits. To distinguish predictors and outcomes, it may be useful to define factors such as connectedness (i.e., the ability to intimately connect with others) and fulfillment of one's sense of belonging as predictors and factors such as the quality of social relationships as outcomes.

Socioeconomic Factors

There is clear evidence for a social gradient, such that those who have a lower socioeconomic position have a shorter life expectancy, increased risk of mental and physical disease, and lower well-being. This appears to be a nonlinear relationship, with income making a greater difference at very low levels of income and less of an impact as income increases. Unfortunately, although narrowing income gaps may have the biggest impact on health and well-being around the world, inequality in most countries is increasing (Pickett & Wilkinson, 2015).

Although it is clear that people who have more money tend to be happier and healthier than people with less money, research suggests that how people spend their money can also influence their happiness. People report greater happiness after spending money on other people

compared with spending money on themselves and derive more satisfaction from purchasing life experiences than purchasing material possessions (Dunn, Aknin, & Norton, 2014).

Environmental Factors

Individuals are not isolated, but are embedded within social and environmental contexts. Over the past century, some of the greatest improvements in physical and mental health have occurred through the availability of clean drinking water, proper sewage treatment, and the development of immunizations and antibiotics. Contact with nature relates to greater physical activity, social interactions with others, increased happiness, enhanced cognitive capacity, and less mental distress (Hartig, Mitchell, de Vries, & Frumkin, 2014). There is also emerging discussion about the potential impact of climate change, increased pollution, economic volatility, exposure to violence, and political displacement. The long-term impact of these external factors is unknown, but it is clear that proactive approaches that create healthy, safe environments are needed.

Cultural Factors

At a broader level, culture has a profound influence on individual characteristics, behavior, social relationships, and even the definition of thriving. For example, North Americans tend to associate happiness with personal achievement more closely than with social harmony, whereas the opposite pattern has been observed for Japanese (Uchida & Kitayama, 2009). Thus, pathways to thriving most likely vary across cultures. However, there are also likely to be some common predictors of thriving across cultures. For instance, people who inhabit regions of Italy, Japan, and the United States that have high proportions of centenarians tend to value family and other social relationships, smoke less, have a healthy diet, engage in moderate physical activity, and are generally engaged in life (Buettner, 2012). There is also evidence that there are universal needs (e.g., social support, respect, mastery, autonomy) that predict subjective well-being across the world, although some cultures might emphasize some needs more than others (Tay & Diener, 2011).

Cultivating Thriving

The different predictors of thriving offer a range of areas that can potentially be targeted to help more people thrive. Although intelligence and personality are often viewed as fixed traits that are not readily amenable to change, people can and do change, as part of the natural process of maturation, through intervention, and through goals to change. Such changes in personality longitudinally predict changes in life satisfaction and psychological well-being (Boyce, Wood, & Powdthavee, 2013).

As personality influences behavior, it may be possible for individuals to emulate the behaviors of those who are higher in conscientiousness, extraversion, and intelligence and lower on neuroticism. For example, leading a healthier lifestyle could produce some of the health-related benefits of being more conscientious.

Positive psychology has developed various interventions and activities to build psychological and social well-being, such as gratitude exercises, discovering and using one's strengths, random acts of kindness, mindfulness, and active constructive responding. Workplaces and schools have successfully incorporated some of these strategies, providing preliminary support for lower healthcare costs, happier employees, and greater profit.

The extent to which temporary changes in behavior and short-term well-being exercises impact consequential life outcomes, such as long-term social relationships and length of life, is unknown. Changing a behavior (e.g., overeating) will not reduce mortality risk if it is simply replaced by another unhealthy behavior (e.g., drinking). There are likely to be multiple mechanisms by which a trait exerts its influence on thriving, some pathways may be more malleable than others, and the causal impact of many mechanisms are yet to be determined (Friedman & Kern, 2014). Thus, it may be more effective to change the underlying characteristics than to target multiple mediators.

Public health and public policy are well suited to target the external context—creating conditions that support individual and community thriving. Environments can be designed to make healthy behaviors easier to do and more psychologically attractive. For instance, designing parks, communities, transportation systems, schools, and buildings that encourage physical activity may also produce a wide range of environmental, economic, and physical and mental health benefits. Countries where people must opt out of organ donation have higher consent rates (80–100%) than those with an opt-in policy (5–30%; Johnson & Goldstein, 2003).

Policies and legislation can also be used to create social norms and provide checks on behavior. For instance, many countries now levy high taxes on cigarette and tobacco, making it economically unsustainable to smoke. At the same time, policy makers need to tread carefully; not all factors that influence thriving are equally amenable to legislation. For example, screenings for prostate cancer successfully detect many cases, but many of those diagnosed will die of another disease long before the cancer develops. As treatment can cause substantial negative impacts on psychosocial functioning, blindly requiring screenings to target one indicator of thriving (physical health, indicated by lack of cancer) can needlessly undermine functioning in another domain. A systemic consideration of the potential impact that any action will have across multiple domains of thriving is needed.

As a whole, different strategies will likely be effective for different people. From a socioecological perspective, interventions that target multiple levels of influence should result in more powerful and sustained changes than interventions only targeting a single level.

Future Directions

Although optimal functioning has long been an area of scholarly interest, the rapid expansion and application of positive psychology has made it more visible and is bridging research and applications in the real world. However, many gaps remain. Research often looks for simple, singular, linear causes, whereas life is complex, interconnected, and dynamic. Future research will benefit from incorporating a systems perspective, interdisciplinary scholarship, and closer connections between research and practice.

Methodologically, thriving in life is challenging to study. Advances in computational social science provide opportunities for bringing together diverse data sources, which capture both patterns across large populations, and momentary behavioral expressions of thoughts, attitudes, and behaviors. The methodologies, tools, and data that are most useful to understanding thriving are yet to be determined, and as all methodologies have strengths and limitations, multimethod approaches are likely to produce more robust and comprehensive insights.

Much of the research and application to date has focused on individuals, but individual thriving is interconnected with the environment in which a person resides. Questions remain around how individual behaviors and choices influence the well-being of close and distant others and how to balance individual well-being with collective needs. Additional research around person

and environment interactions could address how individuals relate to, are affected by, and impact upon the environment in which they live, as well as the broader biosphere.

Finally, although various interventions have been developed to cultivate thriving, the extent to which these are beneficial across individuals from different backgrounds, different periods of life, and under what circumstances is unclear. It is important to further develop a more nuanced understanding of individual fingerprints of thriving, which may depend on cultural differences, socioeconomic backgrounds, and individual values, needs, and motivations.

Conclusion

Thriving involves holistic success across multiple domains of life. Multiple factors impact a person or group's level of thriving at any given point in time. Although we have reviewed a range of factors that correlate with higher levels of well-being, the relative importance of different factors may vary between individuals. In this way, just as different plants thrive under different climates, there is no one-size-fits-all intervention or magic bullet for enhancing well-being; whereas different plants need different amounts of water, individuals may differ in the dose and types of social interaction they need to thrive. We also take the view that optimal well-being involves having a balanced life, with a mix of daily activities that enable individuals to fulfill a variety of needs. This implies that just as you can overwater a plant, it is also possible to have too much of one factor, at the cost of other domains, which undermines thriving as a whole.

Thriving in life not only involves functioning well at a single point in time but also involves one's trajectory over time. A focus on pathways emphasizes the importance of cultivating personal resources and supportive environments across the lifespan, as well as the need to understand how individual and social factors shift one's wellness trajectory over time. Harmful trajectories are not immutable, but early influences play a key role in setting a dominant trajectory. It may be relatively more effective to intervene at an early stage to establish healthier pathways in the first place than to try to correct unhealthy pathways in adulthood and old age. We need to know how different factors contribute to thriving across the lifespan, both naturally and through intervention.

Thriving in life is a complex, multiply-determined phenomenon. There are some universal and necessary ingredients, but the specific combination and importance of different factors will differ between individuals and even within a person at different periods of life. Research across a variety of disciplines is focusing on ways to enhance thriving, with potential benefit to both individuals and society as a whole.

Author Biographies

Dr. Margaret L. Kern is a senior lecturer at the Centre for Positive Psychology at the University of Melbourne's Graduate School of Education. She received her undergraduate degree from Arizona State University; a PhD in social/personality psychology from the University of California, Riverside; and postdoctoral training at the University of Pennsylvania. Her research examines questions around who flourishes in life, why, and what enhances and hinders healthy life trajectories.

Jessie Sun is a graduate student in the Department of Psychology at the University of California, Davis. She received her bachelor of arts (Hons.) from the University of Melbourne. She is interested in researching the interplay between personality, self-knowledge, social experiences, and well-being, especially using methods for studying dynamic processes in daily life.

References

- Arlinghaus, A., & Nachreiner, F. (2014). Health effects of supplemental work from home in the European Union. *Chronobiology International*, 30, 1197–1202. <http://dx.doi.org/10.3109/07420528.2014.957297>
- Baltes, P. B., & Baltes, M. M. (1990). Psychological perspectives on successful aging: The model of selective optimization with compensation. In P. B. Baltes & M. M. Baltes (Eds.), *Successful aging: Perspectives from the behavioral sciences* (pp. 1–34). New York, NY: Cambridge University Press.
- Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, 117, 497–529. <http://dx.doi.org/10.1037/0033-2909.117.3.497>
- Boyce, C. J., Wood, A. M., & Powdthavee, N. (2013). Is personality fixed? Personality changes as much as “variable” economic factors and more strongly predicts changes to life satisfaction. *Social Indicators Research*, 111, 287–305. <http://dx.doi.org/10.1007/s11205-012-0006-z>
- Buettner, D. (2012). *The blue zones: Lessons for living longer from the people who've lived the longest*. Washington, DC: National Geographic.
- Calvin, C. M., Deary, I. J., Fenton, C., Roberts, B. A., Der, G., Leckenby, N., & Batty, G. D. (2011). Intelligence in youth and all-cause-mortality: Systematic review with meta-analysis. *International Journal of Epidemiology*, 40, 626–644. <http://dx.doi.org/10.1093/ije/dyq190>
- Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. *Sleep*, 33, 585–592.
- Dunn, E. W., Aknin, L. B., & Norton, M. I. (2014). Prosocial spending and happiness: Using money to benefit others pays off. *Current Directions in Psychological Science*, 23, 41–47. <http://dx.doi.org/10.1177/0963721413512503>
- Friedman, H. S., & Kern, M. L. (2014). Personality, well-being, and health. *Annual Review of Psychology*, 65, 719–742. <http://dx.doi.org/doi:10.1146/annurev-psych-010213-115123>
- Hartig, T., Mitchell, R., de Vries, S., & Frumkin, H. (2014). Nature and health. *Annual Review of Public Health*, 35, 207–228. <http://dx.doi.org/10.1146/annurev-publhealth-032013-182443>
- Hawkey, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine*, 40, 218–227. <http://dx.doi.org/10.1007/s12160-010-9210-8>
- Johnson, E. J., & Goldstein, D. (2003). Do defaults save lives? *Science*, 302, 1338–1339. <http://dx.doi.org/10.1126/science.1091721>
- Kern, M. L., & Friedman, H. S. (2008). Do conscientious individuals live longer? A quantitative review. *Health Psychology*, 27, 505–512. <http://dx.doi.org/10.1037/0278-6133.27.5.505>
- Khaw, K.-T., Wareham, N., Bingham, S., Welch, A., Luben, R., & Day, N. (2008). Combined impact of health behaviours and mortality in men and women: The EPIC-Norfolk prospective population study. *PLoS Medicine*, 5(1), e12. <http://dx.doi.org/10.1371/journal.pmed.0050012>
- McKee-Ryan, F., Song, Z., Wanberg, C. R., & Kinicki, A. J. (2005). Psychological and physical well-being during unemployment: A meta-analytic study. *The Journal of Applied Psychology*, 90, 53–76. <http://dx.doi.org/10.1037/0021-9010.90.1.53>
- Pedersen, B. K., & Saltin, B. (2006). Evidence for prescribing exercise as therapy in chronic disease. *Scandinavian Journal of Medicine and Science in Sports*, 16, 3–63.
- Pickett, K. E., & Wilkinson, R. G. (2015). Income inequality and health: A causal review. *Social Science & Medicine*. <http://dx.doi.org/10.1016/j.socscimed.2014.12.031>
- Steel, P., Schmidt, J., & Shultz, J. (2008). Refining the relationship between personality and subjective well-being. *Psychological Bulletin*, 134, 138–161. <http://dx.doi.org/10.1037/0033-2909.134.1.138>
- Tay, L., & Diener, E. (2011). Needs and subjective well-being around the world. *Journal of Personality and Social Psychology*, 101, 354–365. <http://dx.doi.org/10.1037/a0023779>
- Uchida, Y., & Kitayama, S. (2009). Happiness and unhappiness in east and west: Themes and variations. *Emotion*, 9, 441–456. <http://dx.doi.org/10.1037/a0015634>

Suggested Reading

- Friedman, H. S., & Kern, M. L. (2014). Personality, well-being, and health. *Annual Review of Psychology*, 65, 719–742. <http://dx.doi.org/doi:10.1146/annurev-psych-010213-115123>
- Huppert, F. A. (2014). The state of wellbeing science: Concepts, measures, interventions, & policies. In F. A. Huppert & C. L. Cooper (Eds.), *Wellbeing: A complete reference guide, volume VI, interventions and policies to enhance wellbeing* (pp. 1–50). West Sussex: Wiley. <http://dx.doi.org/10.1002/9781118539415.wbwell01>
- Ryan, R. M., & Deci, E. L. (2001). On happiness and human potentials: A review of research on hedonic and eudaimonic well-being. *Annual Review of Psychology*, 52(1), 141–166. doi:10.1146/annurev.psych.52.1.141

Unrealistic Optimism and Health

James A. Shepperd¹ and Jennifer L. Howell²

¹*Psychology Department, University of Florida, Gainesville, FL, USA*

²*Psychology Department, Ohio University, Athens, OH, USA*

When asked to predict their future, people are not evenhanded in their prognostications. Rather, they show a strong preference for optimism, believing that favorable outcomes are more likely on their horizon than is realistically possible. This unrealistic optimism occurs for a seemingly endless number of outcomes. For example, college women report that they are less likely than their peers to experience an unwanted pregnancy, smokers believe they are less likely than others smokers to suffer smoking-related illnesses, drivers report that they are less likely than other drivers to experience an automobile accident, and people generally believe they are less likely than are their peers to experience diseases including HIV, HPV and chlamydia, and cancer (for a review, see Shepperd, Klein, Waters, & Weinstein, 2013). The bias seems remarkably stable across time (Shepperd, Helweg-Larsen, & Ortega, 2003) and impervious to interventions designed to reduce it (Weinstein & Klein, 1995).

At first blush, unrealistic optimism seems problematic in that it could prompt people to take unnecessary risks, fail to take precautions, or otherwise make decisions that increase their odds experiencing harm. However, the relationship between unrealistic optimism and behavior is more complex. Understanding when unrealistic optimism is or is not problematic requires defining unrealistic optimism and understanding why it occurs. We distinguish between two types of unrealistic optimism, review the causes of unrealistic optimism, and discuss the consequences of unrealistic optimism for health.

Defining Unrealistic Optimism

Unrealistic optimism refers to the prediction that one's personal outcomes will be more favorable than can possibly be. On the surface, unrealistic optimism shares some overlap with *dispositional optimism*, which represents a trait tendency to hold positive expectation about the future. However, unrealistic optimism is empirically and conceptually distinct. Unrealistic

optimism is not just a positive expectation (like dispositional optimism); it is a positive expectation that cannot possibly be true.

But how do we determine that a person's predictions are unrealistically optimistic? Researchers have identified two types of unrealistic optimism that differ in the standard used to evaluate the predictions (Shepperd et al., 2013). The first type—unrealistic *absolute* optimism—occurs when people make overly favorable predictions about their futures relative to an objective, quantitative standard such as base rate data, prior experience, or what is realistically possible.

Several studies demonstrate unrealistic absolute optimism: researchers have shown that students overestimate the starting salaries they will receive after graduation from college, financial advisors overestimate future corporate earnings, and taxpayers overestimate how quickly they will file their income tax forms (see Carrol, Sweeny, & Shepperd, 2006 for a review). Other research demonstrates unrealistic optimism relative to base rate or epidemiological evidence. For example, relative to local baseline information, homeowners underestimate the chances that their house has radon gas (Weinstein & Lyon, 1999), and college students underestimate their personal risk for STDs including chlamydia and HPV (Rothman, Klein, & Weinstein, 1996). Other research demonstrates unrealistic optimism by comparing people's personal estimates with the experiences of a comparable control group. For example, female US Marines in one study estimated on average a 14.5% chance that they would get pregnant in the next 12 months, which was significantly lower than the 12-month pregnancy rate (22.0%) of a comparable group of female Marines (Gerrard, Gibbons, & Warner, 1991).

The second type of unrealistic optimism—unrealistic *comparative* optimism—occurs when people erroneously conclude that unfavorable outcomes are less likely to happen, and favorable outcomes more likely to happen, to them than to their peers. Researchers measure unrealistic comparative optimism in two ways. The first involves comparing an individual's comparative estimate with the comparative estimate indicated by an objective standard. To illustrate, a woman may predict that she is less likely than the average woman to get breast cancer. However, if her true risk, based on epidemiological predictors (e.g., family history, age at first menstruation, genetic predisposition), is average or above average, then she is displaying unrealistic comparative optimism. Evidence for this manifestation of unrealistic comparative optimism comes from studies of breast cancer, smoking-related diseases, and fatal heart attacks (see Shepperd et al., 2013).

The second manifestation of unrealistic comparative optimism occurs at the group level and does not identify whether any specific individual is unrealistically optimistic. It occurs when group members on average rate their risk of experiencing an unfavorable event as below average relative to other members of the group. The assumption is that the mean individual risk estimate of the group members for a particular event should be average, with estimates of people whose risk is below average balancing out the estimates of people whose risk is above average. If the group's mean estimate of experiencing an unfavorable event is below average, then the group is displaying unrealistic comparative optimism. For example, college students overwhelmingly report that they are less likely than other college students to become obese or to have their first marriage end in divorce (Rothman et al., 1996). Because personal estimates should balance out to be "average" rather than "below average," these students are displaying unrealistic comparative optimism.

Causes of Unrealistic Optimism

Researchers have identified three reasons why people are unrealistically optimistic (Shepperd, Waters, Weinstein, & Klein, 2015): (a) people are motivated to expect favorable future events,

(b) people overrely on personal information when making judgments, and (c) people process information in ways that lead them to be overly optimistic. First, people may be motivated to believe or have others believe that they are unlikely to experience unfavorable future events. The motivation may stem from several causes (Shepperd, Carroll, Grace, & Terry, 2002). For instance, it may arise because it feels good to imagine a positive future and feels bad to imagine a negative future. It also may arise from superstitious thinking, such as the belief that imagining favorable outcomes may somehow increase the likelihood that the favorable outcomes will transpire. Such thinking is not entirely superstitious in that an imagining a favorable outcome may facilitate actions designed to bring about the outcome. Finally, the motivation may reflect a perception of what other people believe they should think. Indeed, people believe that it is better to be unrealistically optimistic in most circumstances than to be accurate or pessimistic.

The second reason people may display unrealistic optimism arises from an overreliance on personal information when making judgments. People know their personal history, current actions, and future plans, and they use this information when predicting their personal likelihood of experiencing various outcomes. These future plans are typically plans to bring about desired outcomes and avoid undesired outcomes and may insufficiently weight, or fail to include altogether, possible setbacks, roadblocks, competing demands, and past failures. Consistently, people display greater unrealistic optimism for controllable events than uncontrollable events, presumably because they assume they will take more action to bring about desired outcomes than they actually do (see Shepperd et al., 2015).

Conversely, people often neglect or are unaware of other people's plans, intentions, and goal-directed behavior (i.e., that other people are employing, or intend to employ, actions to avoid undesired outcomes and achieve desired outcomes). Thus, they underweight other's experiences when making comparative estimates. In fact, supplying people with information about others' goal-directed behavior or intentions diminishes unrealistic optimism (see Shepperd et al., 2015). The reduction may occur because the information prompts people to recognize personal similarity with the comparison target and thus modify their estimates accordingly. Alternatively, the information may make the comparison target seem less abstract and more humanlike. Consistent with the latter possibility are findings that people display less unrealistic comparative optimism when the comparison target is a concrete, specific person as opposed to the amorphous *average person* (see Shepperd et al., 2015). Together, this evidence suggests that people often overrely on personal information and fail to use information about others when making comparative judgments.

The third reason people display unrealistic optimism arises from the way people process information. Three processes are particularly important (Shepperd et al., 2015). The first is that people making likelihood judgments gauge their chances of experiencing an outcome based on how well they match their stereotype of people who experience the outcome. Stereotypes, however, are often inaccurate or exaggerated. For example, the stereotype of the person who gets a sexually transmitted disease may be someone who has frequent unprotected sex with multiple sex partners. In comparison with this stereotype, many people naturally estimate that their risk is lower. The second process is that people making comparative judgments often omit the comparison part of the judgment. Instead, of comparing their chances relative to some standard or to the chances of the average person, people often merely consider their own chances, transforming the comparative judgment into a personal judgment with no clear reference group. Third, people making judgments often weight information more heavily to the extent it suggests that favorable outcomes lie ahead. Indeed, people seem to fail to encode information that optimism is unwarranted (Sharot, Korn, & Dolan, 2011).

The Consequences of Unrealistic Optimism

Examining the consequences of unrealistic optimism is difficult because the research is almost entirely correlational. Unrealistic optimism is challenging to manipulate, making it difficult to assess causal relationships between unrealistic optimism and health outcomes. Although unrealistic optimism may influence behavior, it is also possible that people display unrealistic optimism because of their current or intended behavior or some other third variable.

Three types of studies allow researchers to better examine the health consequences associated with unrealistic optimism: (a) longitudinal research, which can show that unrealistic optimism predates health decisions, (b) quasi-experimental studies that provide research participants the opportunity to engage (or not engage) in health-related behaviors, and (c) studies that equate participants on pertinent risk factors or past behavior and then examine whether participants high versus low in unrealistically optimism differ in health decisions, behaviors, or outcomes. All of the studies described in the sections that follow use at least one of these methodological approaches. To facilitate our discussion, we first discuss evidence suggesting that unrealistic optimism is beneficial for health followed by evidence that unrealistic optimism is problematic for health.

Health Benefits of Unrealistic Optimism

Numerous studies link dispositional optimism to hope, positive affect, and greater goal-directed persistence. Given the overlap (albeit a modest overlap) between dispositional optimism and unrealistic optimism, it is reasonable to assume that unrealistic optimism might have benefits. Consistent with this reasoning are the results of two studies. The first examined unrealistic absolute optimism among HIV-infected gay men, who reported that they were less likely than a comparison group of non-infected men to develop AIDS (Taylor et al., 1992). More importantly, greater unrealistic optimism among these HIV-infected men corresponded with reports of health-promoting behaviors such as a healthy diet, jogging, and getting adequate sleep.

The second study examined unrealistic comparative optimism among patients in a cardiac rehabilitation program. Although all study participants experienced some coronary event (myocardial infarction, bypass surgery, angina, etc.), some patients displayed unrealistic comparative optimism about their risk of experiencing a future cardiac event, whereas others did not. The patients who reported the greatest unrealistic comparative optimism were less likely to experience a cardiac event (such as readmission to a hospital for chest pain/angina or experience of a heart attack) over the next 12 months, even when controlling for true risk (Hevey, McGee, & Horgan, 2014).

Health Costs of Unrealistic Optimism

The two studies just described are quite unique in demonstrating benefits of unrealistic optimism. Most published studies examining the consequences of unrealistic optimism suggest that unrealistic optimism can be problematic for health. Because unrealistic optimism represents an expectation that exceeds what is realistically possible, it can lead people to take unnecessary risks. To understand the health problems linked to unrealistic optimism, we examine the two forms of unrealistic optimism (absolute and comparative) separately.

Unrealistic Absolute Optimism

Unrealistic absolute optimism has a number of problematic consequences for health. For instance, it can lead to misplaced hope, as illustrated by a study of participants in a phase I clinical cancer trial. The researchers explained that the purpose of such trials is to establish the feasibility of a future randomized trial and that no participant was likely to experience benefits as a consequence of their participation. Nevertheless, 60% of the participants in the trial reported that they were more likely than the other trial participants to experience health benefits from the trial (Jansen et al., 2011). These estimates reflect unrealistic optimism because the trial participants believed they would experience health benefits when they were explicitly told that they would not. The findings also illustrate unrealistic comparative optimism in that patients believe that they were more likely than fellow clinical trial patients to experience benefits when they were not.

Other studies have demonstrated that unrealistic absolute optimism causes disappointment, regret, and other negative emotions when the future does not unfold as expected. For example, students in one study who awaited the results of a classroom examination felt worse about their exam performance if they were unrealistically optimistic about their performance than if they were accurate or unrealistically pessimistic, regardless of their actual exam performance (Sweeny & Shepperd, 2010).

Unrealistic absolute optimism can also have behavioral consequences. In a nationally representative sample of smokers, the more unrealistically optimistic smokers were about their lung cancer risk, the less likely they were to list quitting as a means to decrease lung cancer risk, and the more curable they viewed lung cancer to be (Dillard, McCaul, & Klein, 2006). Moreover, despite the fact that quitting smoking is more difficult the longer a person smokes, smokers typically overestimate their ability to quit, which may delay or undermine future attempts to quit. Indeed, greater unrealistic absolute optimism corresponded with lower intentions to quit smoking in the nationally representative sample of smokers just mentioned (Dillard et al., 2006).

Other negative consequences also arise from unrealistic absolute optimism. For example, one study examined attention to relevant health information among college students who engaged risky sexual activity. Compared with realists—who accurately perceived themselves to be at high risk for pregnancy and STDs—unrealistically optimistic students perceived an informational pamphlet about STDs and pregnancy as less relevant to them. As a result, their personal risk estimates were less responsive to the information provided in the pamphlet (Wiebe & Black, 1997). Perhaps most sobering is the finding that greater unrealistic optimism about risk for heart disease in a sample of older adults corresponded with the thickness of the carotid artery (Ferrer et al., 2012), a subclinical marker of atherosclerosis that is linked to increased heart attack risk.

Unrealistic Comparative Optimism

Research exploring the consequences of unrealistic comparative optimism for health is generally sparse, but several studies have demonstrated that it too can have negative health consequences. In one study, college drinkers who were unrealistically optimistic about their risk of experiencing alcohol-related problems in the next year reported consuming more alcohol than did realistic participants. More importantly, 6, 12, and 18 months after the initial survey, the drinkers who displayed unrealistic comparative optimism also reported more alcohol-related negative events (hangover, trouble with the police, missed classes, etc.) than did the realistic participants (Dillard, Midboe, & Klein, 2009). In a second study, older adults who reported

unrealistic comparative optimism about their risk for heart attack reported less worry about their risk level than did participants who were accurate or unrealistically pessimistic. They also displayed less knowledge of risk factors and poorer recall of an essay they read earlier about heart attack risk factors (Radcliffe & Klein, 2002). Other research finds that people who displayed unrealistic comparative optimism about avoiding the H1N1 virus reported lower intentions to wash their hands and use hand sanitizers (Kim & Niederdeppe, 2013).

Other research suggests that the combination of high unrealistic comparative optimism and high dispositional optimism can be particularly problematic. People high in both types of optimism tend to be more defensive and more likely to display behaviors that infirm their health. For example, college students in one study imagined that a dental visit revealed that they needed a root canal. Students who were high in both dispositional and unrealistic comparative optimism were more dismissive of the hypothetical dental threat in the near future than were college students high in dispositional but low in comparative optimism. Moreover, when provided the opportunity to seek additional health information, these students were less likely to do so (Fowler & Geers, 2015).

Summary

Unrealistic optimism is remarkably pervasive, stable over time, and resistant to change. It appears in two forms: absolute and comparative unrealistic optimism. Specifically, people can be unrealistically optimistic relative to objective indicators of their risk (unrealistic absolute optimism) or in estimates of how their risk compares with the risk of their peers (unrealistic comparative optimism). Unrealistic optimism can arise from a motivation to believe (or have others believe) that one's future will be rosy, from disparity in information about oneself versus the objective standard or comparison target, or from cognitive processes that emerge when people are asked to evaluate their risk (e.g., reliance on stereotypes, insufficiently considering others, differential weight of favorable versus unfavorable information). Finally, although unrealistic optimism can have positive health consequences, a number of studies suggest that it can also produce negative health consequences. Perhaps the most challenging task facing researchers studying unrealistic optimism is clarifying when unrealistic optimism is linked to positive versus negative health outcomes.

Author Biographies

James A. Shepperd, PhD, is the R. David Thomas endowed professor of psychology at the University of Florida and the research director for the Southeast Center for Research to Disparities in Oral Health. Most of his research examines how people manage threatening information, which includes topics such as optimism, maintaining desired self-views, and information avoidance. Much of his work involves applying psychological theory to addressing health concerns such as screening for cancer.

Jennifer L. Howell, PhD, is an assistant professor at Ohio University. Her research focuses primarily on psychological threat management and health. She seeks to understand the proactive and reactive strategies people use to manage bad news, particularly about their health. This broad umbrella encompasses work on decision making, responses to feedback, defensiveness, physical health behaviors, risk, coping, individual differences, social cognition, attitudes, and communication.

References

- Carroll, P., Sweeny, K., & Shepperd, J. A. (2006). Forsaking optimism. *Review of general psychology*, 10(1), 56–73.
- Dillard, A. J., McCaul, K. D., & Klein, W. M. P. (2006). Unrealistic optimism in smokers: Implications for smoking myth endorsement and self-protective motivation. *Journal of Health Communication*, 11(Suppl 1), 93–102. doi:10.1080/10810730600637343
- Dillard, A. J., Midboe, A. M., & Klein, W. M. P. (2009). The dark side of optimism: Unrealistic optimism about problems with alcohol predicts subsequent negative event experiences. *Personality and Social Psychology Bulletin*, 35(11), 1540–1550. doi:10.1177/0146167209343124
- Ferrer, R. A., Klein, W. M. P., Zajac, L. E., Sutton-Tyrrell, K., Muldoon, M. F., & Kamarck, T. W. (2012). Unrealistic optimism is associated with subclinical atherosclerosis. *Health Psychology*, 31(6), 815–820. doi:10.1037/a0027675
- Fowler, S. L., & Geers, A. L. (2015). Dispositional and comparative optimism interact to predict avoidance of a looming health threat. *Psychology & Health*, 30(4), 456–474. doi:10.1080/08870446.2014.977282
- Gerrard, M., Gibbons, F. X., & Warner, T. D. (1991). Effects of reviewing risk-relevant behavior on perceived vulnerability among women marines. *Health Psychology*, 10(3), 173–179. doi:10.1037/0278-6133.10.3.173
- Hevey, D., McGee, H. M., & Horgan, J. H. (2014). Comparative optimism among patients with coronary heart disease (CHD) is associated with fewer adverse clinical events 12 months later. *Journal of Behavioral Medicine*, 37(2), 300–307. doi:10.1007/s10865-012-9487-0
- Jansen, L. A., Appelbaum, P. S., Klein, W. M. P., Weinstein, N. D., Cook, W., Fogel, J. S., & Sulmasy, D. P. (2011). Unrealistic optimism in early-phase oncology trials. *IRB: Ethics & Human Research*, 33(1), 1–8.
- Kim, H. K., & Niederdeppe, J. (2013). Exploring optimistic bias and the integrative model of behavioral prediction in the context of a campus influenza outbreak. *Journal of Health Communication*, 18(2), 206–222. doi:10.1080/10810730.2012.688247
- Radcliffe, N. M., & Klein, W. M. P. (2002). Dispositional, unrealistic and comparative optimism: Differential relations with the knowledge and processing of risk information and beliefs about personal risk. *Personality and Social Psychology Bulletin*, 28(6), 836–846. doi:10.1177/0146167202289012
- Rothman, A. J., Klein, W. M., & Weinstein, N. D. (1996). Absolute and relative biases in estimations of personal risk. *Journal of Applied Social Psychology*, 26(14), 1213–1236. doi:10.1111/j.1559-1816.1996.tb01778.x
- Sharot, T., Korn, C. W., & Dolan, R. J. (2011). How unrealistic optimism is maintained in the face of reality. *Nature Neuroscience*, 14(11), 1475–1479. doi:10.1038/nn.2949
- Shepperd, J. A., Carroll, P., Grace, J., & Terry, M. (2002). Exploring the causes of comparative optimism. *Psychologica Belgica*, 42(1-2), 65–98.
- Shepperd, J. A., Helweg-Larsen, M., & Ortega, L. (2003). Are comparative risk judgments consistent across time and events? *Personality and Social Psychology Bulletin*, 29(9), 1169–1180. doi:10.1177/0146167203254598
- Shepperd, J. A., Klein, W. M. P., Waters, E. A., & Weinstein, N. D. (2013). Taking stock of unrealistic optimism. *Perspectives on Psychological Science*, 8(4), 395–411. doi:10.1177/1745691613485247
- Shepperd, J. A., Waters, E. A., Weinstein, N. D., & Klein, W. M. P. (2015). A primer on unrealistic optimism. *Current Directions in Psychological Science*, 24(3), 232–237. doi:10.1177/0963721414568341
- Sweeny, K., & Shepperd, J. A. (2010). The costs of optimism and the benefits of pessimism. *Emotion*, 10(5), 750–753. doi:10.1037/a0019016
- Taylor, S. E., Kemeny, M. E., Aspinwall, L. G., Schneider, S. G., Rodriguez, R., & Herbert, M. (1992). Optimism, coping, psychological distress, and high-risk sexual behavior among men at risk for acquired immunodeficiency syndrome (AIDS). *Journal of Personality and Social Psychology*, 63(3), 460–473. doi:10.1037/0022-3514.63.3.460

- Weinstein, N. D., & Klein, W. M. (1995). Resistance of personal risk perceptions to debiasing interventions. *Health Psychology, 14*(2), 132–140. doi:10.1037/0278-6133.14.2.132
- Weinstein, N. D., & Lyon, J. E. (1999). Mindset, optimistic bias about personal risk and health-protective behaviour. *British Journal of Health Psychology, 4*, 289–300. doi:10.1348/135910799168641
- Wiebe, D. J., & Black, D. (1997). Illusional beliefs in the context of risky sexual behaviors. *Journal of Applied Social Psychology, 27*(19), 1727–1749. doi:10.1111/j.1559-1816.1997.tb01622.x

Suggested Reading

- Chambers, J. R., & Windschitl, P. D. (2004). Biases in social comparative judgments: The role of non-motivated factors in above-average and comparative-optimism effects. *Psychological Bulletin, 130*, 813–838. doi:10.1037/0033-2909.130.5.813
- Shepperd, J. A., Klein, W. M. P., Waters, E. A., & Weinstein, N. D. (2013). Taking stock of unrealistic optimism. *Perspectives on Psychological Science, 8*(4), 395–411. doi:10.1177/1745691613485247
- Shepperd, J. A., Waters, E. A., Weinstein, N. D., & Klein, W. M. P. (2015). A primer on unrealistic optimism. *Current Directions in Psychological Science, 24*, 232–237. doi:10.1177/0963721414568341

Waiting for Health News

Angelica Falkenstein, Michael D. Dooley,
and Kate Sweeny

Department of Psychology, University of California, Riverside, CA, USA

Uncertain waiting periods are a regular part of the healthcare experience. Patients await uncertain news when they undergo diagnostic medical tests, engage in periods of “watchful waiting,” or anticipate learning the efficacy of an administered treatment. Medical waiting periods can range from minutes to months, with patients left in limbo until they are contacted with results. Although research documenting the psychological effects of such waiting periods is relatively sparse, empirical evidence underscores the exceptional distress that patients experience while awaiting news related to their health. For example, waiting for medical results can provoke the same levels of stress as receiving the bad news of a cancer diagnosis (Lang, Berbaum, & Lutgendorf, 2009). One study even found that people awaiting medical news experience anxiety equivalent to patients admitted to psychiatric facilities for generalized anxiety disorder (Scott, 1983), underscoring the significant distress health uncertainty can cause.

In this entry, we discuss the nature of uncertain and highly important medical waiting periods as well as how waiting experiences change over time. We then discuss how waiting experiences predict responses to both desirable and undesirable news, ending with suggestions for ways to make medical waiting periods easier for patients to endure.

The Central Role of Anxiety

A small but growing body of research documents the distressing nature of both medical and nonmedical waiting periods. Although the experience of waiting for inevitable events (e.g., waiting rooms, queuing) may be frustrating and uncomfortable, people experience particular distress when they await uncertain news. Uncertainty is characterized by heightened anxiety (Sweeny & Falkenstein, 2015), which is often accompanied by rumination (Sweeny & Andrews, 2014). Generally speaking, people experiencing self-relevant uncertainty feel motivated to seek out information to alleviate uncertainty and associated anxiety. During medical

waiting periods, however, additional reliable information is largely unavailable until the test results are in.

Because medical waiting periods are characterized by irresolvable uncertainty, anxiety persists as the predominant emotional experience (Sweeny & Cavanaugh, 2012; Sweeny & Falkenstein, 2015). Indeed, patients report anxiety as the most prevalent emotion during diagnostic waiting periods, exceeding levels of anger, confusion, tension, and intrusive thoughts (Montgomery & McCrone, 2010). Research with patients undergoing biopsy procedures similarly documents high levels of anxiety (Northouse, Jeffs, Cracchiolo-Caraway, Lampman, & Dorris, 1995), surpassing levels among college students, high-risk controls (Maxwell et al., 2000), and, in some cases, clinical thresholds (Poole et al., 1999). High anxiety during medical waiting periods not only exerts deleterious effects during the waiting period itself, such as an inability to concentrate and plan (Thorne, Harris, Hislop, & Vestrup, 1999), but may also influence patients' cognitive processing abilities down the line (Scott, 1983).

Anxiety experienced during waiting periods surpasses even levels of anxiety in response to bad news, suggesting that in some respects, waiting can be more difficult than receiving bad news (Sweeny & Falkenstein, 2015). To illustrate, a study of women undergoing fertility treatment found that these women experienced high levels of anxiety while awaiting results of in vitro fertilization, contrasted by high levels of disappointment but relatively low levels of anxiety in the face of negative pregnancy test results (Boivin & Lancaster, 2010), indicating that both waiting and receiving bad news are difficult but qualitatively different experiences.

Change Across the Waiting Period

Although waiting periods provoke high levels of anxiety and rumination, the intensity of these experiences fluctuates over time. Trends of anxiety and rumination tend to follow a U-shaped curve, with the highest levels occurring at the beginning of a wait (e.g., directly after a medical test), followed by a respite, only to ramp up again as news becomes imminent (Sweeny & Andrews, 2014). The shape of this pattern likely mirrors patterns of attentional focus and outcome salience and highlights people's tendency to shirk optimism and embrace pessimism in an attempt to brace for the worst when news is imminent (Shepperd, Ouellette, & Fernandez, 1996; Sweeny & Krizan, 2013). Pessimistic expectations are often accompanied by anxiety (Sweeny & Andrews, 2014; Sweeny, Reynolds, Falkenstein, Andrews, & Dooley, 2016), and people lower their expectations to the greatest extent right before news arrives (Sweeny & Krizan, 2013).

The pattern of anxiety during nonmedical waiting periods translates to medical waiting periods. For instance, the majority of patients awaiting an elective cardiac catheterization reported increases in anxiety over time as the procedure approached (Harkness, Morrow, Smith, Kiczula, & Arthur, 2003). Similarly, women awaiting a breast biopsy procedure increasingly sought social support as the biopsy neared, with open-ended responses revealing that many women experienced increases in anxiety during this period (Lebel et al., 2003). Women who reported relatively low anxiety immediately after a breast biopsy also experienced increases in anxiety while waiting for the biopsy results (no change for women already high in anxiety; Poole et al., 1999). Despite the stress of waiting in the healthcare context, past research has often ignored the diagnostic phase, instead focusing on understanding patients' experiences after a diagnosis. However, a growing literature illustrates that diagnostic phases are emotionally dynamic and difficult for patients to endure (Montgomery & McCrone, 2010).

Variability Across Different Types of Waiting Periods

Waiting for medical news often has the potential to cause high levels of anxiety, but not all outcomes provoke the same levels of distress. Intuitively, waiting experiences depend on the importance of the news at hand; surely waiting for news of a tumor biopsy is more nerve-racking than awaiting news of a cholesterol test. Situational factors, such as the importance of an outcome or the risk associated with a negative outcome, are indeed one source of variability in people's waiting experiences. For instance, couples recalled greater distress while trying to conceive their youngest to the extent that they had placed greater importance on parenthood during that time (Sweeny, Andrews, Nelson, & Robbins, 2015). Couples who had a higher risk of complications also recalled experiencing more anxiety and intrusive thoughts as they tried to conceive.

Furthermore, the length of the wait influences how people respond to news. When waiting for a potentially desirable outcome, people prefer shorter waiting periods. However, when the news is likely to be undesirable or has potentially detrimental ramifications, people prefer longer waiting periods that ostensibly give them time to prepare for the devastating blow (Montgomery & McCrone, 2010).

It is important to note that not all medical waiting periods elicit anxiety and rumination. In a study investigating the wait for genetic risk information, patients did not seem to be particularly distressed (Phelps, Bennett, Iredale, Anstey, & Gray, 2006), an observation that differs dramatically from studies of women awaiting breast biopsy results (e.g., Poole et al., 1999). Additionally, pregnancy may be an inherently unique medical waiting period. Even women who have previously miscarried see the wait for baby as a generally positive experience, although women with recurrent miscarriages experience more stress (Nelson, Robbins, Andrews, & Sweeny, 2015).

Individual Differences Affecting the Waiting Experience

In addition to situational factors, research has linked dispositional factors and a broad range of personal resources to experiences of uncertainty. Higher dispositional optimism is often associated with less distress while waiting (Stanton & Snider, 1993; Sweeny et al., 2015). However, dispositional optimists are just as likely as their pessimistic peers to brace for the worst as the moment of truth approaches (Sweeny & Falkenstein, 2017). Chronic anxiety (Novy, Price, Huynh, & Schuetz, 2001), intolerance of uncertainty (Sweeny et al., 2015), and negative appraisal patterns (Stanton & Snider, 1993) exacerbate waiting distress, making people high in these characteristics especially ill-suited to wait for health news.

Conversely, people with more social, emotional, and cognitive resources demonstrate greater ability to cope with uncertainty-related distress. For example, people with more personal resources recall less distress during the time when they were trying to conceive (Sweeny et al., 2015), and people high in self-esteem are buffered against some indicators of waiting distress (Shepperd et al., 1996; Sweeny & Andrews, 2014). Ample research highlights strong support networks as pivotal to positive health-related outcomes, which may stem from supportive others helping people reduce or cope with uncertainty (Uchino, 2009). Together, these findings suggest that ensuring the availability of social, emotional, and cognitive resources may be instrumental in improving patients' ability to navigate medical waiting periods.

Although some individual differences consistently predict waiting distress or the lack thereof, studies investigating demographic characteristics have yielded inconsistent associations with waiting experiences. For example, some studies have found that age predicts distress during the wait for a potential breast cancer diagnosis (e.g., Novy et al., 2001), but others have failed to find such a link (Montgomery & McCrone, 2010). Some studies also show that family health history (Novy et al., 2001) and previous experience with the issues at hand (e.g., screenings, miscarriages; Maxwell et al., 2000; Schnur et al., 2008) predict heightened distress while waiting, although a quantitative review found that the greatest declines in expectations (i.e., more pessimism) precede news about unfamiliar outcomes (Sweeny & Krizan, 2013). Further research is needed to determine the role that characteristics such as age, previous experience, socioeconomic status, and gender play into how patients wait for medical news.

Effects of Waiting Experiences on Responses to Health News

Beyond predictors of waiting experiences, researchers have also examined how waiting experiences shape reactions to news once it arrives. For instance, even pessimists overwhelmingly prescribe optimism to others faced with uncertain, self-relevant outcomes, revealing a normative belief that there is some inherent benefit in expecting good outcomes (Armor, Massey, & Sackett, 2008). Research exploring expectations during waiting periods supports this idea, linking optimistic expectations with reduced distress (Sweeny & Andrews, 2014; Sweeny et al., 2016). Optimism, however, seems to backfire when confronted with bad news. Disconfirmed optimistic expectations are met with greater disappointment and shock compared with confirmed pessimistic expectations (Krizan, Miller, & Johar, 2010; Sweeny et al., 2016). Optimism also provides no benefit when the news is good: people with optimistic expectations are less elated by good news than are people who lower their expectations and brace for the worst (Sweeny et al., 2016).

In contrast, pessimistic expectations may provide motivational benefits to prepare for bad news. People who expect a bad outcome might take steps to mitigate some of the difficulties that could accompany bad news (Sweeny, Carroll, & Shepperd, 2006). For example, a woman anticipating a breast cancer diagnosis could inquire about her employer's extended leave policy or arrange for childcare. The literature on defensive pessimism convincingly shows that at least for some people, pessimistic expectations and associated anxiety can serve as a motivating force to take action (Norem & Cantor, 1986). However, pervasive distress can impede daily functioning and sound decision making. Thus, delaying the descent toward pessimism until the end of a wait may allow patients to capitalize on the benefits of both optimism and pessimism, suggesting that well-timed optimism and well-timed shifts from optimism may be key to an optimal overall medical waiting experience (Sweeny et al., 2006).

Key Future Directions for Research on Waiting for Health News

Because poorly timed pessimism and prevalent distress may reduce patients' ability to function and thus cope with uncertainty surrounding their health, interventions are needed to target patients' distress and promote positive strategies to cope with uncertainty. For example, patients may also better cope with a wait if they are able to take their mind off of the news at hand. Although some research points toward distraction as a harmful method of coping (Lebel

et al., 2003; Sweeny et al., 2016), other studies support the efficacy of the strategy (e.g., Boivin & Lancaster, 2010).

One possibility is that distraction is only helpful to the extent that people are actually cognitively diverted from their worries and fears, with unsuccessful attempts further compounding the problem. If this is the case, research aimed at understanding activities that effectively distract from anxiety and intrusive thoughts could highlight promising interventions for those experiencing highly stressful medical waiting periods. For instance, research on flow demonstrates that autonomous activities that match one's skill level can create cognitive immersion in the task as well as an energized focus, such that one loses reflective self-consciousness and awareness of time. Flow has been associated with motivation to perform and decreased anxiety (Csikszentmihalyi & Rathunde, 1993), indicating it may foster an action orientation in patients and help decrease emotional distress. Other methods of distraction, such as fostering cognitive busyness, may also prove fruitful in helping people cope while waiting for uncertain medical results.

Healthcare providers may also be able to meaningfully shape patients' waiting experiences for the better by communicating effectively with patients. For example, women with abnormal mammogram results experience less anxiety when waiting for follow-up testing if they are satisfied with the initial information they received from their healthcare provider (Pineault, 2007). Doctors and nurses with the responsibility of communicating such information may be in a unique position to ease patients' future distress by ensuring that patients not only comprehend the information communicated to them but also are satisfied with the amount of information they receive. Patients may also have an easier time waiting to the extent that they know what to expect. If medical staff and healthcare facilities provide information to patients about typical waiting experiences in the form of counseling or informational pamphlets, patients may be better able to endure the wait for health news.

Additionally, word use during medical visits, much like the quality of rapport between physician and patient, is associated with important patient outcomes such as patients' feelings toward their physician and adherence to treatment recommendations (Falkenstein et al., 2016). For example, patients liked physicians more when physicians used fewer negative emotion words and reported greater adherence intentions when physicians used less "I" language. Further investigation of word use may illuminate similar relationships with patients' waiting experiences after a medical consultation, pointing to a relatively simple way to decrease distress while patients wait.

Medical waiting periods are unavoidable and generally stressful experiences for patients. As research on extended periods of health-related uncertainty burgeons, we hope to find ways for patients and healthcare providers to ease the burden of waiting for health news.

Author Biographies

Angelica Falkenstein is a doctoral candidate in Social/Personality Psychology program at the University of California, Riverside. Her research focuses on expectation management processes as people anticipate important news in their lives and how expectations for news relate to other key waiting experiences such as emotion regulation and cognition. Angelica is also interested in waiting experiences in the context of healthcare and how such experiences and healthcare interactions predict patient outcomes.

Michael D. Dooley is a doctoral candidate in the Social/Personality Psychology program at the University of California, Riverside. Mike's interest focuses on the provision of social

support in close relationships, particularly when support needs are uncertain or unclear. How do supportive others recognize when support is desired? How do they decide what support to provide? His research covers supportive behaviors and decision making in a variety of contexts, including waiting for uncertain health news.

Kate Sweeny is an associate professor of psychology at the University of California, Riverside. Her work seeks to understand the experience of awaiting uncertain news. What makes waiting so stressful? Do some people have an easier time waiting than others? Are there ways to “wait well?” She addresses these questions in contexts ranging from the wait for an exam grade to the wait for biopsy results, news of pregnancy, and the outcome of medical tests.

References

- Armor, D. A., Massey, C., & Sackett, A. M. (2008). Prescribed optimism: Is it right to be wrong about the future? *Psychological Science*, 19(4), 329–331. doi:10.1111/j.1467-9280.2008.02089.x
- Boivin, J., & Lancaster, D. (2010). Medical waiting periods: Imminence, emotions and coping. *Women's Health*, 6(1), 59–69. doi:10.2217/whe.09.79
- Csikszentmihalyi, M., & Rathunde, K. (1993). The measurement of flow in everyday life: Toward a theory of emergent motivation. In J. E. Jacobs (Ed.), *Nebraska symposium on motivation, 1992: Developmental perspectives on motivation. Current theory and research in motivation* (Vol. 40, pp. 57–97). Lincoln, NE: University of Nebraska Press.
- Falkenstein, A., Tran, B., Ludi, D., Molkara, A., Nguyen, H., Tabuenca, A., & Sweeny, K. (2016). Characteristics and consequences of word use in physician-patient communication. *Annals of Behavioral Medicine*, 50, 664–677.
- Harkness, K., Morrow, L., Smith, K., Kiczula, M., & Arthur, H. M. (2003). The effect of early education on patient anxiety while waiting for elective cardiac catheterization. *European Journal of Cardiovascular Nursing*, 2(2), 113–121. doi:10.1016/S1474-5151(03)00027-6
- Krizan, Z., Miller, J. C., & Johar, O. (2010). Wishful thinking in the 2008 US presidential election. *Psychological Science*, 21(1), 140–146. doi:10.1177/0956797609356421
- Lang, E. V., Berbaum, K. S., & Lutgendorf, S. K. (2009). Large-core breast biopsy: Abnormal salivary cortisol profiles associated with uncertainty of diagnosis. *Radiology*, 250(3), 631–637. doi:10.1148/radiol.2503081087
- Lebel, S., Jakubovits, G., Rosberger, Z., Loiselle, C., Seguin, C., Cornaz, C., ... Lisbona, A. (2003). Waiting for a breast biopsy: Psychosocial consequences and coping strategies. *Journal of Psychosomatic Research*, 55(5), 437–443. doi:10.1016/S0022-3999(03)00512-9
- Maxwell, J. R., Bugbee, M. E., Wellisch, D., Shalmon, A., Sayre, J., & Bassett, L. W. (2000). Imaging-guided core needle biopsy of the breast: Study of psychological outcomes. *The Breast Journal*, 6(1), 53–61. doi:10.1046/j.1524-4741.2000.98079.x
- Montgomery, M., & McCrone, S. H. (2010). Psychological distress associated with the diagnostic phase for suspected breast cancer: Systematic review. *Journal of Advanced Nursing*, 66(11), 2372–2390. doi:10.1111/j.1365-2648.2010.05439.x
- Nelson, S. K., Robbins, M. L., Andrews, S. E., & Sweeny, K. (2015). Disrupted transition to parenthood: Gender moderates the association between miscarriage and uncertainty about conception. *Sex Roles*. doi:10.1007/s11199-015-0564-z
- Norem, J. K., & Cantor, N. (1986). Defensive pessimism: Harnessing anxiety as motivation. *Journal of Personality and Social Psychology*, 51(6), 1208–1217. doi:10.1037/0022-3514.51.6.1208
- Northouse, L. L., Jeffs, M., Cracchiolo-Caraway, A., Lampman, L., & Dorris, G. (1995). Emotional distress reported by women and husbands prior to a breast biopsy. *Nursing Research*, 44(4), 196–201.
- Novy, D. M., Price, M., Huynh, P. T., & Schuetz, A. (2001). Percutaneous core biopsy of the breast: Correlates of anxiety. *Academic Radiology*, 8(6), 467–472. doi:10.1016/S1076-6332(03)80617-7

- Phelps, C., Bennett, P., Iredale, R., Anstey, S., & Gray, J. (2006). The development of a distraction-based coping intervention for women waiting for genetic risk information: a phase 1 qualitative study. *Psycho-Oncology*, 15(2), 169–173.
- Pineault, P. (2007). Breast cancer screening: Women's experiences of waiting for further testing. *Oncology Nursing Forum*, 34(4), 847–853. doi:10.1188/07.onf.847-853
- Poole, K., Hood, K., Davis, B. D., Monypenny, I. J., Sweetland, H., Webster, D. J. T., ... Mansel, R. E. (1999). Psychological distress associated with waiting for results of diagnostic investigations for breast disease. *The Breast*, 8, 334–338.
- Schnur, J. B., Montgomery, G. H., Hallquist, M. N., Goldfarb, A. B., Silverstein, J. H., Weltz, C. R., ... Bovbjerg, D. H. (2008). Anticipatory psychological distress in women scheduled for diagnostic and curative breast cancer surgery. *International Journal of Behavioral Medicine*, 15(1), 21–28. doi:10.1007/BF03003070
- Scott, D. W. (1983). Anxiety, critical thinking and information processing during and after breast biopsy. *Nursing Research*, 32, 24–28.
- Shepperd, J. A., Ouellette, J. A., & Fernandez, J. K. (1996). Abandoning unrealistic optimism: Performance estimates and the temporal proximity of self-relevant feedback. *Journal of Personality and Social Psychology*, 70(4), 844. doi:10.1037/0022-3514.70.4.844
- Stanton, A. L., & Snider, P. R. (1993). Coping with a breast cancer diagnosis: A prospective study. *Health Psychology*, 12(1), 16–23. doi:10.1037/0278-6133.12.1.16
- Sweeny, K., & Andrews, S. E. (2014). Mapping individual differences in the experience of a waiting period. *Journal of Personality and Social Psychology*, 106(6), 1015–1030. doi:10.1037/a0036031
- Sweeny, K., Andrews, S. E., Nelson, S. K., & Robbins, M. L. (2015). Waiting for a baby: Navigating uncertainty in recollections of trying to conceive. *Social Science & Medicine*, 141, 123–132. doi:10.1016/j.socscimed.2015.07.031
- Sweeny, K., Carroll, P. J., & Shepperd, J. A. (2006). Is optimism always best? Future outlooks and preparedness. *Current Directions in Psychological Science*, 15(6), 302–306. doi:10.1111/j.1467-8721.2006.00457.x
- Sweeny, K., & Cavanaugh, A. G. (2012). Waiting is the hardest part: A model of uncertainty navigation in the context of health news. *Health Psychology Review*, 6(2), 147–164. doi:10.1080/17437199.2010.520112
- Sweeny, K., & Falkenstein, A. (2015). Is waiting the hardest part? Comparing the emotional experiences of awaiting and receiving bad news. *Personality and Social Psychology Bulletin*, 41(11), 1551–1559. doi:10.1177/0146167215601407
- Sweeny, K., & Falkenstein, A. (2017). Even optimists get the blues: Interindividual consistency in the tendency to brace for the worst. *Journal of Personality*, 85(6), 807–816.
- Sweeny, K., & Krizan, Z. (2013). Sobering up: A quantitative review of temporal declines in expectations. *Psychological Bulletin*, 139(3), 702–724. doi:10.1037/a0029951
- Sweeny, K., Reynolds, C. A., Falkenstein, A., Andrews, S. E., & Dooley, M. D. (2016). Two definitions of waiting well. *Emotion*, 16(1), 129–143. doi:10.1037/emo0000117
- Thorne, S. E., Harris, S. R., Hislop, T. G., & Vestrup, J. A. (1999). The experience of waiting for diagnosis after an abnormal mammogram. *The Breast Journal*, 5(1), 42–51. doi:10.1046/j.1524-4741.1999.005001042.x
- Uchino, B. N. (2009). Understanding the links between social support and physical health: A life-span perspective with emphasis on the separability of perceived and received support. *Perspectives on Psychological Science*, 4(3), 236–255. doi:10.1111/j.1745-6924.2009.01122.x

Suggested Reading

- Montgomery, M., & McCrone, S. H. (2010). Psychological distress associated with the diagnostic phase for suspected breast cancer: Systematic review. *Journal of Advanced Nursing*, 66(11), 2372–2390. doi:10.1111/j.1365-2648.2010.05439.x

- Pineault, P. (2007). Breast cancer screening: Women's experiences of waiting for further testing. *Oncology Nursing Forum*, 34(4), 847–853. doi:10.1188/07.onf.847-853
- Sweeny, K. (2012). Waiting well: Tips for navigating painful uncertainty. *Social and Personality Psychology Compass*, 6(3), 258–269. doi:10.1111/j.1751-9004.2011.00423.x
- Sweeny, K., & Cavanaugh, A. G. (2012). Waiting is the hardest part: A model of uncertainty navigation in the context of health news. *Health Psychology Review*, 6(2), 147–164. doi:10.1080/17437199.2010.520112

Index

- 23andMe 89
 - longitudinal study 92
- absent-exempt heuristic 522
- accelerometers
 - output (counts per unit time) 448
 - selection 448
 - validation studies, results 453
 - validity outcomes 453
- accelerometry
 - application 449
 - electronic diaries, combination 454–455
- accelerosensors, usage 447–448
- acceptance (grief stage) 43
 - occurrence/increase 44
- acceptance cues, overlooking 618
- acculturation
 - alcohol use, relationship 3
 - bicultural integration 6–7
 - cigarette use, relationship 3
 - conceptual/theoretical considerations 6–7
 - contemporary models, bidimensional/multicomponent process 2
 - cultural stress, separation 4
 - differential acculturation theory (DAT) 5
 - illicit drug use, relationship 3
 - longitudinal/epidemiological studies 9
 - methodological concerns 8–9
 - multidimensional construct 7
 - multidimensional model 6
 - receiving context, effects (understanding) 8
 - substance use
 - mechanisms, understanding 4–6
 - substance use, relationship 1, 2–3
 - unidimensional studies 5
 - unidimensional view 2
 - within-group diversity 8
- accuracy
 - consequences, benefits 14
 - defining 13–14
 - downsides 17–18
 - interpersonal perception, accuracy 13
 - motivation 582–583
 - “realistic accuracy” approach 13
- acquired immunodeficiency syndrome (AIDS)
 - beliefs, assessment 495
 - mortality, risk 486
 - partner loss, coping 415–416
 - risk 495
- action
 - change stage 367
 - control, top-down/bottom-up fashion 333
 - planning 187–188
 - plans 315
 - absence 319
- active engagement (relationship-coping strategy) 66
- activity-based variables 452
- acute discrimination 232
- acute grief, distress 44
- acute infections, death (reduction) 61

- acute prototype 318
 - chronic prototype, contrast 318
- adaptation
 - coping, contrast 65–66
 - core systems, operation 273
 - protective/vulnerability processes, impact 271
- adaptation neglect 22
 - impact 23
 - reduction, interventions 23–234
- adaptive tasks framework 62
- addiction risk 159
- additive counterfactuals 72
- adequacy, self-perceptions (challenges) 587
- adherence 395. *See also* patient adherence
 - achievement (factors), information-motivation-strategy model (usage) 399
 - educational interventions 399–400
 - electronic monitors, accuracy 398
 - improvement, interventions 399–400
 - measurements 398
 - rates, increase (economic benefits) 397
 - routines, facilitation 323
- adjustment
 - approach-oriented coping, benefits 65
 - good adjustment 66
- adolescents
 - health risk behavior 517
 - PWM research 518–519
- adrenocorticotrophic hormone (ACTH) levels, elevation 663–664
- adults
 - life (well-being challenges), physical/social pain (impact) 459
 - PWM research 519
- Adverse Childhood Experiences (ACE) research project 257–258
- adversity exposure 271
- affect-based risk perception, characteristics 552–553
- affect heuristic 552–553
 - consequence/implication 553
- affective forecasting 21
 - health, relationship 21–22
- affective states, perception 611
- Affordable Care Act, enrollment process (streamlining requirement) 293
- African Americans (sickle cell disease) 169
- age, disability (relationship) 169
- agency 218
- Agency for Healthcare Research and Quality (AHRQ) 570
- aggressive tendencies, origins 36
- agreeableness 412, 423–424
 - conscientiousness, connection 434
 - health behaviors, relationship 423–424
 - tendency 433–434
- alcohol
 - consumption, sex differences 634
 - myopia theory 600
 - overconsumption 159
 - use 729
 - acculturation, connection 3
 - increase, agreeableness (relationship) 424
- Alcohol Attention-Control Training Program (AACTP) 332
- Alcoholics Anonymous (AA), enrollment 219
- All Aspects of Health Literacy Scale (self-report measure) 288
- allocations, perception (dispositional tendencies) 227
- allostasis 272
- allostatic capacities, taxation 273
- allostatic load 272 541
- Alzheimer's disease (AD), anxious attachment 340
- ambiguity
 - aversion 50–51
 - meaning 50
- ambiguous information 49, 50
- ambulatory activity assessment, impact 453
- ambulatory activity monitoring 454–455
 - impact 449
 - reactivity, findings 454
- ambulatory assessment methods 374–375
 - Electronically Activated Recorder (EAR), contrast 378–379
 - longitudinal ambulatory assessments 374–375
 - physiology/experience 375
- American College of Sports Medicine (ACSM)
 - intervention techniques 197
- American Indians, suicide 170
- amygdala, threat detection involvement 698
- anchoring-and-adjustment heuristic 549
- anger (grief stage) 43
- anger, trait hostility (association) 530
- anorexia nervosa, anxiety/avoidance (report) 34
- antecedents 520
- anterior insula
 - activity, reduction 460–461
 - threat detection involvement 698
- anterior temporal pole, activity (increase) 35
- anticipated discrimination, differentiation 232
- anticipatory coping 56

- anxiety
 - disorders 256
 - elicitation 783
 - excessive counterfactual, connection 73
 - pattern (nonmedical waiting periods) 782
 - reduction, smoking (usage) 34
 - role 781–782
- appraisal-coping cycle 64
- appreciation
 - feelings, oscillatory systems coherence/entrainment (relationship) 264
 - gratitude, feelings 261
- approach, avoidance (contrast) 65
- approach/avoidance tasks, bias training usage 332
- approach bias training 332–333
- approach imagery 610
- approach-oriented coping, benefits 65
- artificial intelligence (AI) research, usage 51
- Ashkenazi Jews, Tay-Sachs disease 170
- Asians, liver cancer 169
- asset factors, effects 270–271
- asthma (chronic inflammatory process) 322
- Asthma (Puerto Ricans) 170
- asthmatics, bronchoconstriction (physiological measure) 477
- atherosclerosis 256
 - physiological pathway (optimism) 387
- attachment
 - anxiety 542
 - predictions 36
 - avoidance
 - dimension 32
 - physical health, link 32–33
 - capacity 269
 - defining 32
 - differences, health behavior (link) 33–35
 - dyadic influence 340
 - health
 - behavior, relationship 340
 - relationship 339–340
 - individual differences 31, 36
 - physical health outcomes, linking mechanisms 33–37
 - insecurity 339–340
 - orientation 31, 32
 - physical health, link 32–33
 - social support, link 33
 - physical health
 - associations, examination 38–39
 - correlates 339–340
 - problems, emotional regulation skill problems (association) 35
 - processes, health (relationship) 31
 - theory
 - dyadic framework 37–38
 - proposal 43
- attentional focus, target (inhibition) 558
- attention-allocation model 600
- attentional retraining 331–332
 - repetition, randomized controlled trial (RCT) 332
- attentional skills, application 137
- attention deficit hyperactivity disorder (ADHD) 449
- attention, reduction 669
- attitudes 521
 - definition 747–748
 - situation, comparison 248–249
 - subjective norms/attitudes 521
- autobiographical narratives
 - collection 218
 - data, representation 217
 - quantification 218–219
 - transformational processing 219
- autobiographical reasoning 216
- autobiographical scenes, providing 217
- autoimmune conditions, childhood trauma (link) 257–258
- automatic decisions 162
 - neuroendocrine axis 659
- autonomic nervous system (ANS)
 - activity, modulation 664
 - oscillatory systems coherence/entrainment, appreciation feelings (relationship) 264
 - rumination, relationship 560
 - sleep disturbances, impact 541
 - sympathetic branch, activation 256
- autonomy 206
- avoidance. *See* treatment seeking/avoidance
 - approach, contrast 65
 - avoidance-oriented coping, impact 310
 - demographic predictors 280–281
 - dimension 32
 - health behavior risks, association 34
 - healthcare treatment seeking, relationship 297
 - negativity 300–301
 - prediction, education (impact) 281
 - strategies 413
- avoidant attachment
 - characteristics, impact 32–34
 - immune function, link consistency (reduction) 36
- Bandura, Albert 185
- bargaining (grief stage) 43

- baseline comparisons, formation 316–317
- Beall, Sandra 149–150
- Beecher, Henry 475
- behavior
 - assessment 375
 - attitudes 756
 - behavior-related moderators 356–358
 - counterfactuals, relationship 75–76
 - effectiveness, belief 213
 - expectations 520
 - identification/defining (PAPM step) 502
 - interference, stress/distress/dysregulated emotion (impact) 135
 - intervention
 - materials, usage 196–198
 - process 195–196
 - target, identification 194–195
 - maladaptive behaviors, engagement 135
 - modification 366
 - moment-salient behaviors 161
 - nature 193–194
 - negative behaviors (increase), counteractuals (impact) 76
 - one-way behavior change, occurrence 76–77
 - prediction, health belief model (usage) 213–214
 - present codings, absent codings (contrast) 377–378
 - real-time capture, EAR (usage) 376–377
 - types 519
 - willingness, definition 517–518
- behavioral aggression 530
- behavioral avoidance 65
- behavioral change, health belief model 212
- behavioral coping 65
- behavioral goals 194
- behavioral strategy 186
- behavioral willingness 185
- behavior change
 - active ingredients 198
 - difficulty 502
 - interventions 758–759
 - development/evaluation, PAPM (usage) 502–503
 - model 364
 - norms, usage 727–729
 - occurrence 76
 - persuasion, role 747
 - stage theories, impact (process) 495–496
 - theories, cataloguing attempt 183
- behavior maintenance
 - description 203–204
 - factors 205–206
 - importance 203
 - interventions/research, directions 207
 - operationalization process 204–205
 - promotion 206
 - regulatory focus, effect 206
 - satisfaction, role 205
 - self-determination theory (SDT), application 206
- beliefs, meanings (variation) 316
- benefit finding 154
- bereavement 43
 - acceptance, occurrence/increase 44
 - adaptation, trajectories 44
 - broken-heart phenomenon 45
 - persistent complex bereavement disorder (PCBD) 44
 - physiological changes 45
 - widowhood effect 45
- beta-band frequency (physiological measure) 477–478
- bias
 - bias-reduction strategies, success 513–514
 - documentation 512
 - reduction 105
 - role 14
 - training, usage 332
- biased treatment 232
- bicultural identity integration (BII) 6–7
- bicultural integration 6–7
- big data collection 381
- binge drinking intentions (reduction), counterfactual thinking (impact) 77
- biography, cultural concept 216
- biological embedding, theory 257–258
- biological hyperarousal, reduction 273
- biological processes, impact 35–36
- biological third variables, involvement 431
- biomarkers, measurability 134
- bipolar disorders 449
- Black patients, narcotic medication (prescription likelihood) 511
- blood pressure, spousal support (impact) 341
- bodily metaphoric framings, exposure (impact) 123
- bodily metaphors 122
 - experimental evidence 123–124
 - helpfulness, timing 125–126
 - implications 124–128
 - pain 126–128
 - qualitative evidence 122–123
 - qualitative linguistic analyses 124

- body
 - physiological systems, wear and tear 134
 - stress, impact 256
- body mass index (BMI) 649
 - gains, risk 652
 - increase, marriage (association) 653
 - social relationships, impact 651–652
- boosters, incorporation 179
- breast cancer
 - couples, coping 144
 - patients (engagement coping), optimism (association) 416
- Breast Cancer Literacy Assessment Tool 289
- broaden-and-build theory 490
- broken-heart phenomenon 45
- buffering model 660
- bulimia nervosa, anxiety/avoidance (report) 34
- cancer
 - adaptation, couple-relationships 81
 - adherence rates 396
 - cancer-related behaviors/emotions, uncertainty (association) 308–309
 - functional abilities (decline), care/assistance (couples discussion) 84
 - partner/family member care 81
 - patients/partners, QOL challenges 82–84
 - patients, spirituality (presence) 713
 - survival, increase 81
 - treatment, pattern challenges 83–84
 - war
 - metaphor 123–124
 - vocabulary 126
- “Cancer by the Carton” (Norr) 114
- cancer screenings 570
 - decisions 21
 - guideline, conflicting information 49
 - improvement 569
 - uptake 572
- cardiac arrhythmias 256
- cardiovascular activity, rumination (association) 560
- cardiovascular ailments 257
- cardiovascular disease
 - high blood pressure, risk factor 387
 - optimism, impact 387
- cardiovascular health, relationships (impact) 342
- cardiovascular pathway, positive relationship interactions 342
- cardiovascular reactivity 256
 - increase, perceived prejudice/discrimination (impact) 233
 - reduction, friend social support (impact) 240
- cardiovascular responding 539
- cardiovascular smoking-related diseases, tobacco company refutations 114
- cardiovascular system, functioning (health indicator) 338
- care seeking 321
- car injury (likelihood), seat belt avoidance (relationship) 35
- casual sex, willingness (increase) 522
- causal geography 168
- cause (CSM component) 100
- cell-mediated immunity, optimism (relationship) 388
- Center for Epidemiological Studies Depression Scale (CESD) 485–486
- central nervous system, nonsteroidal anti-inflammatory analgesic (impact) 460–461
- central route processing 366
- challenge, attitude 440
- change. *See* behavior change
 - barriers 186
 - stages 186, 367
- childhood, peer rejection/victimization 766
- childhood trauma, autoimmune conditions (link) 257–258
- Choosing Life: Empowerment! Action! Results! (CLEAR) 197
- chronic conditions, adherence 321
- chronic disease, prevalence (growth) 116–117
- chronic illness
 - adaptation 62–64
 - cognitive adaptation 63–64
 - coping 61
 - defining 61–62
 - management/coping, social comparison (role) 248
 - stresses 62
 - timeline, length 62
- chronic pain
 - coping, positive affect (impact) 463
 - social interventions 464
 - vulnerability/distress, social contributors 461–462
- chronic prototype, acute prototype (contrast) 318
- chronic stress, resilience 717
- chronic trauma, experience 258
- chronotype, impact 672–673
- cigarettes
 - lethality, modification 193–194
 - smoking, addictive properties 159
 - tax, doubling (impact) 116
 - use, acculturation (relationship) 3

- circadian phase (chronotype) 672
- circulating pro-inflammatory cytokine levels,
 - optimism (relationship) 388
- clinical decision making, stereotyping/prejudice
 - (influence) 510–512
- clinical depression, diagnosis 531
- clinical disease-related outcomes, rumination
 - (link) 563
- clinical preventive services, USPSTF
 - recommendations 570
- clinical recommendations, stereotyping/prejudice
 - link (absence) 512–513
- coaching (self-efficacy belief source) 186
- codes of conduct, importance 709
- cognitive avoidance 65
- cognitive behavioral-based approaches 156
- cognitive closure, requirement 308
- cognitive coping 65
- cognitive dissonance theory 751
- cognitive economy, impact 582
- cognitive fatigue 179
- cognitive habits 736–737, 738
- cognitive mistrustfulness 530
- cognitive reasoning, capacity 269
- cognitive restructuring 45
 - emotion-focused coping activity 413
- cognitive space, freeing 177
- coherence 264
 - psychological sense, restoration 273
- college student drinking, personalized normative
 - feedback (context) 728–729
- colorectal cancer (CRC), perceived risk 573
- colorectal/prostate screening, uptake study 572
- commitment, attitude 440
- commitment, control, and challenge (three Cs)
 - 440–441
- commonsense model (CSM) 99, 315
 - acute prototype 318
 - baseline comparisons, formation 316–317
 - chronic conditions, adherence 321
 - components, assessment (timing) 320–322
 - descriptions 101
 - episodic prototype 318–319
 - framework 100
 - illness prototypes/representations, multilevel
 - process 318–320
 - long-term self-management, action
 - (automation) 322–323
 - method/theory, confusion 320
 - principles/structure 315–316
 - representations/prototypes 317–318
 - differentiation, importance 316–317
 - representations (activation), reasoned processes
 - (involvement) 317
 - structure/operation 316–323
 - treatments, prototypes/representations
 - (structure/content similarity) 319
 - use/misuse 320–323
- commonsense modeling (CSM) 315
- common sense, representation 321
- communication patterns (challenges), cancer
 - treatment (impact) 83–84
- communion 218
- community structure, geographic
 - differences 173
- comparative optimism 249
- comparison
 - mode 248
 - social comparison 247
- compensation hypothesis 609
- competence 206, 269
- compliance stereotyping 511
- complicated grief 44
 - development 45
 - randomized controlled trials 45
- compulsive self-sufficiency 34
- computer-tailored interventions 365–366
- concomitant adversity 271
- concrete metaphors, reliance 122
- conduct problems, hardiness (negative
 - relationship) 442
- confidence
 - issue 385
 - mechanism 581–582
- conflict
 - issue 48–49
 - physiological correlates 341
- conflicting health information 47
 - conceptual typology 48–49
 - working definition 48
- conflicting information
 - artificial intelligence (AI) research,
 - usage 51
 - construct, defining 47–48
 - evidence heterogeneity 49
 - multiplicity 49
 - related phenomena 49–50
 - sources, trustworthiness 52
 - temporal inconsistency 49
 - theoretical models 50–52
- confrontations, fear 125
- confuse, term (defining) 50
- confusing information 49, 50
- connectedness, perceptions 682

- conscientiousness 412, 422–423
 - agreeableness, connection 434
 - health-compromising behaviors, avoidance (relationship) 422–423
 - trait
 - associations 415
 - predictions 432
- conscious control, freedom 178
- consciousness raising 184
 - behavioral strategy 186
- conscious processes 178
- construals, role 738–739
- Consumer Assessment of Health Providers and Systems—Clinician and Group Surveys (CG-CAHPS) 404
- contamination 218
- contemplation (change stage) 186, 367
- contemplators (behavior) 521
- content goals 194
- context change interventions 330–331
- context-related moderators 356, 358–359
- contexts, defining (considerations) 430
- contingency management (behavioral strategy) 186
- continuous positive airway pressure (CPAP), treatment 579
- continuum models 183–186
 - effectiveness, comparison 188
 - target variables, setting 184
- control
 - attitude 440
 - control, life satisfaction pillar 684
 - locus, external causes 606
 - patterns (challenges), cancer treatment (impact) 83–84
 - perceived control, increase 284
- controllable stressors 705
- coping 413–414
 - adaptation, contrast 65–66
 - adjustment 416–417
 - appraisal 184
 - behaviors 141, 145
 - not-in-the-moment coping behavior 142
 - chronic illness 61
 - conceptualization 141–142
 - expansion, transactional models (usage) 55
 - contrasts 413–414
 - coping-relevant facets 144
 - counterfactuals, relationship 73–74
 - daily diaries 142–143
 - daily measurement 142
 - dyadic coping 66–67
 - ecological momentary assessment 142–143
 - family coping 67–68
 - five-factor model, relationship 415
 - in-the-moment coping strategies, EMA (impact) 143
 - measurement 141–142
 - naturalistic observation 143–144
 - optimism, relationship 415–416
 - personality, relationship 411, 414–416
 - perspectives 145
 - planning 187–188
 - proactive coping 56, 414
 - process 56
 - social influences 58
 - sibling coping 68
 - social context 57–68
 - social factors, positive effects 463
 - social threat, impact 462
 - strategies 55–56, 310
 - categories 65
 - selection 57
 - stress and coping paradigm 64–65
 - style 235
 - systemic transactional model (STM) 67
 - theories 64–65
 - theory building 145
 - transactional model 309–310
 - types, contrast 56
- coronary artery bypass surgery, short-term health outcome 386
- coronary heart disease (CHD)
 - diagnosis (risk increase), depression (association) 531
 - diagnosis/mortality, reliability 531
 - environmental factors 531–533
 - psychosocial factors 529
 - social support 532
 - trait hostility/anger ratings, elevation 530
 - work stress 531–532
- corticotrophin-releasing hormone (CRH), disruption 256
- cortisol
 - increase 642
 - social-evaluative threat (SET), impact 643–644
 - levels, disruption 256
 - partner levels 338
 - reactivity, increase 233–234
 - social-evaluative threat (SET), impact 641–642
 - reactivity, social-evaluative threat (SET) components (link) 642–643

- cortisol (*cont'd*)
 - release 36, 337–338
 - response, exaggeration 540
 - rumination, relationship 561
- Counterfactual Inference Test (CIT) 74
- counterfactuals
 - behavior, relationship 75–76
 - coping, relationship 73–74
 - counterfactual-driven behavior change, focus 76
 - health decisions/health-relevant behaviors 75–78
 - helpfulness 75
 - intentions, relationship 76–77
 - motivation, relationship 78
 - trauma survivor generation 73
- counterfactuals, types 71–72
- counterfactual thinking
 - binge drinking, relationship 77
 - consequences 77
 - links 73
 - reduction
 - Parkinson's disease, relationship 74
 - schizophrenia, relationship 74–75
- counterfactual thought 71
 - deficits 74–75
 - excess 72–73
 - personal control, chronic deficits (role) 73
 - prevalence, elevation 73
- counterstereotyping 513
- couple-based interventions 84–85
 - confidence intervals 85
 - randomized controlled trials, review/meta-analysis 84
 - weighted mean effect sizes 85
- couple-relationships, cancer adaptation 81
- cravings 163
- cue-behavior implicit associations, strength 179
- cultivation hypothesis 609
- cultural competency (psychosocial concept) 226
- cultural differences
 - exploration 241–242
 - factors 357
- cultural identifications 2
- cultural practices 2
- cultural stress, acculturation (separation) 4
- cultural values 2
- culture, impact 424, 650
- daily diaries
 - methodologies, benefits 142
 - usage 142–143
- daily events (knowledge improvement), EMA (impact) 142–143
- data check 451
- data reliability, limitations 50
- deceased, kinship relationship 44
- decisional remorse, avoidance 580–581
- decision making, frontal lobes (involvement) 74–75
- decision-making processes, emotion (integration) 75
- decision simplification 330–331
- deeming rule 116
- deep-level prototypes, disease-specific prototypes (connection) 318
- de facto selective exposure 580
- defensive resistance 589–590
- demand-control hypothesis 531
- demand-withdrawal communication patterns, increase 341
- demand-withdraw interactions 377
- demographics, DTC genetic testing 90
- demographic variables, patient satisfaction (relationship) 406
- denial (grief stage) 43
- depression
 - CHD diagnosis, risk increase (association) 531
 - decrease 135
 - emotion regulation difficulties 35
 - excessive counterfactual, connection 73
 - grief stage 43
 - metaphor 122
 - metaphorical understanding 124
 - risk, magnification 271
 - social exclusion, relationship 460
 - stress-reactive rumination, role 558
- depressive symptoms 154
 - HPA functioning, disruption (relationship) 256
 - reduction, R/S (impact) 712
- Descartes, René 616
- descriptive norms 572–573
 - motivational underpinnings 725–726
 - screening behaviors, relationship 727
- destination, metaphor 122
- developmental status, consideration 274
- diabetes, adherence rates 396
- Diabetes Numeracy Test 289
- diagnosis (delay), rumination (effect) 563
- diagonals (behavior) 520–521
- DiClemente, Carlo 186
- diet
 - culture, impact 649–650
 - differences, impact 172

- family relationships 652–653
- Lifestyle Triple P intervention 653
- modeling, impact 653–654
- parenting 652–653
- romantic relationships, effects 653
- social factors 649
- social networks, effects 652
- social norms 650–651
- social support 651–652
- socioeconomic status (SES) 650
- differential acculturation theory (DAT) 5
- direct cultural geographic factors 171–173
- direct effects 232
 - model 240, 241
 - systemic/interpersonal mistreatment 232–233
- direction, metaphor 122
- direct physical geographical factors 170–171
- direct-to-consumer (DTC) genetic testing 89
 - advertising 92
 - barriers/benefits, perception 91
 - consequences 92–94
 - demographics 90
 - health behaviors, changes 93
 - healthcare professional consultations 92–93
 - knowledge/awareness 91
 - psychological effects 93
 - psychosocial predictors 90–92
 - regret, anticipation 92
 - results
 - comprehension 93
 - misinterpretation, possibility 91
 - viewing, avoidance 93–94
 - risk framing 902
 - usage/nonusage, self-reported reasons 90–91
- disability, age (relationship) 169
- disability paradox 23
- discounting, theory 162
- discrimination
 - adult reports 234
 - direct effects 232–233
 - effect, moderators 235–236
 - forms 232
 - health consequences 231
 - indirect effects 233–235
 - perception/coping, fairness beliefs
 - (impact) 236
 - screening, relationship 574–575
 - stress, coping 235
 - systemic/interpersonal mistreatment 232–233
 - terms, defining 231–235
- disease
 - burden, health behaviors (relationship) 363
 - chronic disease, prevalence (growth) 116–117
 - disease-prone personalities, health trajectories 430
 - disease-related variables, impact 99
 - disease-specific prototypes, parameters (impact) 318
 - disease-specific worry 97
 - genetic screening, association 573–574
 - onset 529
 - pathogenesis, effects 538
 - perceived risk 571
 - perceptions 249
 - proneness 430
 - recovery, relationship quality (connection) 339
 - risk, learning (avoidance) 282
 - rumination, impact 563–564
 - severity, meta-analysis 299
 - social comparison, relationship 248–249
 - susceptibility 131
 - worry, degree (determination) 99–101
 - worry, question 98–99
- disengagement
 - avoidant techniques 414
 - coping, engagement coping (contrast) 414
 - disengagement-related strategies 415
- dispositional optimism 309
- dissonance, reduction 588
- distal motivational goals 742
- distal TMHM responses 743
- distant intercessory prayer 712–713
- distress
 - experience 131
 - minimization 33
 - expression, minimization 33
 - mitigation, avoidance-oriented coping (impact) 310
 - psychological distress, increase 81
 - reduction, avoidance (usage) 57
 - social contributors 461–462
- distributive justice
 - beliefs 227
 - social justice, relationship 224–226
- divorce
 - likelihood 15
 - psychosocial effects, immune system (link) 540
- “Do Defaults Save Lives?” (Johnson/Goldstein) 197
- domain-specific self-efficacy, measurement 608
- dorsal anterior cingulate cortex (dACC)
 - activation levels, increase 459
 - activity, reduction 460–461
 - threat detection involvement 698
- downward comparisons 63–64
 - role, evaluation 249

- downward counterfactuals 71
 - likelihood 74
- downward social comparison, addition (impact) 251
- dramatic relief (behavioral strategy) 186
- drug use (increase), agreeableness (relationship) 424
- dual effects hypothesis 690
- dual-process account 519
- dual-process approaches 162–165
- dual processing, evidence 522, 523–524
- dual-process models 520
 - theme 178
- dual-process theories 160, 161
- Duval, Shelley 597
- dyadic coping 66–67
 - partners, benefit 67
 - promotion 83
- dyadic framework 37–38
- dyads, focus 58
- dysregulation emotion, impact 135
- early trauma, experience 258
- eating disorders, appearance concerns 601
- eating, social facilitation (trigger) 651
- Ebola outbreak (2014), American response 348
- ecological momentary assessment (EMA) 105, 142–143
 - advantages 106–107
 - characteristics 105–106
 - data collection approaches, extension 110
 - event-contingent EMA methods, usage 108
 - measurement considerations 107–108
 - methodology, longitudinal nature (advantage) 107
 - methods
 - daily life capture (ability) 109
 - development 106
 - directions 110–111
 - usage, opportunities/benefits 110
 - sampling approaches 108–109
 - studies, limitations 108
 - success/applications 109–110
 - time-based EMA sampling approaches, usage 109
 - timing, attention 105–106
 - usage, increase 145
- Ecological Momentary Intervention (EMI) 110
- education, discrimination 233
- efficaciousness (self-efficacy belief source) 186
- effort-reward imbalance model 531
- elaboration likelihood model (ELM) 366, 748–749
 - principles 749
- Electronically Activated Recorder (EAR) 143–144, 376–379
 - ambulatory assessment methods, contrast 378–379
 - challenges 379
 - complex/molar psychological construct 377
 - data, psychometric properties (assessment) 378
 - iEAR app
 - privacy button 378
 - randomization feature 377
 - information capture 378
 - method, ethical considerations 378–379
 - methodology, usage 379–380
 - sampling rate, programming 376–377
 - sound files, analysis process 377–378
- electronic diaries, accelerometry (combination) 454
- embodied cognition, perspectives 121
- embodied health 121
- emergency care setting, care seeking 321
- emotional anger 530
- emotional approach coping 65
- emotional/cognitive processes, cravings/desires (impact) 163
- emotional consequences, prediction (accuracy) 27
- emotional disclosure 135–136
- emotional distress 704
 - management 64–65
 - pain-related emotional distress 459
- emotional eating, discrimination (association) 234
- emotional experience/response, dynamic features 133
- emotional expression, enhancement/suppression 136–137
- emotional health, adversity (impact) 271–272
- emotional intensity, prediction (accuracy) 26
- emotional moments, experience 97
- emotional pain, intensity (underestimation) 24
- emotional/physiological states 186
- emotional stability 424–425
- emotional states
 - experience 134
 - impact 164
- emotional worry, transfer 123
- emotion-focused coping
 - categories 413
 - efforts 55–56
 - emotional distress management 64–65
 - problem-focused coping, contrast 413–414

- emotion regulation 35, 131–133
 - differences, intrinsic/extrinsic factors 137
 - divorce, relationship 542
 - effects, differences 137
 - ineffectiveness 135
 - predictions 133–136
 - skill, individual/developmental variability 136–137
- emotions
 - context-appropriate regulation, engagement 136
 - emotion-related behavior, factors 133
 - forecast, accuracy 25
 - management 133
- EmotionSense apps, ability 381
- emotions, impact
 - overestimation 22–24
 - underestimation 24–25
- empathy gap
 - experience 24–25
 - possibility 511
- employment discrimination 233
- enabling hypothesis 609
- endocrine dysregulation, sleep disturbances (impact) 541
- endocrine pathway, positive relationship
 - interactions 341–342
- end-of-life arrangements, making 65
- endothelin-1 (vasoconstrictor role) 560
- energy/enthusiasm, decrease 669
- energy expenditure (EE) 447, 452
 - expenditure-based variables 452
- energy, loss 673
- engagement coping, disengagement coping (contrast) 414
- environmental cues, impact 163
- environmental variation, adjustment (biological mechanisms) 272
- environment, quality (policies) 174
- episodic prototype 318–319
- epistemic uncertainty, appearance 50
- epoch (time interval) 448
- escape model 601
- esteem contingencies, research 743
- estimation strategies 374
- ethnic disparities 233
- ethnicity, health literacy predictor 290
- ethnic minorities, physical activity (increase) 234
- evaluative conditioning (EC) tasks, pairings 332
- event-contingent EMA methods, usage 108
- events (importance), appraisal (stability) 26
- evidence heterogeneity 50
- evocation, pathway 431
- excessive counterfactual
 - depression/anxiety, connections 73
 - personal control, chronic deficits (role) 73
- executive control, loneliness (impact) 697
- executive functioning, compromise 669
- exercise, sex differences 634
- expectancy-value process models, usage 413–414
- expected subjective utility 549–550
- experience
 - ambulatory assessment methods 375
 - momentary data collection 106
 - openness 412
- experienced discrimination, differentiation 232
- experience sampling method (ESM) 106
- explicit bias 510
- explicit health-related attitudes/behaviors (change), persuasion (impact) 748–750
- explicit stereotyping/prejudice 508–509
- expressive writing (EW) 149
 - administration, guidelines 150
 - demand characteristics 151
 - effectiveness 150
 - health benefits 152–153
 - impact
 - mechanisms 155–156
 - studies 152
 - testing 154
 - individual differences 154
 - instructions, differences 154–155
 - intervention 154–155
 - medical outcomes 152–153
 - meta-analysis, usage 150
 - moderating factors 153–155
 - physical functioning 152–153
 - physical health, relationship 151–152
 - physiological outcomes 150
 - psychosocial functioning 153
 - research, early years 149–151
 - studies, implication 151
 - usage 542
 - uses/limitations 152
 - well-being, relationship 153
- extraversion 412
 - characteristics 432–433
 - health-compromising behaviors, relationship 423
- failure feedback, administration 600
- fairness, beliefs (impact) 236
- fair process effects 225
- false self, maintenance 601

- familial environments (parental psychological health impact), assessment (EAR usage) 380
- familial ethnic socialization 5
- family
 - coping 67–68
 - development system 5–6
 - family systems theory 67
 - interactions (investigation), EAR (usage) 380
 - members, social support (effect size) 240
 - unitary system (family systems theory) 67
- Family Smoking and Prevention Tobacco Control Act 116
- fatigue, feelings 673, 674
- fat-thin IAT 5109
- fear 184
 - appeals, role 748
 - emotion, activation 319
 - excess, problems 127
- feedback (self-efficacy belief source) 186
- feelings
 - feeling bad, avoidance 580–581
 - feeling good 580–581
 - impact 615
 - qualitative nature 552–553
- fibromyalgia, pain (adaptation) 463
- fight-or-flight response 134, 257, 429
- firearm use, legislation 172
- first-generation immigrants, heritage (reconciliation/integration) 6
- Fisher, Ronald 199
- five-factor model 412, 413, 415
- flourishing, term (usage) 765
- focalism 22
 - impact 23
 - results 25
- food
 - overconsumption 159
 - selection, culture (impact) 649–650
- Food and Drug Administration (FDA), deeming rule 116
- food choices
 - criticism 691
 - modeling, impact 653–654
- foot-in-the-door (FITD) technique 752
- forecast accuracy, promotion (process) 23
- forecasting
 - affective forecasting 21–22
 - bias, magnitude/direction (factors) 25–26
 - errors, recognition 24–25
 - strengths/weaknesses, identification 25–27
- foreign-born individuals, global south origin 1
- Framingham Heart Study (FHS) 652
- frequent electrodermal activity and event logging (FEEL) system 145
- friendship
 - health correlates/consequences 239
 - epidemiological evidence 239–240
 - health link, mechanisms 240–241
 - individual differences 241–242
 - interventions
 - friends, usage 242
 - support groups, usage 242
 - negative influences 241
- frontal lobe, impairment 74–75
- fruits and vegetables (FV)
 - consumption 650
 - intake, environmental determinants 651
- fulfillment, cognitive factors 161–162
- functional abilities, decline 84
- functional social support, longitudinal studies 532
- future distress, overestimation 23
- future emotion, intensity (judgement) 25–26
- future events, emotional impact
 - overestimation 22–24
 - underestimation 24–25
- fuzzy-trace theory 51
- gain-framed message 355, 360
 - persuasion 358
 - responsiveness, increase 357
- Galen 429
- gambling, legislation 172
- gender-based discrimination 234
- general justice beliefs, descriptions 227
- general self-efficacy 609
 - concept, development 607
 - measurement 608
- genetic polymorphism, presence 539
- genetic screening 569–570, 573–574
- genetics-related self-efficacy, decrease (report) 83
- genetic test, completion (simplicity) 89
- genetic testing
 - decisions 21
 - web-based information, offering 574
- genomic technology, advances 574
- geography
 - causal geography 168
 - direct cultural geographic factors 171–173
 - direct physical geographical factors 170–171
 - dynamism 174
 - health, geography 167
 - indirect geographical factors 169–170

- geography as demography 167
- global self-reports, concerns 375
- glucocorticoid regulation, impact 540
- glucose, production 256
- goals
 - abandonment 625–626
 - automatic strategies 626–627
 - characteristics 624–625
 - cognitive strategies 627
 - differences, motivational focus (impact) 624–625
 - distal motivational goals 742
 - external imposition 625
 - goal-directed health behavior 601–602
 - health goals, adoption 624
 - planning strategies 626
 - pursuit, predictor 625
 - selection, sleep loss (impact) 674
 - setting 624–626
 - predictor 625
 - striving 626–628
- go/no-go task, bias training usage 332
- gratitude
 - causal influence, test 262
 - defining 261
 - health
 - behaviors, relationship 263–265
 - implications 261
 - link, moderators 265
 - outcomes, relationship 374
 - letters, writing (impact) 265
 - physical health, relationship 263–265
 - positive affect 262
 - psychological well-being, relationship 262–263
 - sleep, link (importance) 264
 - social relationships 263
 - trait gratitude, increase 264–265
- gravitational acceleration, measurement 448
- gravitational constant 448
- grief
 - acute grief, distress 44
 - complicated grief 44–45
 - coping 81
 - medicalization 45
 - occurrence, reasons 43
 - stages, theories 43–44
- grit, hardiness (connection) 443
- grounded cognition, perspectives 121
- group behavior basis, social identity (link) 679–680
- group identification, influence 235
- Groups 4 Health (G4H)
 - intervention 684–685
 - modules 685
- habits 177
 - defining 177–178
 - formation, process 179
 - health behavior 178
 - research 178–179
 - directions 179–180
 - theory 178
- habitual responses, triggering cue (association) 179
- hardiness
 - approach 439
 - evolution 441–442
 - assessment, development 440
 - conduct problems, negative relationship 442
 - grit 443
 - levels (males) 442
 - optimism, relationship 442
 - practice applications 443
 - predictors, comparison 442–443
 - studies (military personnel) 442
 - training (IBT managers) 443
- health. *See* public health
 - affective forecasting, relationship 21–22
 - attachment
 - dyadic influence 340
 - processes, relationship 31
 - relationship 339–341
 - beliefs, meta-analysis 299
 - causal role (playing), confidence (increase) 168
 - concerns, worry 98
 - condition, perceived susceptibility 358
 - consequences 231, 239
 - life stories correlates 215
 - coping, adjustment 416–417
 - decisions
 - counterfactuals, relationship 75–78
 - improvement 330–331
 - defining 261–262
 - discrimination/prejudice, effect (moderators) 235–236
 - dyadic framework 37–38
 - economics/policy, PRO role (increase) 110
 - embodied health 121
 - emotion
 - impact 223–224
 - regulation, prediction 133–136
 - explicit health-related attitudes/behaviors (change), persuasion (impact) 748–750

- health. *See* public health (*cont'd*)
- friendships, link
 - epidemiological evidence 239–240
 - mechanisms 240–241
 - friendships, negative influences 241
 - friends, influence 242
 - geographic influences 173–174
 - geography 167
 - goals, adoption 624
 - gratitude
 - health implications 261
 - link, moderators 265
 - health-compromising behaviors, harm 421
 - health-enhancing behaviors, relationships (impact) 339
 - health-promoting behaviors 589–590
 - analysis 240
 - health-protection action, rational thought 102
 - health-related attitudes
 - change, self-persuasion (impact) 750–752
 - persuasion, role 747
 - health-related behaviors
 - change, self-persuasion (impact) 750–752
 - research, directions 179–180
 - health-related information, understanding/communication (difficulty) 508
 - health-related intentions, associations (meta-analyses) 185
 - health-related policy, effects 113
 - health-related propositions 48
 - health-related social control, research 691
 - health-related social influence 691–692
 - health-related uncertainty 305
 - positive primary appraisal, achievement 310
 - health-relevant behaviors
 - counterfactuals, impact 75–78
 - engagement, psychological factors (influence) 599
 - health-relevant intentions, counterfactual thinking (consequences) 77
 - health-relevant pathopsychological pathway 539–540
 - health-relevant symptoms, self-perceptions 599
 - hormones, role 633
 - implicit health-related attitudes (change), persuasion (impact) 750
 - improvement
 - expressive writing (EW), impact/mechanisms 155–156
 - PA increase, impact (question) 490–491
 - policy, impact 113
 - inequity, social justice framework (connections) 225
 - information
 - conflict 47
 - self-affirmation/defensive resistance 589–590
 - intentions, prediction (ability) 757–758
 - latitude 171
 - maintenance 302
 - mental models, metaphor shaping 127
 - messages, framing recommendations 356
 - methodological themes/issues 485–487
 - natural disasters, impact 171
 - news
 - responses, waiting experiences (effects) 784
 - waiting 781
 - objective disease-specific health indicators, assessment 151
 - pastimes, relationship 172
 - pathways 430–432
 - positive affect (good feelings), impact 433
 - explanation 489–490
 - problem
 - discrimination/prejudice mechanisms 232
 - susceptibility, perception 212
 - traumatic experiences, association 257
 - traumatic exposures, impact 257
 - type/intensity 299
 - promotion
 - campaigns 193
 - PWM research 519
 - self-regulation, role 623
 - protective behaviors 726–727
 - psychology
 - findings 132
 - screening, relationship 570
 - self-efficacy, usage 609–610
 - psychosocial study, ecological momentary assessment 105
 - regional demographic differences 169–170
 - relationship dissolution, association 537
 - research, EAR methodology (usage) 379–380
 - resilience, health implications 269, 274–275
 - resource model 618–619
 - rumination, relationship 559
 - screening 195
 - selective exposure 579, 580
 - self-affirmation, relationship 587, 589
 - self-and-social-bonds model 619
 - self-awareness
 - relationship 597
 - research 599–602
 - self-efficacy, relationship 605
 - self-esteem, relationship 615
 - process 617–618
 - reasons 618–619

- sex differences, relationship 631
- social comparison, health correlates/
 - consequences 274
- social identity
 - approach, schematic representation 686
 - importance 680
- social isolation
 - impact 696
 - relationship 695
- social support, relationship 703
- spirituality/religiosity (S/R), relationship 709
 - mechanisms 710–712
- subjective health norms 725
- symptoms, attachment-related differences 34
- trauma, impact 256
- traumatic events, health effects 255
- treatment 302
 - evaluation, PRO role (increase) 110
- unrealistic optimism
 - benefits 776
 - costs 776–778
 - relationship 773
- WHO definition 261–262, 764
- health action process approach (HAPA)
 - 187–188, 571
- health behavior
 - attachment
 - differences, association 33–35
 - relationship 340
 - changes 183
 - description, prediction equation 496
 - DTC genetic testing results, impact 93
 - implicit processes, relationship 329
 - interventions, development/
 - implementation/evaluation 199
 - social cognitive theory applications 196
 - change theories, self-efficacy (usage) 609
 - cognitive/emotional perspectives,
 - integration 162–165
 - commonsense modeling (CSM) 315
 - cravings/temptations 163
 - engagement
 - likelihood 431
 - study 580
 - failed control 159
 - goal-directed health behavior 601–602
 - gratitude, relationship 263–265
 - improvement, social control attempts
 - (doubt) 690
 - indirect effects 234–235
 - interventions 193
 - modality, selection 197–198
 - success, determination 198–199
 - intrapersonal resources 163–165
 - involvement 431
 - maintenance 203
 - models, behavior maintenance (operationalization
 - process) 204–205
 - modification 397
 - norms, influences 726–727
 - personality
 - relationship 421
 - traits, association 422–425
 - prediction
 - ability 757–758
 - implicit attitudes, impact 750
 - reasoned action approach, usage 757
 - process models 204
 - promotion, conscious monitoring (increase) 331
 - relationship dissolution 540–541
 - relationship quality, impact 338
 - research 178–179
 - rumination, impact 562–563
 - sex differences 633–634
 - social norms, impact 331
 - stage models 204–205
 - theories 160
 - categorization 183–184
- health belief model (HBM) 184, 211, 316,
 - 367, 571
 - behavioral change, health belief model 212
 - cognitive constructs 212
 - constructs 212–213
 - expansion 212–213
 - history 211–212
 - interventions, assessment (aspects) 211–212
 - usage 213–214
- healthcare
 - avoidance 297
 - prejudice/stereotyping 507
 - occurrence/impact, reduction 513–514
 - professionals, DTC testing consultations
 - 92–93
 - setting, prejudice/discrimination 233
 - treatment seeking 297
 - treatment seeking/avoidance, context 300
- healthcare provider (HCP)
 - recommendation, absence 571–572
 - trust problems 235
- health information avoidance 279
 - affective concerns 281–282
 - behavior 279–280
 - challenge 282
 - causes 281–283
 - defining 279–280
 - demographic predictors 280–281

- health information avoidance (*cont'd*)
 - interpersonal motives 282–283
 - motives contemplation 283
 - perceived control, increase 284
 - personality measures 281
 - predictors 280–281
 - prevalence 280
 - problem 280
 - reduction 283–284
 - self-affirmation 283
 - self-view/worldview, challenge 282
- health literacy 287
 - adequacy, exception 292
 - improvement, interventions 291–293
 - low level, monetary cost 290
 - measures 288–289
 - medical decision making, relationship 290–291
 - NAAL measurement 289
 - NIH definition 287
 - physical health outcomes, relationship 291
 - predictors 289–290
 - ethnicity, impact 290
 - rates 289
 - skills, impact 287
 - visual aids/images, usage 292
- health outcomes
 - behavior maintenance, importance 203
 - dietary differences, impact 172
 - emotional impact forecasts, inaccuracy 23
 - gratitude, relationship 374
 - personality, relationship 429
 - prediction 373–374
 - relationship dissolution, association 542–543
- health risk
 - behaviors 726
 - social comparison, role 247
 - feedback, avoidance (reduction) 281
 - PWM research 519
- healthy habits, discussion 177
- healthy nudges 351–352
- heart attack mortality, decrease 167
- heart disease, risk (reduction) 634
- heart rate variability (HRV) 660
 - increase 662
 - reduction 661
 - RSA index 539
- helplessness, feelings (reduction) 66
- heteronomy 358
- heuristic cues, influence 366
- heuristic processing 517
- heuristics 749
 - strategies 374
- high blood pressure, long-term cardiovascular
 - activation (association) 256
- hippocampal cell density, reduction 35
- Hippocrates 429
- Hispanic/Latino populations, nationality
 - (importance) 8
- homeostatic function, maintenance 134
- homosexuality, APA perspective 635
- hormones
 - role 632
 - signals, release 660
- hostility
 - construct 530–531
 - potential 530
- hot/cool system theory 160–161
- housing
 - discrimination 233
 - disparities 232–233
- human immunodeficiency virus (HIV)
 - adherence rates 396
 - beliefs, assessment 495
 - regimes
 - adherence, health literacy predictor function 291
 - nonadherence, race differences 290
 - testing, racial contrast 575
- human papillomavirus (HPV)
 - female knowledge, study 308
 - vaccination, maintenance 204
- humor (emotion-focused coping
 - activity) 413
- hybrid third culture, emergence 7
- hyperarousal 4256
 - biological hyperarousal, reduction 273
- hypercoagulation 256
- hypertension, representations (coherence) 321
- hypocrisy approach 751
- hyporeactivity 539
- hypothalamic-pituitary-adrenal (HPA)
 - activation 619
 - activity
 - impact 257
 - modulation 664
 - axis 659
 - functioning, cortisol (relationship) 661
 - chronic activation, problem 36
 - stress-responsive HPA axis, discrimination
 - activation 233–234
- hypothalamic-pituitary-adrenal (HPA) axis
 - activation 35–36, 228, 256–258
 - relationship quality/endocrine function
 - research 337–338

- identity (illness domain) 99
- if-then plans, formation 333
- illicit drug use, acculturation (relationship) 3
- Illinois Bell Telephone (IBT) project 440–441
 - mangers, hardiness training 443
- illness
 - adaptation 62–64
 - sibling coping 68
 - cause (CSM component) 100
 - commonsense model (CSM) 99
 - dynamic process, CSM descriptions 101
 - emotion, impact 223–224
 - identity/cause 99
 - likelihood estimates, connection 99–100
 - mastery, regaining 63
 - meaning making 63
 - pathways 430–432
 - prototypes/representations, multilevel
 - process 318–320
 - representation 316
 - domains 99
 - impact 320
 - risk, representations (impact) 101
 - self-esteem, restoration 63–64
 - social comparison, relationship 248–249
 - susceptibility 131
 - symptoms, impact 100
 - timeline
 - likelihood estimates, connection 99–100
 - perceptions 100
 - work-related illness, risk 531
- Illness Perception Questionnaire (IPQ/IPQ-R) 320
- immune cell function, decrease 36
- immune functioning 539–540
- immune function, non-cardiovascular disease
 - outcomes (relationship) 387–389
- immune pathway, positive relationship
 - interactions 342–343
- immune response
 - non-specific immune responses, suppression 561
 - optimism, negative relationship 388–389
 - stress, association 540
- immune system
 - divorce/separation, psychosocial effects (link) 540
 - impact 460
 - rumination, impact 561
- impact bias 22
 - reversal 25
- implementation intentions 330, 736
 - perspective 737–738
- Implicit Association Test (IAT) 510
 - fat-thin IAT 510
- implicit bias 512
 - assessment 509–510
 - research 224–225
- implicit health-related attitudes (change), persuasion (impact) 750
- implicit learning (cognitive processing measure) 477
- implicit processes
 - health behavior change, relationship 329
 - inclusion 330
- implicit stereotyping/prejudice 509–510
- impulses, effortful inhibition 628
- impulsive information processing 329
- inactive treatments, effects (alteration) 478
- income, geographical differences 173
- income inequality, impact 173
- indirect effects 232–235
 - health behaviors 234–235
 - stress 233–234
- indirect geographical factors 169–170
- individual-difference variables/characteristics 363–364
- individuation 513
- infant bonding 43
- infant mortality, analysis 173
- infection
 - condition severity, perception 212
 - risk, increase 538
- infectious diseases, marital status (association) 538
- inflammation, loneliness (operation) 698
- inflammation-related diseases, reporting (likelihood) 38
- information
 - information-processing modes, types 329
 - information-related psychological phenomena 279–280
 - overload 349
 - processing, validity (requirement) 308
- informational justice 225–226
- informational norms 651
- information avoidance
 - definition 279
 - prevalence, variation 280
- information-motivation-strategy model 399
- inhibition process 735
- inhibitory skills, application 137
- injunctive norms 572
 - motivational underpinnings 725–726
 - priming 331
 - screening behaviors, relationship 727

- in-office genetic testing 91
- insomnia
 - creation, stressors (impact) 670–671
 - formation/development, stress markers (impact) 671
 - risk (elevation), separation/divorce (impact) 541
- intellect, impact 424, 434
- intent, action (mediation) 187–188
- intentional nonadherence 396
- intentions
 - counterfactuals, relationship 76–77
 - strengthening 76–77
 - willingness
 - comparison 520
 - relationship 521
- interactional justice 225–226
- intercessors, recruitment 712
- interference effect 609
- interference hypothesis 609
- interleukin 6 (IL-6)
 - blood levels, reduction 342
 - levels, increase 36
- international aid, charitable giving (studies) 683
- international migrants, flow (increase) 1
- interpersonal conflict, personality prediction 431
- interpersonal justice 226
- interpersonal mistreatment 232–233
- interpersonal motives 282–283
- interpersonal perception, accuracy 13
 - bias, role 14
 - psychological consequences 15–16
 - relationship/social consequences 14–15
- intervention
 - approaches, identification 329–330
 - computer-tailored interventions 365–366
 - framework, implicit processes (inclusion) 330
 - modality 197–198
 - social comparison, relationship 250–251
 - tailored interventions, efficacy (increase) 367
- in-the-moment coping strategies, EMA (impact) 143
- intimate relationships, physical health (relationship) 337
- invincibility, capacity 269
- ischemic heart disease, mortality proportion 172
- isolation, risk factor 462
- issue, uncertainty type 306
- job outcomes (prediction), dominance (impact) 433
- job performance evaluations, availability 440
- joint coping 84
- joint impact hypothesis, support 599–600
- journey, metaphor 122, 123
- justice
 - beliefs, descriptions 227
 - justice-oriented cognitions 223
 - multidimensional nature 227
 - psychosocial perception 226
- latitude, seasonal affective disorder (correlation) 171
- lesbian, gay, and bisexual (LGB) youth, substance use (increase) 234
- lesbian, gay, bisexual, or transgender (LGBT)
 - healthcare needs 635
 - identification 508
 - older adults, explicit bias 510
- life events
 - biobehavioral responses 538
 - R/S buffer 711
- life stories
 - correlates, health consequences 215
 - health consequences 219–220
 - measurement 217–218
 - representation 216
 - temporal coherence 216
- life stressors, impact 532
- lifestyle
 - behaviors, modification 397
 - changes 366
- Lifestyle Triple P intervention 653
- life, thriving 763
- likelihood estimates, illness identity/cause/timeline (connection) 99–100
- likelihood estimation (risk perception) 548–549
- likelihoods, utilities (combination) 550–551
- literacy, functional type 287–288
- liver
 - cancer (Asians) 169
 - stimulation, glucose (production) 256
- lives, complication 216
- living room language, usage 292
- locus of control, external causes 606
- locus, uncertainty type 306
- loneliness 663–664
 - feelings 461
 - impact 697
 - mindfulness-based stress reduction (MBSR) training 699
- longevity, PA effects 487–488
- longitudinal ambulatory assessments 374–375

- long-term cardiovascular activation, high blood pressure (association) 256
- long-term health goals, theory of discounting 162
- long-term self-management, action (automation) 322–323
- Los Angeles basin, air quality-control policies (establishment) 174
- loss-framed messages 355
 - emphasis 357
- lung cancer claims, tobacco company refutations 114
- main-effect model 660
- maintenance (change stage) 186, 367
- maintenance self-efficacy 608
- major depression 449
- maladaptation 269
- maladaptive behaviors, engagement 135
- mapping 121–122
- marital conflict, engagement 341
- marital dissatisfaction, psychological morbidity (association) 83
- marital quality, relationship status (connection) 337–339
- marital status, infectious diseases (association) 538
- marriage (end), sleep (lifestyle factor) 541
- massage (emotion-focused coping activity) 413
- mass customization process, application 368
- mass-media messages, impact 357
- Master Settlement Agreement (MSA), implementation 115
- mastery
 - experiences 610
 - regaining 63
- meaning making 63, 154
- meaning, worth (relationship) 683–684
- mediation analyses 509
- medical attention (seeking), rumination (negative effect) 563
- medical decision making 347
 - health literacy 290–291
- medical decisions
 - difficulty 347
 - future outcomes, discounting 349–350
 - strategic behavior 350
 - trade-offs 348–349
- Medical Interview Satisfaction Scale (MISS-29) 404
- medical jargon/abbreviations
 - avoidance 292
 - usage 405
- medical mistrust (psychosocial concept) 226
- medical resources, allocation 348
- medical treatment, type/source 299–300
- medical waiting periods, anxiety/rumination 783
- medication labels (misinterpretation), health literacy (impact) 290
- meditation (emotion-focused coping activity) 413
- memory construction, bias (reduction) 105–106
- MEMScap, usage 398
- mental associations, creation 121–122
- mental health, well-being (relationship) 72–75
- mental imagery 610
- mesolimbic reward system, subcortical areas (activity) 178
- message framing 355
 - application 359–360
 - behavior-related moderators 356–357
 - effectiveness, decrease 359
 - self-efficacy, moderator function 358
- message framing effects
 - colors, usage 359
 - mediators 359
 - moderators 356–359
- message resistance, research 589
- message tailoring 363
 - delivery modes, availability 368–369
 - function 365–366
 - explanation 366–368
 - health communication strategy 363
 - mass customization process, application 368
 - meta-analyses 365
 - process 363–365
- metaphoric framing 123
 - exposure 124
- metaphoric language, consequences 127
- metaphoric phrases, exposure 125
- metaphors
 - bodily metaphors 122
 - cognitive tool 121–122
 - conceptual metaphor theory 125
 - concrete metaphors, reliance 122
 - images/schemas 126
 - impact 127–128
 - implications 124–128
 - misleading direction 127
- method/theory, confusion 320
- microelectromechanical systems (MEMS), usage 448
- mindfulness-based stress reduction (MBSR) training 699
- mindless behaviors 162

- Minnesota Multiphasic Personality Inventory (MMPI) 442
- misclassification bias 450
- miscommunication, metaphors (impact) 127–128
- misinformation 49–50
 food/nutrition misinformation, ADA position statement 50
- mobile sensing methods 380–382
 ethical concerns 381
- moderate to vigorous physical activity (MVPA) 452
- momentary self-report assessments 375
- moment-salient behaviors 161
- mood
 disorders 256
 dysregulation, social problems (risk factors) 462
 effects 374
 improvement 160
 negative mood signals, action 164
- morbidity
 irregular/uneven people, connection 429
 PA effects 488–489
 sex differences 632
- mortality
 income inequality, impact 173
 irregular/uneven people, connection 429
 PA effects 487–488
 relationship quality, connection 339
 risk, increase 704
 sex differences 631–632
 thoughts, accessibility 744
- motivation
 absence 673–674
 beliefs, impact 735–736
 counterfactuals, relationship 78
 phase 187
- motivational focus, impact 624–625
- motivational orientation 357
 systematic differences 357–358
- motivational resource, depletion 735
- motivational self-efficacy 608
- motives contemplation 283
- movement-based volume variables 452
- multidimensional communication strategy 363–364
- Multiple Risk Factor Intervention Trial (MRFIT) 5309
- multiplex genetic susceptibility testing (MGST) 574
- multiplicity 49
- music therapy (emotion-focused coping activity) 413
- mutual dependence, network 661–662
- μ -opioid receptor gene, polymorphisms 460–461
- myocardial infarction 256
- narratives. *See* autobiographical narratives
- National Action Plan to Improve Health Literacy (HHS) 293
- National Assessment of Adult Literacy (NAAL), health literacy measurement 289
- National Center for PTSD, trauma/treatment information 258
- natural disasters, impact 171
- naturalistic observation 143–144
- natural resources, distribution 171
- negative affect (NA)
 absence 490
 PA/morbidity effects, independence 488
 role 486–487
- negative affectivity 134
- negative behaviors (increase), counteractuals (impact) 76
- negative emotions
 increase 35
 regulation 136
 role 132–133
 secretory immunoglobulin A (S-IgA), release 134
- negative feedback, receiving 22
- negative mood
 persistence 164–165
 signals, action 164
- negative relationship interactions 341
- negative social factors, consequences 461
- negative state relief, research 164–165
- net risk, yield 271
- network support, importance 83
- network ties, usage 704
- neuroendocrine activity, social functioning (association) 660–661
- neuroendocrine function
 concepts/research approaches 660–661
 social factors 659
 social influences 660
- neuroticism 412, 424–425
 associations 415
 lifespan perspective, importance 435
 tendency 434–435
- neurotics, health vigilance 435
- Newest Vital Sign 288–289
- Nicomachean Ethics* (Aristotle) 239

- nicotine, physiological addiction 159
- Nietzsche, Friedrich 440
- nocebo effects 475, 477
 - historical context 475–476
 - neurobiological mechanisms 479
 - psychological mechanisms 478–479
- nonadherence. *See* intentional nonadherence; unintentional nonadherence
 - adverse health effects 397
 - consequences 396–397
 - economic burden 396–397
 - prediction, patient-related factors 398
 - predictors 398–399
 - rates 396
 - regimen-related factors 398–399
- non-cardiovascular disease outcomes, immune
 - function (relationship) 387–389
- nonconscious bias 512
- nonconscious death thoughts (responses), esteem
 - contingencies/cultural beliefs (dependency) 743–744
- nonconscious processes 178
- nonfulfillment 395
- nonmedical waiting periods, anxiety
 - patterns 782
- nonphysical pain, physical pain (shared neural activation) 459–460
- non-rapid eye movement (NREM) 669
- non-specific immune responses, suppression 561
- nonsteroidal anti-inflammatory analgesic, impact 460–461
- non-tailored messages, tailored information (comparison) 365
- non-wear periods 451–452
- non-wear time, measurement 451
- normative data referencing 729
- normative feedback, effectiveness (reduction) 729
- normative statistical rules, following 549
- norms 521
 - descriptive norms 572–573
 - influences 726–727
 - informational norms 651
 - injunctive norms 331, 572
 - norm-based interventions, moderators 729–730
 - subjective health norms 725
 - subjective norms/attitudes 521
 - subjective norms, examination 725
 - usage 727–729
- Norr, Roy 114
- not-in-the-moment coping behavior 142
- nudges
 - nudge-style intervention, benefits (discussion) 197
 - types 351–352
- numeracy 309, 350–351
 - levels, problem 351
- Nyquist principle 448
- obesity
 - culture, impact 649–650
 - family relationships 652–653
 - modeling, impact 653–654
 - parenting 652–653
 - romantic relationships, effects 653
 - sex differences 634
 - social factors 649
 - social networks, effects 652
 - social norms 650–651
 - social support 651–652
 - socioeconomic status (SES) 650
- objective assessments, self-reports (contrast) 449–450
- objective disease-specific health indicators, assessment 151
- objective gains, perception 549–550
- objective health outcome measures 373–374
- objective reality, divergence 618
- objective self-awareness theory 597–599
- objective social isolation, measures 696
- observational learning/modeling (self-efficacy belief source) 186
- obstacle, metaphor 122
- occupational differences, production 171
- older adults, loneliness (mindfulness-based stress reduction training) 699
- openness 424, 434
- opposite-sex confederate, support 662
- optimism
 - cardiovascular impact 387
 - circulating pro-inflammatory cytokine levels, relationship 388
 - coping, relationship 415–416
 - defining 385
 - dimensionality 390
 - disappointment, vulnerability 388
 - effects 386–389
 - epidemiological studies 386–387
 - expectancy-value process models, usage 413–414
 - five-factor model 413
 - hardiness, relationship 442

- optimism (*cont'd*)
 - immune response, negative relationship 388–389
 - measurement 386
 - physical health, relationship 385
 - psychological/behavioral mechanisms 389–390
 - research 386
 - self-efficacy beliefs, contrast 605–606
- ordinary magic
 - process 269
 - support 273
- organizational injustice models 531–532
- organizational justice measurement
 - framework 226
- oscillatory systems coherence/entrainment,
 - appreciation feelings (relationship) 264
- ostracism, feelings 461
- others, transformation 682–683
- outcomes. *See* health outcomes
 - defining, considerations 430
 - evaluation, action plans 315
 - expectations/evaluations 186
 - improvement, physician-patient communication (impact) 470–471
 - outcome expectancies, reference 606
 - perception, dispositional tendencies 227
- overeating 162
- pain
 - adaptation (promotion), social/environmental factors (usage) 462–464
 - catastrophizing, rumination (examination) 562
 - experience, social factors (positive effects) 463
 - management, medication 507
 - memories 461
 - PA effect 489
 - pain-related emotional distress 459
 - pain-related learning, social threat (impact) 462
 - pain-related outcomes 562
 - pain-relevant difficulties, isolation (risk factor) 462
 - perception framework 561–562
 - treatment, pro-White bias (impact) 511
- pair-bonded monogamous adults, infant (bonding) 43
- pair bonding 43
- parasympathetic nervous system (PSNS) 659
 - activity, examination 663
 - influence, reduction 661
- parent-child communication, increase (reports) 523
- parenting practices, food (reward usage) 652–653
- Parkinson's disease
 - counterfactual thinking reduction, association 74
 - patients, motor performance 477–478
- partner QOL, couple-based interventions (effects) 84–85
- pastimes, geographical differences 172
- patient adherence 395
 - definition 395–396
 - improvement, physician communication skills (impact) 470–471
 - meta-analysis 299
 - physician-patient communication, association 399
- Patient Protection and Affordable Care Act (ACA) 224, 349
- patient-reported outcomes (PROs), usage (increase) 110
- patients
 - decisions, emotion (impact) 21–22
 - interactions, stereotyping/prejudice (influence) 510–512
 - network support, importance 83
 - oral communication 292–293
 - QOL outcomes, couple-based interventions (effects) 84–85
 - stereotyping 511
- patient satisfaction 403
 - assessment, concerns 404
 - assessment/measurement 403–404
 - correlates 405–406
 - demographic variables, relationship 406
 - literature, directions 406
 - measurement, challenges 404
 - patient demographics/health status, impacts 405–406
 - physician-patient communication skills, importance 405
 - prediction 405
 - research findings 404–405
- Patient Satisfaction Questionnaire 403–404
- Patient Satisfaction Questionnaire-18 (PSQ-18) 404
- peer pressure, impact/importance 518
- Pennebaker, James 149–150
- perceived barriers 367–368
- perceived behavioral control 756
 - term, usage 606
- perceived benefits 367–368
- perceived hopelessness 532
- perceived racism (psychosocial concept) 226

- perceived risk 521–522, 571
- perceived self-efficacy, requirement 187–188
- perceived severity 184, 367–368
- perceived social support 704
- perceived support 662
- perceived susceptibility 184
- perceivers
 - biological processes 16
 - psychological consequences 15
- perception accuracy
 - biological processes 16–17
 - downsides 17–18
 - formation, psychological health (relationship) 15–16
 - interpersonal/intrapersonal benefits 16
- performance
 - action plans 315
 - job performance evaluations, availability 440
 - near-chance levels 349
 - performance-enhancing drugs, usage (intention) 520
- periaqueductal gray, threat detection
 - involvement 698
- peripheral nervous system, parasympathetic branch 134
- peripheral route processing 366
- persistent complex bereavement disorder (PCBD) 44–45
- personal identifiers, personalization/inclusion 364
- personality
 - coping, relationship 411, 414–416
 - description 421–422
 - five-factor model 412
 - hardiness 439
 - health behaviors, relationship 421
 - health outcomes, relationship 429
 - interventions 435–436
 - measures 281
 - prediction, health (impact) 431
 - psychology 411–413
 - role 416
 - traits
 - consistency 704
 - health behaviors, association 422–425
 - trajectories 432–435
 - variables, placebo effects (relationships) 479–480
- personalized normative feedback 728
 - usage 728–729
- personal justice beliefs, descriptions 227
- personal mastery experiences 186
- personal persistence, claim 216
- person-related moderators 356, 357–358
- perspective taking 513
- persuasion
 - cognitive dissonance theory 751
 - definition 747
 - elaboration likelihood model (ELM) 748–749
 - fear appeals, role 748
 - impact 748–750
 - role 747
 - self-affirmation 749–750
 - self-perception processes 751–752
 - self-persuasion, impact 750–752
- pervasive discrimination 232
- phase-specific self-efficacy, measurement 608, 609–610
- phenylketonuria (PKU), genetic screening 569–570
- physical activity
 - accelerosensors 447–448
 - data check 451
 - data processing 451–452
 - decrease/increase, role 449
 - factors, influence 452–453
 - increase 234
 - measurement
 - days, number 451
 - issues 451–452
 - principles/technology 447–448
 - reliability/validity/reactivity 453–454
 - monitoring 447
 - national level 195
 - non-wear periods 451–452
 - outcome variables 452
 - perspectives 450
 - valid day, identification 452
- physical appearance, changes 63–64
- physical exercise (emotion-focused coping activity) 413
- physical health
 - defining 262
 - discrimination/prejudice consequences 232
 - expressive writing, relationship 151–152
 - gratitude, relationship 263–265
 - intimate relationships, connection 337
 - optimism
 - effects 386–389
 - relationship 385
 - outcomes
 - health literacy, relationship 291
 - individual attachment differences, linking mechanisms 33–37

- physical health (*cont'd*)
 - problems 704
 - relationship breakups, impact 538
 - rumination, effects (conceptual model) 560
 - social functioning, link 660
 - trauma
 - exposure, impact (indirect pathways) 256
 - indirect/direct links 258
- physical health attachment
 - associations, examination 38–39
 - avoidance, link 32–33
 - orientation, link 32–33
- physical illness, rumination (impact) 563–564
- physical pain
 - immune system, impact 460
 - intervention studies 460–461
 - nonphysical pain, shared neural activation 459–460
 - social pain, shared mechanisms 459–461
 - well-being challenge 459
- physical symptoms, perceptions 599–600
- physical well-being 617–618
 - QOL challenge 82–83
- physician-patient communication 469
 - collaboration 471
 - implications 511–512
 - outcomes, improvement 470–471
 - patient adherence, association 399
 - problems 470
 - skills
 - importance 405
 - training 471–472
- physicians
 - communication skills
 - impact 470–471
 - improvement, techniques 471
 - implicit bias 511
 - pro-White bias 511
 - visits, reduction 154
 - waiting time, correlation 300
- physiological regulation (disruption), personality (impact) 430–431
- physiological response systems 35–37
- physiological systems (activation), social-evaluative threat (impact) 644–645
- physiology, ambulatory assessment methods 375
- placebo-controlled RCTs, design 476
- placebo effects 475
 - classification 476–477
 - clinical translation 480–481
 - dependent measures 477–478
 - enhancement, patient-provider interaction (usage) 481
 - historical context 475–476
 - moderators 479–480
 - neurobiological mechanisms 479
 - non-specific effect 476
 - personality variables, relationships 479–480
 - psychological mechanisms 478–479
 - reconceptualization 476–477
 - research 477–478
- planned behavior. *See* theory of planned behavior
- Planned behavior model 185
- planning-related cognitions, frontal lobes (involvement) 74–75
- pleasure, derivation 160
- poor health, pathways (discrimination/prejudice mechanisms) 232
- positive affect (PA) (good feelings) 485
 - active ingredients, issue 486
 - broaden-and build theory 490
 - duration (measurement tool) 486
 - gratitude, relationship 262
 - impact 433
 - explanation 489–490
 - increase, impact (question) 490–491
 - longevity/mortality 487–488
 - main effect model 489
 - morbidity 488–489
 - PA-health findings, generalizability 486
 - PA-health review 487–489
 - pain, relationship 489
 - self-related health outcomes, association 489
 - stress-buffering model 489–490
 - study, methodological themes/issues 485–487
 - survival 488
- Positive and Negative Affect Schedule (PANAS) 485
- positive emotions
 - perspective 132–133
 - secretory immunoglobulin A (S-IgA), release 134
- positive information, selective access 163
- positive psychology, developments 767–768
- positive relationship interactions 341–343
- positive strategies, examples 691
- postpartum depression (PPD) 718
- posttraumatic growth (PTG) 66
- post-traumatic stress disorder (PTSD)
 - emotion regulation difficulties 35
 - expressive writing, impact 151
 - supportive, impact 273
 - symptoms, cardiovascular system activation (association) 256
 - victims, upward counterfactual generation 73

- posttraumatic stress (PTS) symptoms, EW effect 153
- “Powerful Placebo, The” (Beecher) 475
- precaution adoption process model (PAPM) 186–187, 205, 495–500
 - behavior change, difficulty 502
 - between-stage differences 503
 - description 497–498
 - inaction, stages 500
 - research usage 503–504
 - stage 1 (unaware) 498
 - stage 2 (unengaged) 499
 - stage 3 (undecided) 499
 - stage 4 (decided not to act) 499
 - stage 5 (decided to act) 499–500
 - stage 6 (acting) 499–500
 - stage 7 (maintenance) 500
 - stage-based interventions, criteria 500–502
 - stages 497
 - assessment 501
 - justification 498–500
 - progress, determination (factors) 498
 - transitions, intervention strategies 503
 - stage-targeted messages, delivery 501
 - unstaged messages, superiority 501
 - usage 502–503
- precontemplation
 - change stage 186, 367
 - stage 184
- pregnancy
 - anxiety 718
 - close relationships, importance 719
 - health behaviors 719–720
 - health disparities 718
 - mood disorders 718
 - postpartum depression (PPD) 718
 - prenatal depression 718
 - preterm birth/low birthweight, socioeconomic status (risk factor) 718–719
 - sociocultural influences 720
 - stereotyping 717–718
 - stressors, ethnic/racial disparities 719
 - stress/resilience 717
- prejudice
 - clinical recommendations, link (absence) 512–513
 - direct effects 232–233
 - effect, moderators 235–236
 - explicit prejudice 508–509
 - healthcare, relationship 507
 - health consequences 231
 - implicit prejudice 509–510
 - indirect effects 233–235
 - influence 510–512
 - likelihood 513
 - occurrence/impact, reduction 513–514
 - perception/coping, fairness beliefs (impact) 236
 - stress, coping 235
 - systemic/interpersonal mistreatment 232–233
 - terms, defining 231–235
 - preparation (change stage) 186, 367
 - preterm birth/low birthweight, socioeconomic status (risk factor) 718–719
 - prevention behavior, motivation 123–124
 - prevention-focused individual, behavior reinforcement 206
 - preventive coping 56
 - preventive health behaviors (promotion), mass-media messages (impact) 357
 - preventive services, usage (increase) 211
 - primary appraisal (stressors) 64
 - private/public education, racial/ethnic disparities 233
 - proactive coping 56, 414, 415
 - probability estimation, normative rules (disregard) 549
 - probability neglect, demonstration 551
 - problem-focused conversations 144
 - problem-focused coping 64–65
 - emotion-focused coping, contrast 413–414
 - facilitation 56
 - strategies 235
 - procedural justice
 - beliefs 227
 - concept 225
 - instrumental functions, focus 225
 - relational function 225
 - process imagery 610
 - process models 204
 - Prochaska, James 186
 - Profile of Mood States (POMS) 485
 - pro-inflammatory profiles, exhibition 17
 - prompts, provision 331
 - prospective control 738
 - classes 737
 - prostate-specific antigen (PSA) levels, health literacy (impact) 290
 - protection motivation theory (PMT) 184, 500, 571
 - protective buffering (relationship-coping strategy) 66
 - protective factors, characterization 271
 - protective processes, influence 271

- prototypes 520–521
 - definition 518
 - development 524
 - dimensions 520–521
 - healthy prototype, effects 519
 - manipulation 522–523
 - visualization 522–523
- prototypes/representations
 - multilevel process 318–320
 - structure/content, similarity 319
- prototype-willingness model (PWM) 185, 517
 - adolescents 518–519
 - adults 519
 - antecedents 520
 - components 517–518
 - dual processing, evidence 522, 523–524
 - integrative theoretical frameworks 533–534
 - interventions 523–524
 - laboratory studies 521–522
 - manipulations 520
 - prototypes, manipulation 522–523
 - research (age/experience) 518–519
 - safe sex images 522
 - socioeconomic status (SES) 532–533
 - effects, meta-analytic evidence 533
 - Strong African American Families (SAAF) Program, development 523
 - ultraviolet (UV) protection lab studies 523
- provider-patient-spouse triads, coping perspective (application) 58
- pro-White bias, impact 511
- proximity-seeking functions 43
- psychic numbing 551
- psychoactive drugs, usage/impact 671
- psychobiologic systems, demand (excess) 272
- psychological appraisal (stressors) 64
- psychological consequences 15–16
- psychological distress
 - decrease 154–155
 - increase 81
 - reduction 542
- psychological functioning 13
 - problems, report 17
- psychological health, perception accuracy (relationship) 15–16
- psychological immune system (strength), anticipation (failure) 22
- psychological justice 226–227
 - health effects, occurrence 228
- psychological morbidity, marital dissatisfaction (association) 83
- psychological threats 587
- psychological well-being 617
 - gratitude, relationship 262–263
 - increase, gratitude letter writing (impact) 265
 - QOL challenge 82–83
- psychology, ambulatory activity monitoring 454–455
- psychomotor energy expenditures, deceleration 460
- psychopathological disorders, risk 136
- psychophysical numbing 551
- psychophysiological state, chronotype (impact) 673
- psychosocial functioning, expressive writing (relationship) 153
- psychosocial risk factor, defining 529–530
- public health
 - improvement 174
 - weather, acute implications 171
- public policy, health (geographic influences) 173–174
- Puerto Ricans, asthma 170
- pulmonary diseases, adherence rates 396
- quality-adjusted life year (QALY), metric 348
- quality of life (QOL)
 - cancer patient/partner challenges 82–84
 - couple-based interventions
 - effects 84–85
 - weighted mean effect sizes/confidence intervals 85t
 - post-cancer thoughts 81
- quitting, action stage 184
- race-based discrimination 234
- race stereotyping 511
- racial disparities 233
- racial minorities
 - medical treatments 225
 - physical activity, increase 234
- Rapid Estimate of Adult Literacy in Medicine (questionnaire) 288
- rapid eye movement (REM) 669
- reaction time (cognitive processing measure) 477
- reactivity model 660
- “realistic accuracy” approach 13
- reasoned action
 - approach 756–759
 - theory 185
- reasoned pathway 517
- recall
 - assessments, EMA advantages 106–107
 - bias, problem 450

- construction, bias (reduction) 105–106
- retrospective recall (limitations), ESM methods (circumvention) 106–107
- recall-related errors 374
- received support 662–663
- receiving context, effects (understanding) 8
- recovery self-efficacy 608
- Redemption 218
- redemptive story, manifestation 218
- reflective-impulsive model (RIM), information-processing modes (types) 329
- reflective information process 329
- reflective system, burden (reduction) 330
- rehospitalization, short-term health outcome 386
- rejection, threat (impact) 619
- relatedness 206
- relational regulation theory (RRT) 704
- relationship consequences 14–15
- relationship-coping strategies 66
- relationship dissolution
 - allostatic load 541
 - cardiovascular responding 539
 - health
 - association 537
 - behaviors 540–541
 - outcomes 542–543
 - health-relevant responses 538–540
 - immune functioning 539–540
 - morbidity/mortality 537–538
 - psychological processes 542–543
- relationship-focused coping, usage 56
- relationship functioning 13
- relationship quality
 - disease recovery, connection 339
 - health behavior, connection 338
 - mortality, connection 339
 - physiological health correlates 337–338
- relationships. *See* social relationships
- gratitude, effects 263
- negative relationship interactions 341
- positive relationship interactions 341–342
- quality, personality prediction 431
- social control element 692
- status, marital quality (connection) 337–339
- relationship-threatening information 17
- relationship well-being, QOL challenge 83–84
- relative deprivation, concept 226
- relaxation (emotion-focused coping activity) 413
- religiosity/spirituality (R/S)
 - distant intercessory prayer 712–713
 - health
 - behaviors 710–711
 - research 712–713
 - health, relationship 709
 - mechanisms 710–712
 - negative R/S variables 711
 - positive R/S variables 711
 - psychological processes 711–712
 - religiousness/spirituality, impact 720
 - sense, beliefs 709–710
 - social support 710
 - topics 712–713
 - variables 711
- religiousness, impact 720
- relining policies 232–233
- repetitive thinking/thoughts 558
- rumination, relationship 563–564
- reporting bias, problem 450
- representations/prototypes, differentiation (importance) 316–317
- representations (activation), reasoned processes (involvement) 317
- reproductive health, sexuality (impact) 635
- resilience
 - construct 270
 - developmental model, influence (levels) 274–275
 - developmental process 270
 - diagnosis, information (requirement) 269
 - explanatory mechanisms 272–273
 - health implications 269, 274–275
 - influences, consideration 274
 - mechanisms 273
 - ordinary magic, support 273
 - process, influences 270–272
 - psychological mechanisms, operation 272–273
- respiratory sinus arrhythmia (RSA) index 539
- response efficacy 125
- response styles theory, extension 558
- responsibility patterns, (challenges), cancer treatment (impact) 83–84
- rest and digest calming response 134
- retrospective recall (limitations), ESM methods (circumvention) 106–107
- retrospective self-reports, concerns 375
- risk
 - acceptance 552
 - aversion 356
 - cognitions, social comparison (moderator function) 250
 - disease-related variables, impact 99
 - estimate, conveyance 307
 - evaluation, understanding 547, 552
 - factors, effects 270–271
 - inequality, perception 553
 - magnitude 552

- risk (*cont'd*)
 - net risk, yield 271
 - opportunity, openness 517–518
 - perceived risk 521–522
 - risk-taking behavior, evidence 636
 - uncertainty, relationship 347–348
- risk perception 547
 - affect heuristic 552–553
 - expected subjective utility 549–550
 - likelihood estimation 548–549
 - likelihoods, utilities (combination) 550–551
 - understanding 547
 - victims, numbers (perception) 551
- risky behaviors
 - peer pressure, impact/importance 518
 - sex differences 636
 - social comparison, relationship 250
- risky health behavior, usage 600
- rumination 557, 783
 - autonomic nervous system, physiological parameter 560
 - cardiovascular activity, physiological parameter 560
 - cortisol, relationship 561
 - effects, conceptual model 560
 - goals, role (focus) 448–449
 - health
 - behaviors 562–563
 - relationship 559
 - impact 35, 542
 - models/definitions 557–559
 - negative effect 563
 - neuroendocrine/immune activity 560–561
 - non-stressor-related ruminative thoughts 558
 - physical illness/disease, relationship 563–564
 - physiological parameters 560–561
 - prevention/intervention 564
 - reduction 564
 - repetitive thinking 558
 - sleep, relationship 562
 - social-evaluative threat (SET), link 644
 - somatic complaints 561–562
 - stress-reactive rumination, role 558
 - stress-related rumination 560
 - thoughts, integration 561–562
- sadness, increase 35
- safe sex images 522
- salivary cortisol (physiological measure) 477
- Sandman, Peter 186
- schizophrenia 449
 - counterfactual thinking reduction, relationship 74–75
- Schwarzer, Ralf 187
- screening
 - attitudes 571
 - barriers 571–572
 - behavior 569
 - injunctive/descriptive norms, relationship 727
 - discrimination 574–575
 - health psychology, relationship 570
 - intraindividual factors, risk/attitudes (perception) 570–571
 - issues 576
 - physician recommendation 575–576
 - racial/ethnic disparities 575
 - social factors, representation 572–573
 - social networks, role 573
 - social norms, relationship 572–573
 - social stigma 574–575
 - spousal influence 573
- screening varieties/targets 569–570
- seasonal affective disorder (SAD)
 - depressive/bipolar episodes 171
 - latitude, relationship 171
- secondary appraisal (stressors) 64
- second-generation immigrants, heritage (reconciliation/integration) 6
- secretory immunoglobulin A (S-IgA), release (positive/negative emotion link) 134
- seeking. *See* treatment seeking/avoidance
- positivity 300–301
- segregation, impact 232–233
- selective exposure 579–580
 - accuracy motivation 582–583
 - amplification 581
 - barrier, laziness (impact) 582
 - cognitive economy, impact 582
 - confidence mechanism 581–582
 - de facto selective exposure 580
 - health, relationship 580
 - mechanisms/moderators 580–583
- self
 - distributive/procedural justice 227
 - others, transformation 682–683
 - private/public aspects, focus 598
 - sense, reliance 616
- self-affirmation 283, 749–750
 - consequence, focus 588
 - definition 588
 - developments/issues 591, 592
 - health
 - information, link 589–590
 - relationship 587, 589

- individual differences 591–592
- manipulations/interventions, effects
 - (detection) 590
- research 589
- stress, link 590–591
- theory
 - perspective 587
 - usage 588
- well-being, link 590–591
- self-awareness
 - appearance concerns 601
 - core hypotheses, support 598
 - eating disorders 601
 - effect 599–600
 - elevation 599
 - escape
 - alcohol consumption, usage 600
 - risky health behavior, usage 600
 - goal-directed health behavior 601–602
 - health, relationship 597
 - level (elevation), development (problems) 601
 - objective self-awareness theory, self-regulation
 - (link) 597–599
 - physical symptoms, perceptions 599–600
 - research 599–602
 - self-regulation, usage 601–602
 - term, usage 598–599
- self-concept
 - changes 63–64
 - perception 606
- self-control
 - capacity, reduction 179
 - challenges 738
 - cognitive factors 161–162
 - depletion 163
 - dual-process theory 161
 - failure
 - domains 159
 - emotional factor 164
 - improvement 735
 - knowledge perspective 734
 - motion 160
 - objectives 161
 - problem 669
 - strategies 737
 - success 733, 738
 - theories 160–161
- self-determination theory (SDT) 196
 - application 206
- self-distraction (avoidant strategy) 413
- self-efficacy 184, 736
 - beliefs
 - concepts, distinction 605–606
 - influence (sources) 186
 - optimism, contrast 605–606
 - concept, addition 211–212
 - defining 185–186
 - domain-specific self-efficacy, measurement 608
 - enhancement 196
 - generality dimension 607
 - general self-efficacy 607–609
 - health, relationship 605
 - increase 196
 - metaphor, usage 126
 - methods 610–611
 - level dimension 607
 - measurement 607–609
 - measures, dimensionality 607–608
 - message frame moderator 358
 - perceived self-efficacy, requirement
 - 187–188
 - phase-specific self-efficacy, measurement 608, 609–610
 - scales, criticism 608
 - self-manifesting mechanism 606–607
 - sources 610
 - measurement 608–609
 - strength dimension 607
 - term, usage 606
 - usage 608–610
- self-esteem
 - bolstering, distal motivational goals 742
 - crystallization 616–617
 - defining 615–617
 - health, relationship 615
 - process 617–618
 - reasons 618–619
 - increase, downward comparisons 63–64
 - negative information, disclosure 17
 - resource model 618–619
 - restoration 63–64
 - temperament, association 616
- self-focus, term (usage) 598–599
- self-healing personalities, health
 - trajectory 430
- self-imposed punishment, demonstration 737
- self-improvement motivation, upward
 - counterfactual (relationship) 71
- self-integrity, protection 587
- self-management, health condition
 - requirement 351
- self-monitoring
 - behaviors, impact 187
 - promoting, prompts (impact) 331
- self-perception processes, usage 751–752
- self-persuasion, impact 750–752

- self-regulation 330, 333–334, 623
 - automatic strategies 626–627
 - capacity 269
 - cognitive strategies 627
 - conceptualization 623
 - difficulty level, differences 625
 - goal setting 624–626
 - importance 623
 - impulses, effortful inhibition 628
 - loneliness, impact 697
 - objective self-awareness theory, link 597–599
 - planning strategies 626
 - real-life context, complexity 623–624
 - usage 601–602
- self-reinforcing system, failure 205
- self-reliance 34
- self-reported goals/norms, change (impact) 331
- self-reported health outcomes, PA
 - (association) 489
- self-reported outcomes 150–151
- self-reported physical symptoms, decrease 135
- self-reports
 - instruments, differences (impact) 450
 - methods, observational methods
 - (combination) 376
 - momentary self-report assessments 375
 - objective assessments 449–450
 - reliance, limitations 486
 - scales, impact 375
 - usage, questions 450
 - value 373–374
- self-serving intent 50
- self-standards, meeting (difficulty) 601
- self-verification, experiences (enhancement) 16
- self-view, challenge 282
- separation (psychosocial effects), immune system
 - (link) 540
- serotonin transporter gene (SERT), genetic
 - polymorphism 539
- serotonin transporter genotypes, impact
 - 271–272
- Set of Brief Screening Questions (SBSQ) 288
- severity judgments, CSM framework factors 100
- sex, attitudes (gender differences) 635
- sex differences
 - alcohol consumption 634
 - biology 632–633
 - exercise 634
 - gender-related traits 636
 - health
 - behaviors 633–634
 - relationship 631
 - morbidity 632
 - mortality 631–632
 - nurturant roles 637
 - obesity 634
 - risky behavior 636
 - smoking 633–634
 - sociocultural factors 635–637
 - sexuality, reproductive health (relationship) 635
 - sexually transmitted infections (STIs),
 - contracting 34
 - sexual risk behavior reduction (HIV prevention
 - interventions), CDC website list 1907
 - shared decision making 310–311
 - shopping (avoidant strategy) 413
 - Short Assessment of Health Literacy 288
 - Short Assessment of Health, Newest Vital
 - Sign 288
 - short-term thinking (bias production),
 - threatening emotional states (impact) 164
 - sibling coping 68
 - sickle cell disease (African Americans) 169
 - signaling fatigue 460
 - similarity, perceptions 682
 - Single-Item Literacy Screener 288–289
 - situational justice (state justice) 226
 - situation selection, pathway 431
 - skin barrier recovery 36
 - sleep
 - disorders, adherence rates 396
 - disruption, effects 673–674
 - distractions, reduction 672
 - disturbances, impact 541
 - gratitude, link (importance) 264
 - hygiene 671
 - initiation, problems 670–671
 - interference, psychoactive drugs (usage/
 - impact) 671
 - lifestyle factor 541
 - loss, cognitive/emotional effects 674
 - maintenance, problems 670–671
 - optimal sleep timing (chronotype), impact
 - 672–673
 - propensity, chronotype bias 672–673
 - quantity, deviation 669
 - rumination, relationship 562
 - sleep-relevant behavior, social ties (impact) 671
 - sleep-wake cycle
 - social behavior, mutual dependence 670
 - social interactions, disruptor role 670–671
 - social behavior, impact 670–672
 - social factors 669
 - texting 671

- Sloan Center on Everyday Lives of Families (CELFL) study 376
- smartphones
 - sensing applications, proof of concept (establishment) 381–382
 - sensors, usage 380
- smoker prototypes 524
- smoking
 - addressing, public health professional perspective 117
 - behavior
 - decrease, cigarette taxes (doubling) 116
 - problem 36
 - cessation 161, 213, 524
 - rates 159
 - continuation 587–588
 - denormalization, marketing bans (impact) 115–116
 - long-term negative consequences, warnings 689
 - prevention 524
 - rates (women/men), sex differences 633
 - sex differences 633–634
 - usage 34
 - youth-smoking prevalence, decrease 116
- social acceptance, maintenance 641
- social behavior
 - impact 670–672
 - sleep
 - disruption, effects 673–674
 - impact 672–673
 - sleep-wake cycle, mutual dependence 670
 - time cue 670
- social clock, biological clock (alignment) 673
- social cognitive theories 159–160, 185–186
 - health behavior change applications 196
- social cognitive theory (SCT) 571
- social comparison
 - downward/upward social comparisons, addition (impact) 251
- functions 249
- health correlates/consequences 247
- illness/disease, relationship 248–249
- intervention, relationship 250–251
- moderator function 250
- orientation, moderator function 250, 251
- processes, operation 249
- risky behaviors, relationship 250
- role 247–249
- theory
 - contributions 247–248
 - evaluation 250–251
- social connection 682
- social consequences 14–15
- social context, role (emphasis) 206
- social control 689
 - attempts 690–691
 - dual effects 689–690
 - effects, doubt 690
 - health-related social control 691
 - hypothesis 240
- social engineering 224
- Social Environment Coding of Sound Inventory (SECSI) 378
- social-evaluative stressors 642
- social-evaluative threat (SET) 641
 - components 642–643
 - cortisol reactivity 641–642
 - emotional/cognitive effects 643–644
 - experience, continuation 644
 - impact 644–645
 - manipulation 642
 - non-SET conditions 642–643
 - PRES conditions 642–643
 - remote occurrence 643
 - rumination, link 644
 - threat, presence 643
- social exchange, individual reaction 223
- social exclusion, depression (relationship) 460
- social experiences
 - creation 616
 - optimal sleep timing (chronotype), impact 672–673
- social facilitation, trigger 651
- social factors
 - positive effects 463
 - screening, relationship 572–573
- social functioning 13
 - frontal lobes, involvement 74–75
 - neural/psychological mechanisms 664–665
 - neuroendocrine activity, association 660–661
 - physical health, link 660
- social health equation 373
- social identity 679
 - approach
 - benefits 681
 - schematic representation 686
 - control/efficacy/power 684
 - group behavior basis 679–680
 - Groups 4 Health (G4H)
 - intervention 684–685
 - modules 685
 - health basis 680–681

- social identity (*cont'd*)
 - health-enhancing resource 681–684
 - importance 680
 - meaning, worth (relationship) 683–684
- social influence 689
 - health-related social influence 691–692
- social integration, focus 664
- social intelligence (SI) intervention,
 - development 464
- social interactions
 - disruptor role 670–671
 - naturalistic observation 373, 376
 - quality, neural mechanisms (link) 664–665
 - research, EAR methodology (usage) 379–380
- social isolation
 - behavioral mechanism 697
 - biological mechanism 698
 - group interventions 698
 - health, relationship 695
 - impact 696
 - interventions 698–699
 - measurement 695–696
 - mechanisms 696–698
 - objective social isolation, measures 696
 - one-on-one interventions 698
 - psychological mechanism 697
 - subjective social isolation, measures 696
- social justice
 - characteristics 223–224
 - distributive justice, relationship 224–226
 - framework, health inequity (connections) 225
 - health consequences 223, 228–229
 - health correlates 223
 - psychological justice 226–227
- social negativity 663–664
- social networks
 - access/inclusion 273
 - interactions, quality 464
 - screening, association 573
 - structural aspects 661
- social neuroendocrinology 659–660
- social norms
 - classes, operation 572
 - conformity 671
 - diet 650–651
 - impact 331
 - marketing approach 728
 - misperceptions 727–728
 - obesity 650–651
 - screening, relationship 572–573
- social pain
 - evolutionary value 460
 - immune system, impact 460
 - intervention studies 460–461
 - physical pain, shared mechanisms 459–461
 - well-being challenges 459
- social persuasion 186, 610
- social pressure
 - priming 331
 - sensitivity 729–730
- social problems, risk factors 462
- social prototypes 185
- social reaction path 517
- social relationships
 - gratitude, association 263
 - impact 651–652
 - importance 13
 - consideration 433
 - negative aspects 663–664
 - positive aspects 661–663
- social stigma, screening (relationship) 574–575
- social stressors, effect 461
- social support 44, 186
 - attachment orientations, link 33
 - availability 704
 - effectiveness 682–683
 - effects 463–464
 - effect size 240
 - environmental factor 532
 - health, relationship 703
 - impact 235
 - measurement 703–704
 - negative outcomes 704–705
 - network ties, usage 704
 - perceived social support 704
 - personality prediction 431
 - person interactions 706
 - resources 132
 - social relationships, positive aspects 661–663
- social threat, impact 462
- social ties-health link, psychological
 - mediators 697
- social well-being 618
 - QOL challenge 83–84
- socioeconomic status (SES) 532–533, 650, 765
 - pregnancy risk factor 718–719
- socio-emotional communication 405
- somatic complaints 257
 - rumination, impact 561–562
- somatic states, perception 611
- source, uncertainty type 306
- spillover effects 376
- spirituality
 - impact 720
 - presence 713
- spirituality/religiosity. *See* religiosity/spirituality

- stage-based interventions, criteria 500–502
- stage-based models 183–184
- stage models 186–188, 204–205
 - effectiveness, comparison 188
- stage-targeted messages, delivery 501
- stage theories
 - elements/assumptions 496
 - impact, process 495–496
- standard behavioral treatment (SBT),
 - usage 242
- startle eyeblink reflex (physiological
 - measure) 477
- state justice 226
- stereotyping
 - clinical recommendations, link (absence) 512–513
 - counterstereotyping 513
 - explicit stereotyping 508–509
 - healthcare, relationship 507
 - implicit stereotyping 509–510
 - influence 510–512
 - likelihood 513
 - occurrence/impact, reduction 513–514
- stimulus attention 558
- stimulus control 186
 - usage 184
- stimulus-response associations 177
- stop signal tasks, bias training usage 332
- strangers, treatment 682
- strategic automaticity 333
- strategic behavior 350
- stress
 - adjustment, biological mechanisms 272
 - alleviation 160
 - cardiovascular reactivity 256
 - coping, physical activity (increase) 234
 - emotional factor 164
 - experience 131
 - adaptive/maladaptive handling 141–142
 - geographic variation 172
 - immune response 540
 - impact 256
 - increase 35
 - indirect effects 233–234
 - management, social support resources (decrease) 532–533
 - markers, role 670–671
 - occurrence/persistence 134
 - process 134
 - reactivity 134
 - recovery, rumination research (association) 559
 - regulation 136
 - response
 - increase 36
 - system, activation 134
 - self-affirmation, link 590–591
 - stress-buffering hypothesis 240
 - stress-buffering models 240, 289–290, 665
 - stress-reactive rumination 558
 - role 558
 - stress-related rumination 560
 - stress-responsive HPA axis, discrimination
 - activation 233–234
 - transactional model 309–310
 - vulnerability 160
- stress and coping paradigm 64–65
- stressors
 - categorization 705
 - cortisol, increase 642
 - cultural stress, separation 4
 - effects 172
 - geographic differences 172
 - primary appraisal 64
 - psychological appraisals 64
 - psychological/physiological responses (reduction), self-affirmation (impact) 590–591
 - recovery 618
 - resolution 57
 - secondary appraisal 64
 - social-evaluative context, manipulation 642
- Strong African American Families (SAAF)
 - Program, development 523
- Stroop interference (cognitive processing
 - measure) 477
- Stroop probe, paradigm (modification) 331–332
- structural social support, longitudinal studies 532
- subjective attitudes 521
- subjective health norms 725
- subjective norms 521, 756
 - examination 725
- subjective social isolation, measures 696
- subjective utility, expectation 549–550
- substance use 195
 - lesbian, gay, and bisexual (LGB) youth (increase) 234
 - measurement 8–9
 - outcomes, norms (associations) 726
 - retreat (avoidant strategy) 413
- substance use, acculturation
 - mechanisms, understanding 4–6
 - relationship 1, 2–3
- subtractive counterfactuals 72
- success, defining 764
- suicide (American Indians) 170

- suicide levels, elevation 172
- sun protection (improvement), photographs (usage) 729
- super-adherers, adherence level 322
- support groups, impact 242
- supportive social connection, impact 273
- survival, PA effects 488
- survivors, spirituality (presence) 713
- sympathetic-adrenal-medullary axis, activation 228
- sympathetic nervous system (SNS) 659
 - activity, indexing 660
 - increase 661
- symptom report, PA effect 489
- systemic inequality 232
- systemic mistreatment 232–233
- systemic transactional model (STM) 67
- systolic/diastolic blood pressure (increase), pessimists (relationship) 387
- tailored information, non-tailored messages (contrast) 365
- tailored interventions
 - efficacy, increase 367
 - sophistication, increase 368
- tailored messaging 364
- tailoring. *See* message tailoring
- Tajfel, Henri 680
- targeted messaging 364
- targets
 - biological processes 16–17
 - psychological consequences 15–16
- task, prior success (self-efficacy belief source) 186
- Tay-Sachs disease (Ashkenazi Jews) 170
- teach-back method 291–292, 471
- teach-to-goal method 291–292
- technological innovations, impact 207
- temperament, self-esteem (association) 616
- temporal coherence 216
- temporal inconsistency 49
- temptations 163, 733
 - availability, regulation 737
 - cognitive habits 736–737
 - construal, role 738–739
 - deleterious effects 734–735
 - implementation intentions 736–737
 - importance 739
 - overcoming 735–739
 - motivation 735–736
 - positive/negative incentives 734
 - prospective control 737–738
 - strategies, classes 737
 - resistance, motivation 735
 - responses, proactive regulation 737
 - subjectivity 733–734
- termination (change stage) 367
- terror management health model (TMHM) 741
 - distal TMHM responses 743
 - implications 744
 - interventional potential 743–744
 - origins 741–742
 - overview 742
 - proximal TMHM responses 742–743
- terror management theory (TMT), development 741
- Test of Functional Health Literacy (TOFHLA) 288
- theory of planned behavior (TPB) 195–196, 755
 - model, description 756
- theory of reason action and planned behavior (TRA/TPB) 368
- theory of reasoned action (TRA) 571, 755
 - model, description 755–756
- threat
 - appraisal 184
 - control, treatments/procedures 315
 - presence 643
 - representation 315
 - rest and digest calming response 134
 - social-evaluative threat (SET) 641
- threatening emotional states, impact 164
- thriving
 - behavioral factors 765
 - cultivation 767–768
 - cultural factors 767
 - daily activities 766
 - definition 763–765
 - environmental factors 767
 - individual factors 765
 - predictors 765–767
 - social factors 766
 - socioeconomic factors 766–767
 - success, involvement 764–765
- thriving in life 763, 765
- time-based amount variables 452
- time-based EMA sampling approaches, usage 109
- tobacco
 - cartoon characters, advertising usage 115
 - fight, education 116–117
 - marketing regulation 115–116
 - regulation
 - federal mandate 116
 - initiation 114–115

- tobacco use
 - proliferation, contributor 114
 - role, growth (history) 113–114
- training impulse/reflection 330, 331–333
- trait hostility
 - anger, association 530
 - research 530–531
- traits
 - gender-related traits 636
 - trait gratitude, increase 264–265
 - trait justice 226–227
- trans-epidermal water loss, wound healing
 - indicator 36
- transformational processing 219
- transtheoretical model (TTM) 184, 186, 187, 205, 571
 - usage 367
- trauma
 - categories 255
 - exposure types 255
 - health problems (prevention), social support/interactions (usage) 258–259
 - impact 256
 - physical health, indirect/direct links 258
 - survivors, counterfactuals (generation) 73
 - therapeutic intervention 258
 - trauma-related memories 155
- traumatic events
 - exposure 461
 - health effects 255
- traumatic experiences, health problems
 - (association) 257
- traumatic stressor, experience 256
- treatment
 - avoidance, defining 298
 - regimen, nonadherence (adverse health effects) 397
- treatment seeking/avoidance 297
 - demographic factors 298
 - factors 298–300
 - hard factors 298
 - healthcare context 300
 - health problem, type/intensity 299
 - health treatment/maintenance 302
 - impact 300
 - incentive programs 301
 - medical treatment, type/source 299–300
 - personality, link 299
 - psychosocial factors 298–299
 - soft factors 298
 - wearable technology, usage 301
- treatment seeking, defining 297–298
- triculturalism, complexities 7
- triggering cue, habitual response (association) 179179
- Turner, John 680
- Type A behavior (TAB) pattern, research 429, 530–531
- ultraviolet (UV) protection lab studies 523
- uncertainty. *See* health
 - behavioral outcomes 307
 - cognitive closure, requirement 308
 - coping 309–310
 - defining 305–306
 - intolerance 308
 - management 309–311
 - mediational analyses 307
 - physical outcomes 307–308
 - preference-sensitive decisions 311
 - psychological outcomes 306–307
 - responses 306–309
 - individual differences 308–309
 - risk, relationship 347–348
 - shared decision making 310–311
 - types 306
- unconscious habits 162
- uncontrollable stressors 642, 705
- Unequal Treatment: Confronting Racial and Ethnic Health Disparities in Health Care* (IOM) 223, 226
- unintentional nonadherence 396
- unplanned pregnancy, risk (increase) 34
- unrealistic absolute optimism 774
 - health costs 777
- unrealistic comparative optimism 774
 - health costs 777–778
- unrealistic optimism
 - causes 774
 - consequences 776
 - defining 773–774
 - display, reasons 774–775
 - health
 - benefits 776
 - costs 776–778
 - relationship 773
 - types 774
- unstaged messages, superiority 501
- upward comparison, role (evaluation) 249
- upward counterfactuals 71
 - extreme/excessive responses 72
 - focus 75
 - impact 77
 - PTSD victim generation 73

- upward identification 248
- upward social comparison, addition (impact) 251
- US Preventive Services Task Force (USPSTF),
 - clinical preventive services
 - recommendations 570
- utilities, likelihoods (combination) 550–551
- vaccination, decisions 21
- Valentine's Day, connotations 25
- verbal persuasion 610
- vicarious experience 186, 610
- victims, numbers (perception) 551
- violent trauma, experience 258
- visual dot probe (VDP) paradigm, modification
 - 331–332
- volitional phase, standards awareness 187
- volitional self-efficacy 608
- volition phase 187
- volume, term (indication) 448
- vulnerability 269
 - factors, characterization 271
 - processes, impact 271
 - social contributors 461–462
- waiting experiences
 - differences 783–784
 - effects 784
- waiting periods
 - changes 782
 - types, variability 783
- walkable green spaces, creation (impact)
 - 173–174
- warm touch intimacy-building intervention 342
- weight-based discrimination 234
- weight loss, health literature focus 194
- Weinstein, Neil 186
- well-being
 - challenges 459
 - emotion regulation prediction 133–136
 - estimates, reduction 23
 - expressive writing, relationship 153
 - mental health, relationship 72–75
 - PA, impact (explanation) 489–490
 - self-affirmation, link 590–591
- Western Collaborative Group Study (WCGS) 530
- “wet foot, dry foot” law 8
- white blood cells, binding ability 540
- Wicklund, Robert 597
- widowhood effect 45
- willingness
 - assessment 518
 - development 524
 - increase 522
 - intention
 - comparison 520
 - relationship 521
- winter SAD, latitude (correlation) 171
- wishful thinking (avoidant strategy) 413
- within-group diversity 8
- women
 - mortality, sex difference 631–632
 - nocturnal blood pressure, decrease 241
 - pregnancy, life experiences 717
- word generation (cognitive processing
 - measure) 477
- working memory (WM) training 331, 333
- work stress (environmental factor) 531–532
- World Happiness Report 684
- worldview, challenge 282
- worldview verification theory 227
- worry
 - consequences 101–102
 - defining 97–98
 - degree, determination 99–101
 - disease-related variables, impact 99
 - emotional worry, transfer 123
 - increase, illness symptoms (impact) 100
 - reduction 564
- worth, meaning (relationship) 683–684
- wound healing
 - indications, trans-epidermal water loss
 - (impact) 36
 - speed, reduction 36
- wound infection 386
- written diaries, usage 106
- X-linked chromosomal abnormalities, female
 - resistance 632
- youth-smoking prevalence, decrease 116

The Wiley Encyclopedia of Health Psychology

Volume 3
Clinical Health Psychology
and Behavioral Medicine

Edited by

C. Steven Richards
Lee M. Cohen

Editor-in-Chief

Lee M. Cohen

WILEY Blackwell

This edition first published 2021
© 2021 John Wiley & Sons Ltd

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by law. Advice on how to obtain permission to reuse material from this title is available at <http://www.wiley.com/go/permissions>.

The right of Lee M. Cohen to be identified as the author of the editorial material in this work has been asserted in accordance with law.

Registered Offices

John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Office

111 River Street, Hoboken, NJ 07030, USA

For details of our global editorial offices, customer services, and more information about Wiley products visit us at www.wiley.com.

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Limit of Liability/Disclaimer of Warranty

While the publisher and authors have used their best efforts in preparing this work, they make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives, written sales materials or promotional statements for this work. The fact that an organization, website, or product is referred to in this work as a citation and/or potential source of further information does not mean that the publisher and authors endorse the information or services the organization, website, or product may provide or recommendations it may make. This work is sold with the understanding that the publisher is not engaged in rendering professional services. The advice and strategies contained herein may not be suitable for your situation. You should consult with a specialist where appropriate. Further, readers should be aware that websites listed in this work may have changed or disappeared between when this work was written and when it is read. Neither the publisher nor authors shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

Library of Congress Cataloging-in-Publication Data

Names: Paul, Robert D., editor.

Title: The Wiley encyclopedia of health psychology / edited by Robert Paul,
St. Louis MO, USA ; editor-in-chief, Lee Cohen, The University of
Mississippi, MS, USA.

Description: First edition. | Hoboken : Wiley-Blackwell, 2021. | Includes
index. | Contents: volume. 1. Biological bases of health behavior.

Identifiers: LCCN 2020005459 (print) | LCCN 2020005460 (ebook) | ISBN
9781119057833 (v. 1 ; cloth) | ISBN 9781119057833 (v. 2 ; cloth) | ISBN
9781119057833 (v. 3 ; cloth) | ISBN 9781119057833 (v. 4 ; cloth) | ISBN
9781119675785 (adobe pdf) | ISBN 9781119675761 (epub)

Subjects: LCSH: Clinical health psychology—Encyclopedias.

Classification: LCC R726.7 .W554 2021 (print) | LCC R726.7 (ebook) | DDC
616.001/903—dc23

LC record available at <https://lcn.loc.gov/2020005459>

LC ebook record available at <https://lcn.loc.gov/2020005460>

Cover image and design: Wiley

Set in 10/12.5pt ITC Galliard by SPi Global, Pondicherry, India

Contents

List of Contributors	vii
Foreword	xxv
Preface	xxvii
Preface Volume 3: Clinical Health Psychology and Behavioral Medicine	xxix
Acknowledgments	xxx
Editor-in-Chief Acknowledgments	xxxiii
Body Image <i>Tyler B. Mason, Kathryn E. Smith, Jason M. Lavender, and Stephen A. Wonderlich</i>	1
Caregivers and Clinical Health Psychology <i>Allison Marziliano and Anne Moyer</i>	9
Close Relationships <i>Kieran T. Sullivan</i>	19
Infertility, Miscarriage, and Neonatal Loss <i>Amy Wenzel</i>	27
Prevention and Health Across the Lifespan <i>John L. Romano</i>	35
Self-Affirmation and Responses to Health Messages: An Example of a Research Advance in Health Psychology <i>Allison M. Sweeney and Anne Moyer</i>	45
Stigma of Disease and Its Impact on Health <i>Lindsay Sheehan and Patrick Corrigan</i>	57

Alcohol Use Disorder: Overview <i>Brittany E. Blanchard, Angela K. Stevens, Molin Shi, and Andrew K. Littlefield</i>	67
Cancer and Psycho-Oncology <i>Lauren N. Harris and Annette L. Stanton</i>	73
Ischemic Heart Disease, Depression, and Tobacco Smoking: Implications for Health Psychology <i>Allison J. Carroll</i>	83
Neurocognitive Disorders and Health Psychology <i>Claudia Drossel</i>	95
Depression and Comorbidity in Health Contexts <i>C. Steven Richards and Lee M. Cohen</i>	101
Depression and Relapse in Health Contexts <i>C. Steven Richards and Lee M. Cohen</i>	107
Eating Disorders <i>Eliot Dennard</i>	117
Headaches <i>Amy Wachholtz, Adam Harris, and Amy Frers</i>	131
Multiple Sclerosis, Walking, and Depression: Implications for Health Psychology <i>Ipek Ensari and Robert W. Motl</i>	143
The Impact of Exposure to Potentially Traumatic Events on Physical Health <i>Candice Presseau, Danielle S. Berke, Julie D. Yeterian, and Brett Litz</i>	153
Schizophrenia <i>Thanh Le, Kyle R. Mitchell, Elana Schwartz, Jessica McGovern, and Alex S. Cohen</i>	161
Obesity <i>Michael G. Perri, Melissa H. Laitner, and Apiva H. Ariel</i>	171
Pain and Health Psychology <i>Robert J. Gatchel and Don McGeary</i>	183
Perinatal Depression <i>Rachel Vanderkruik, Elizabeth Lemon, Laura River, and Sona Dimidjian</i>	193
Suicide in the Context of Health Psychology <i>Maryke Van Zyl-Harrison, Tanya Hunt, Rebekah Jazdzewski, Yimi Omofuma, Paola Mendoza-Rivera, and Bruce Bongar</i>	203
Adherence to Behavioral and Medical Regimens <i>Tricia A. Miller, Summer L. Williams, and M. Robin DiMatteo</i>	217

Tobacco Use Disorder and Its Treatment <i>Victoria A. Torres, Lee M. Cohen, and Suzy Bird Gulliver</i>	223
Psychological Assessment in Medical Settings: Overview and Practice Implications <i>Kelsey A. Maloney and Adam T. Schmidt</i>	235
Complementary, Alternative, and Integrative Interventions in Health Psychology <i>Jeffrey E. Barnett</i>	245
Hypnosis and Health Psychology <i>Steven Jay Lynn, Craig P. Pollizi, Joseph P. Green, Damla E. Aksen, Ashwin Gautam, and James Evans</i>	257
Motivational Interviewing <i>Thad R. Leffingwell</i>	265
Rehabilitation Psychology <i>Monica F. Kurylo and Kathleen S. Brown</i>	273
Administrative Issues in Primary Care Psychology <i>Esther N. Schwartz and David R. M. Trotter</i>	283
Affective Forecasting in Health Psychology <i>Shannon M. Martin, James I. Gerhart, Catherine Rochefort, Laura Perry, and Michael Hoerger</i>	293
Aging <i>Ann Rosenthal, Dara Mickschl, Edith Burns, Elizabeth Neumann, Jay Urbain, Sergey Tarima, and Steven Grindel</i>	301
Trends in Clinical Health Psychology and Behavioral Medicine <i>C. Steven Richards and Lee M. Cohen</i>	311
Biopsychosocial Practice in Health Psychology <i>Timothy P. Melchert</i>	321
Child and Family Health <i>Joaquin Borrego, Jr., Tabitha Fleming, Elizabeth Ortiz-Gonzalez, and Amber Morrow</i>	327
Childhood Cancer <i>Dianna Boone, Savannah Davidson, Jordan Degelia, and Jason Van Allen</i>	335
Ethical Issues for Psychologists in Medical Settings <i>Gerald P. Koocher and Jeanne S. Hoffman</i>	345
History of Clinical Health Psychology <i>Rachel Postupack and Ronald H. Rozensky</i>	355

Older Adults and Perspectives for Researchers and Clinicians Working in Health Psychology and Behavioral Medicine <i>Louise Sharpe</i>	365
Pediatric Psychology <i>Dianna Boone, Thomas J. Parkman, and Jason Van Allen</i>	373
Reproductive Health <i>Jennifer L. Brown and Nicole K. Gause</i>	383
Schools, Eating, and Health Psychology <i>Catherine Cook-Cottone and Rebecca K. Vujanovic</i>	395
Sport and Exercise in Health Psychology <i>Erika D. Van Dyke and Judy L. Van Raalte</i>	407
Sexual Minority Populations and Health <i>Mike C. Parent and Teresa Gobble</i>	417
Ethics Issues in the Integration of Complementary, Alternative, and Integrative Health Techniques into Psychological Treatment <i>Jeffrey E. Barnett</i>	427
Alcohol Use Disorder: Long-Term Consequences <i>Angela K. Stevens, Brittany E. Blanchard, Molin Shi, and Andrew K. Littlefield</i>	437
Body Image Assessment <i>Kathryn E. Smith, Tyler B. Mason, Jason M. Lavender, and Stephen A. Wonderlich</i>	445
Anxiety and Skin Disease <i>Laura J. Dixon and Sara M. Witcraft</i>	451
Insomnia <i>Todd A. Smitherman and Daniel G. Rogers</i>	459
Index	469

List of Contributors

- Amanda M. Acevedo**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Zeba Ahmad**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- Leona S. Aiken**, Department of Psychology, Arizona State University, Tempe, AZ, USA
- Olusola Ajilore**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Damla E. Aksen**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Kristi Alexander**, Department of Psychology, University of Central Florida, Orlando, FL, USA
- Andrea G. Alioto**, Palo Alto University, Palo Alto, CA, USA
Department of Neurology, University of California, Davis, CA, USA
- Michael L. Alosco**, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA
- Christina M. Amaro**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Judith Andersen**, Department of Psychology, University of Toronto, Mississauga, ON, Canada
- Aviva H. Ariel**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Danielle Arigo**, Psychology Department, The University of Scranton, Scranton, PA, USA
- Jamie Arndt**, Psychology Department, University of Missouri System, Columbia, MO, USA
- Robert C. Arnold**, Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
- Melissa V. Auerbach**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Lisa Auster-Gussman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Aya Avishai**, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Hoda Badr**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- David E. Balk**, Department of Health and Nutrition Sciences, Brooklyn College of the City University of New York, Brooklyn, NY, USA

- Kirk Ballew**, Department of Communication, Fontbonne University, St. Louis, MO, USA
Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
Department of Communication, University of Illinois at Urbana-Champaign College of
Liberal Arts and Sciences, Urbana, IL, USA
- Diletta Barbiani**, Department of Neuroscience, University of Turin Medical School, Turin,
Italy
Hypoxia Medicine, Plateau Rosà, Switzerland
- Jeffrey E. Barnett**, College of Arts and Sciences, Loyola University Maryland, Baltimore,
MD, USA
- Petronilla Battista**, Istituti Clinici Scientifici Maugeri IRCCS, Pavia, Institute of Bari, Italy
- Brittney Becker**, Psychology Department, Texas A&M University System, College Station,
TX, USA
- Alexandra A. Belzer**, Colby College, Waterville, ME, USA
- Fabrizio Benedetti**, Department of Neuroscience, University of Turin Medical School,
Turin, Italy
Hypoxia Medicine, Plateau Rosà, Switzerland
- Danielle S. Berke**, Massachusetts Veterans Epidemiology Research and Information Center
(MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Mamta Bhatnagar**, Department of Medicine, University of Pittsburgh Medical Center,
Pittsburgh, PA, USA
- Kelly Biegler**, Department of Psychological Science, University of California, Irvine, CA,
USA
- Susan J. Blalock**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill,
Chapel Hill, NC, USA
- Brittany E. Blanchard**, Department of Psychological Sciences, Texas Tech University,
Lubbock, TX, USA
- Jennifer B. Blossom**, Clinical Child Psychology Program, University of Kansas, Lawrence,
KS, USA
- Ryan Bogdan**, BRAIN Lab, Psychological and Brain Sciences, Washington University in
St. Louis, St. Louis, MO, USA
- Jacob Bolzenius**, Missouri Institute of Mental Health, University of Missouri-St. Louis,
St. Louis, MO, USA
- Bruce Bongar**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Dianna Boone**, Clinical Psychology Program, Department of Psychological Sciences, Texas
Tech University, Lubbock, TX, USA
- Jerica X. Bornstein**, Department of Human Development and Family Sciences, University of
Texas at Austin, Austin, TX, USA
- Joaquín Borrego Jr.**, Department of Psychological Sciences, Texas Tech University, Lubbock,
TX, USA
- Kyle J. Bourassa**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Lara A. Boyd**, Department of Physical Therapy, University of British Columbia, Vancouver,
BC, Canada
Djavad Mowafaghian Centre for Brain Health, University of British Columbia, Vancouver,
BC, Canada
- Patrick Boyd**, Department of Psychology, University of South Florida, Tampa, FL, USA
- Adam M. Brickman**, Columbia University Medical Center, New York, NY, USA
- Athena Brindle**, Department of Psychology, College of Science, The University of Texas at
Arlington, Arlington, TX, USA

- Jennifer L. Brown**, Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati College of Medicine, Cincinnati, OH, USA
- Kathleen S. Brown**, Private Practice, Fort Myers, FL, USA
- Wilson J. Brown**, Pennsylvania State University, The Behrend College, Erie, PA, USA
- Steven E. Bruce**, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Angela D. Bryan**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Katherine R. Buchholz**, VA Boston Healthcare System, Boston, MA, USA
National Center for Post Traumatic Stress Disorder, White River Junction, VT, USA
- Claire Burgess**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Edith Burns**, Medical College of Wisconsin, Milwaukee, WI, USA
- Rachel J. Burns**, Douglas Mental Health University Institute, McGill University, Montreal, QC, Canada
- Johannes B.J. Bussmann**, Erasmus MC University Medical Center Rotterdam, CA Rotterdam, South Holland, The Netherlands
- Fawn C. Caplandies**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Angela L. Carey**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Brandon L. Carlisle**, Department of Psychiatry, University of California, San Diego, CA, USA
- Delesha M. Carpenter**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Allison J. Carroll**, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL, USA
- Charles S. Carver**, Department of Psychology, University of Miami, Coral Gables, FL, USA
Center for Advanced Study in the Behavioral Sciences, Stanford University, Stanford, CA, USA
- Isabella Castiglioni**, Dipartimento di Fisica “G. Occhialini”, Università degli Studi di Milano-Bicocca – Piazza della Scienza, Milan, Italy
- Gretchen B. Chapman**, Department of Psychology, Rutgers University–New Brunswick, New Brunswick, NJ, USA
- Alyssa C.D. Cheadle**, Psychology Department, Hope College, Holland, MI, USA
- Rebecca E. Chesher**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- William J. Chopik**, Psychology Department, Michigan State University, East Lansing, MI, USA
- Nicholas J.S. Christenfeld**, Department of Psychology, University of California San Diego, La Jolla, CA, USA
- L.E. Chun**, University of Colorado, Boulder, CO, USA
- Briana Cobos**, Department of Psychology, Texas State University, San Marcos, TX, USA
- Alex S. Cohen**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Jenna Herold Cohen**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Lee M. Cohen**, Department of Psychology, College of Liberal Arts, The University of Mississippi, Oxford, MS, USA
- Catherine Cook-Cottone**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA

- Sarah A. Cooley**, Department of Neurology, School of Medicine, Washington University in Saint Louis, St. Louis, MO, USA
- Stephen Correia**, Department of Psychiatry and Human Behavior, Alpert Medical School, Brown University, Providence, RI, USA
- Patrick Corrigan**, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
- Juliann Cortese**, School of Information, Florida State University, Tallahassee, FL, USA
- Riley Cropper**, Stanford University Counseling and Psychological Services, Stanford Hospital and Clinics, Stanford, CA, USA
- Robert T. Croyle**, National Cancer Institute, Bethesda, MD, USA
- Tegan Cruwys**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Sasha Cukier**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Gregory Dahl**, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Beth D. Darnall**, Department of Anesthesiology, Perioperative, and Pain Medicine, Stanford University School of Medicine, Stanford, CA, USA
- Savannah Davidson**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Elizabeth L. Davis**, Department of Psychology, University of California, Riverside, CA, USA
- Rachael L. Deardorff**, Center for Biomedical Imaging, Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Janet Deatrick**, Department of Family & Community Health, University of Pennsylvania School of Nursing, Philadelphia, PA, USA
- Lauren Decaportale-Ryan**, Departments of Psychiatry, Medicine, & Surgery, University of Rochester Medical Center, Rochester, NY, USA
- Jordan Degelia**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Anita Delongis**, Department of Psychology, University of British Columbia, Vancouver, BC, Canada
- Naiara Demnitz**, Department of Psychiatry, University of Oxford, Oxford, UK
- Eliot Dennard**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Kristine M. Diaz**, Walter Reed National Military Medical Center, Bethesda, MD, USA
- Sally S. Dickerson**, Department of Psychology, Pace University, New York, NY, USA
- M. Robin DiMatteo**, Department of Psychology, University of California Riverside, Riverside, CA, USA
- Sona Dimidjian**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Genevieve A. Dingle**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Laura J. Dixon**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Michelle R. Dixon**, Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Tiffany S. Doherty**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Michael D. Dooley**, Department of Psychology, University of California, Riverside, Riverside, CA, USA

- Tessa L. Dover**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Colleen Doyle**, Institute of Child Development, College of Education and Human Development, University of Minnesota, Minneapolis, MN, USA
- Ira Driscoll**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Claudia Drossel**, Department of Psychology, Eastern Michigan University College of Arts and Sciences, Ypsilanti, MI, USA
- Sean P.A. Drummond**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Christine Dunkel Schetter**, Department of Psychology, University of California, Los Angeles, CA, USA
- William L. Dunlop**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Jacqueline C. Duperrouzel**, Florida International University, Miami, FL, USA
- Allison Earl**, Department of Psychology, University of Michigan, Ann Arbor, MI, USA
- Amanda C. Eaton**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Ulrich W. Ebner-Priemer**, Karlsruhe Institute of Technology, Karlsruhe, Germany
- Robin S. Edelstein**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Jamie Edgin**, Department of Psychology, The University of Arizona, Tucson, AZ, USA
- Bradley A. Edwards**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Naomi I. Eisenberger**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Amber S. Emanuel**, Psychology Department, University of Florida, Gainesville, FL, USA
- Patrick England**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Ipek Ensari**, Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA
- Emily Evans**, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
- James Evans**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Anne M. Fairlie**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Angelica Falkenstein**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Zachary Fetterman**, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
- Christopher Fink**, Durham Veterans Affairs Medical Center, Durham, NC, USA
- North Carolina State University, Raleigh, NC, USA
- Alexandra N. Fisher**, Psychology Department, University of Victoria, Victoria, BC, Canada
- Tabitha Fleming**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Tasheia Floyd**, Washington University School of Medicine, St. Louis, MO, USA
- Lauren A. Fowler**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA

- Elinor Flynn**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Heather Walton Flynn**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Elizabeth S. Focella**, Department of Psychology, University of Wisconsin Oshkosh, Oshkosh, WI, USA
- Amy Frers**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA
- Howard S. Friedman**, Psychology Department, University of California Riverside, Riverside, CA, USA
- Paul T. Fuglestad**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Kentaro Fujita**, Department of Psychology, The Ohio State University, Columbus, OH, USA
- Kristel M. Gallagher**, Thiel College, Greenville, PA, USA
- Andrea M. Garcia**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Paola García-Egan**, Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA
- Robert García**, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA
- Casey K. Gardiner**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Robert J. Gatchel**, Endowment Professor of Clinical Health Psychology Research, Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA
- Justine Megan Gatt**, Neuroscience Research Australia, Sydney, NSW, Australia
School of Psychology, University of New South Wales, Sydney, NSW, Australia
- Nicole K. Gause**, Department of Psychology, University of Cincinnati, Cincinnati, OH, USA
- Ashwin Gautam**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Fiona Ge**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Andrew L. Geers**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Margaux C. Genoff**, Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, NY, USA
- James I. Gerhart**, Department of Psychosocial Oncology, Cancer Integrative Medicine Program, Rush University Medical Center, Chicago, IL, USA
- Meg Gerrard**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Frederick X. Gibbons**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Arielle S. Gillman**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Marci E.J. Gleason**, Department of Human Development and Family Sciences, University of Texas at Austin, Austin, TX, USA
- Teresa Gobble**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA

- Katharina Sophia Goerlich**, Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany
- Jamie L. Goldenberg**, Department of Psychology, University of South Florida, Tampa, FL, USA
- Cesar A. Gonzalez**, Mayo Clinic Minnesota, Rochester, MN, USA
- Raul Gonzalez**, Florida International University, Miami, FL, USA
- Brittany F. Goodman**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Janna R. Gordon**, San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- Edward C. Green**, Department of Anthropology, The George Washington University, Washington, DC, USA
- Joseph P. Green**, Department of Psychology, Ohio State University at Lima, Lima, OH, USA
- Michael G. Griffin**, Department of Psychological Sciences and Center for Trauma Recovery, University of Missouri–St. Louis, St. Louis, MO, USA
- Steven Grindel**, Medical College of Wisconsin, Milwaukee, WI, USA
- Kristina Gryboski**, Project Hope, Millwood, VA, USA
- Suzy Bird Gulliver**, Baylor and Scott and White Hospitals and Clinics, Waco, TX, USA
School of Public Health, Texas A&M University, College Station, TX, USA
- Megan R. Gunnar**, Institute of Child Development, College of Education and Human Development, University of Minnesota, Minneapolis, MN, USA
- Jenna Haddock**, Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA
- Martin S. Hagger**, Health Psychology and Behavioral Medicine Research Group, School of Psychology and Speech Pathology, Faculty of Health Sciences, Curtin University, Perth, WA, Australia
Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland
School of Applied Psychology and Menzies Health Institute Queensland, Behavioural Bases for Health, Griffith University, Brisbane, QLD, Australia
Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia
- Jada G. Hamilton**, Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, NY, USA
- Paul K.J. Han**, Center for Outcomes Research and Evaluation, Maine Medical Center Research Institute, Portland, ME, USA
- Grace E. Hanley**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Madeline B. Harms**, Department of Psychology, University of Wisconsin-Madison, Madison, WI, USA
- Adam Harris**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA
- Lauren N. Harris**, VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA
- Peter R. Harris**, School of Psychology, University of Sussex, Brighton & Hove, UK
- Philine S. Harris**, School of Psychology, University of Sussex, Brighton & Hove, UK
- William Hart**, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
- M.J. Hartsock**, University of Colorado, Boulder, CO, USA

- Kelly B. Haskard-Zolnierrek**, Department of Psychology, Texas State University, San Marcos, TX, USA
- S. Alexandar Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Catherine Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Karen Hasselmo**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Kathryn S. Hayward**, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada
- Stroke Division, Florey Institute of Neuroscience and Mental Health, University of Melbourne, Melbourne, VIC, Australia
- NHMRC Centre for Research Excellence in Stroke Rehabilitation and Recovery, Parkville, VIC, Australia
- Jodi M. Heaps-Woodruff**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Dietlinde Heilmayr**, Psychology Department, Moravian College, Bethlehem, PA, USA
- Vicki S. Helgeson**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Joseph A. Helpern**, Center for Biomedical Imaging, Department of Neuroscience, Department of Neurology, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Garrett Hisler**, Department of Psychology, Iowa State University, Ames, IA, USA
- Molly B. Hodgkins**, Department of Psychological and Brain Sciences, University of Maine, Orono, ME, USA
- Michael Hoerger**, Department of Psychology, Tulane University, New Orleans, LA, USA
- Jeanne S. Hoffman**, US Department of the Army, Washington, DC, USA
- Stephanie A. Hooker**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Vera Hoorens**, Center for Social and Cultural Psychology, Katholieke Universiteit Leuven, Leuven, Belgium
- Arpine Hovasapian**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jennifer L. Howell**, Psychology Department, Ohio University, Athens, OH, USA
- Robert M. Huff**, Department of Health Sciences, California State University, Northridge, Morgan Hill, CA, USA
- Jacob Huffman**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Department of Psychiatry, Washington University in St. Louis School of Medicine, St. Louis, MO, USA
- Lauren J. Human**, Department of Psychology, McGill University, Montreal, QC, Canada
- Jeffrey M. Hunger**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Tanya Hunt**, Department of Clinical Psychology, Palo Alto University, Palo Alto, California, USA
- Steven Hyde**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- Christina Di Iorio**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA

- Jamie M. Jacobs**, Department of Psychiatry, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA
- Lauren James**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Rebekah Jazdzewski**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Brooke N. Jenkins**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jens H. Jensen**, Center for Biomedical Imaging, Department of Neuroscience, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Donna C. Jessop**, School of Psychology, University of Sussex, Brighton & Hove, UK
- Jolanda Jetten**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Kimberly Johnson**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Doerte U. Junghaenel**, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA
- Vanessa Juth**, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA
- Samantha Kahn**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- David A. Kalkstein**, Department of Psychology, New York University, New York, NY, USA
- Alexander Karan**, Department of Psychology, University of California, Riverside, CA, USA
- Natalia M. Kecala**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri–St. Louis, St. Louis, MO, USA
- Robert G. Kent de Grey**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- Margaret L. Kern**, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia
- Nathan A. Kimbrel**, Department of Veterans Affairs' VISN 6 Mid-Atlantic MIRECC, Durham, NC, USA
Durham Veterans Affairs Medical Center, Durham, NC, USA
Psychiatry and Behavioral Sciences, Duke University School of Medicine, Durham, NC, USA
- Tricia Z. King**, The Neuroscience Institute, Georgia State University, Atlanta, GA, USA
- Nancy Kishino**, West Coast Spine Restoration, Riverside, CA, USA
- Ekaterina Klyachko**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Brian Konecky**, Boise VAMC Virtual Integrated Multisite Patient Aligned Care Team Hub, Boise, ID, USA
- Gerald P. Koocher**, Department of Psychology, DePaul University, Chicago, IL, USA
- Laura E. Korthauer**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Abigail O. Kramer**, Palo Alto University, Palo Alto, CA, USA
Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Joel H. Kramer**, Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Zlatan Krizan**, Department of Psychology, Iowa State University, Ames, IA, USA

- Monica F. Kurylo**, Departments of Medicine and Public Health, University of Kansas Medical Center, Kansas City, KS, USA
- Onawa P. LaBelle**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa H. Laitner**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Mark J. Landau**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Shane Landry**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Kevin T. Larkin**, Department of Psychology, West Virginia University, Morgantown, WV, USA
- Jason M. Lavender**, Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA
- Kristin Layous**, Department of Psychology, California State University East Bay, Hayward, CA, USA
- Thanh Le**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Thad R. Leffingwell**, Department of Psychology, Oklahoma State University, Stillwater, OK, USA
- Angela M. Legg**, Department of Psychology, Pace University, New York, NY, USA
- Jana Lembke**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Elizabeth Lemon**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Richie L. Lenne**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Alex Leow**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Department of Bioengineering, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Howard Leventhal**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Linda J. Levine**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Melissa A. Lewis**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Meng Li**, Department of Health and Behavior Sciences, University of Colorado Denver, Denver, CO, USA
- Huijun Liao**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Alexander P. Lin**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Wendy P. Linda**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Nikolette P. Lipsey**, Psychology Department, University of Florida, Gainesville, FL, USA
- Dana M. Litt**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

- Andrew K. Littlefield**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Brett Litz**, Department of Psychology, Boston University & VA Boston Healthcare System, Boston, MA, USA
- Marci Lobel**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Todd Lucas**, C. S. Mott Endowed Professor of Public Health Associate Professor of Epidemiology and Biostatistics Division of Public Health College of Human Medicine Michigan State University, Wayne State University, Detroit, MI, USA
- Mia Liza A. Lustria**, School of Information, Florida State University, Tallahassee, FL, USA
- Steven Jay Lynn**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Salvatore R. Maddi**, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
- Renee E. Magnan**, Washington State University Vancouver, Vancouver, WA, USA
- Brenda Major**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Vanessa L. Malcarne**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
UC San Diego Moores Cancer Center, San Diego, CA, USA
Department of Psychology, San Diego State University, San Diego, CA, USA
- Kelsey A. Maloney**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Andrew W. Manigault**, Psychology Department, Ohio University, Athens, OH, USA
- Traci Mann**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Soe Mar**, Neurology and Pediatrics, Division of Pediatric Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Shannon M. Martin**, Department of Psychology, Central Michigan University, Mount Pleasant, MI, USA
- Allison Marziliano**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Katilyn Mascatelli**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Tyler B. Mason**, Department of Psychology, University of Southern California, Los Angeles, CA, USA
Neuropsychiatric Research Institute, Fargo, ND, USA
- Kevin S. Masters**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Kevin D. McCaul**, North Dakota State University, Fargo, ND, USA
- Tara P. McCoy**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Michael McCrea**, Brain Injury Research Program, Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA
- Scout N. McCully**, Department of Psychological Sciences, Kent State University, Kent OH, USA
- Don McGeary**, Department of Psychiatry, Department of Family and Community Medicine, The University of Texas Health Science Center San Antonio, San Antonio, TX, USA

- Jessica McGovern**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Robert E. McGrath**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Michael P. Mead**, North Dakota State University, Fargo, ND, USA
- Alan Meca**, Department of Psychology, Old Dominion University, Norfolk, VA, USA
- Matthias R. Mehl**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Timothy P. Melchert**, Department of Psychology, Marquette University, Milwaukee, WI, USA
- Paola Mendoza-Rivera**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Maria G. Mens**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Sai Merugumala**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Rebecca A. Meza**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Dara Mickschl**, Medical College of Wisconsin, Milwaukee, WI, USA
- Tricia A. Miller**, Santa Barbara Cottage Hospital, Santa Barbara, CA, USA
- Department of Psychology, University of California Riverside, Riverside, CA, USA
- Kyle R. Mitchell**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Mona Moieni**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Phillip J. Moore**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- DeAnna L. Mori**, VA Boston Healthcare System, West Roxbury, MA, USA
- Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
- Sandra B. Morissette**, Department of Psychology, The University of Texas at San Antonio College of Sciences, San Antonio, TX, USA
- Amber Morrow**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Ariel J. Mosley**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Robert W. Motl**, Department of Physical Therapy, University of Alabama at Birmingham, Birmingham, AL, USA
- Laurent Mottron**, Department of Psychiatry, Faculty of Medicine, Universite de Montreal, Montreal, QC, Canada
- Anne Moyer**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Elaine M. Murphy**, Population Reference Bureau, Washington, DC, USA
- Carolyn B. Murray**, Psychology, University of California, Riverside, CA, USA
- Christopher S. Nave**, Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA
- Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Eve-Lynn Nelson**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Steven M. Nelson**, VISN 17 Center of Excellence for Research on Returning War Veterans, Waco, TX, USA
- Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA

Center for Vital Longevity, School of Behavioral and Brain Sciences, University of Texas at Dallas, Dallas, TX, USA

Elizabeth Neumann, Medical College of Wisconsin, Milwaukee, WI, USA

Jason L. Neva, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada

Jody S. Nicholson, Department of Psychology, University of North Florida, Jacksonville, FL, USA

Christina Nisson, Department of Psychology, University of Michigan, Ann Arbor, MI, USA

Mary-Frances O'Connor, Psychology Department, University of Arizona, Tucson, AZ, USA

Laura O'Hara, Department of Communication, Fontbonne University, St. Louis, MO, USA

Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA

Gbolahan O. Olanubi, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA

Nils Olsen, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA

Yimi Omofuma, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA

Elizabeth Ortiz-Gonzalez, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Ileana Pacheco-Colón, Florida International University, Miami, FL, USA

Aliza A. Panjwani, Graduate School and University Center, City University of New York, New York, NY, USA

Mike C. Parent, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA

Thomas J. Parkman, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Robert H. Paul, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

Missouri Institute of Mental Health, St. Louis, MO, USA

Stephanie A. Peak, Battelle Memorial Institute, Columbus, OH, USA

Michael G. Perri, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

Laura Perry, Department of Psychology, Tulane University, New Orleans, LA, USA

Alyssa Phelps, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Carissa L. Philippi, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

L. Alison Phillips, Iowa State University, Ames, IA, USA

Paula R. Pietromonaco, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA

Craig P. Pollizi, Department of Psychology, Binghamton University, Binghamton, NY, USA

Seth D. Pollak, Department of Psychology, University of Wisconsin–Madison, Madison, WI, USA

David B. Portnoy, US Food and Drug Administration, Silver Spring, MD, USA

Rachel Postupack, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

- Matthew R. Powell**, Division of Neurocognitive Disorders, Department of Psychiatry & Psychology, Mayo Clinic Minnesota, Rochester, MN, USA
- Candice Presseau**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Sarah D. Pressman**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Rebecca N. Preston-Campbell**, Missouri Institute of Mental Health, St. Louis, MO, USA
- Isabel F. Ramos**, Department of Psychology, University of California, Los Angeles, CA, USA
- Christopher T. Ray**, College of Health Sciences, Texas Woman's University, Denton, TX, USA
- Amanda L. Rebar**, Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia
- Allecia E. Reid**, Colby College, Waterville, ME, USA
- May Reinert**, Department of Psychology, Pace University, New York, NY, USA
- Kelly E. Rentscher**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Tracey A. Revenson**, Department of Psychology, Hunter College, New York, NY, USA
- C. Steven Richards**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Laura River**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Megan L. Robbins**, Department of Psychology, University of California, Riverside, CA, USA
- Michael C. Roberts**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- John D. Robinson**, Department of Surgery and Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine/Hospital, Washington, DC, USA
- Theodore F. Robles**, Psychology Department, University of California Los Angeles, CA, USA
- Catherine Rochefort**, Department of Psychology, Southern Methodist University, Dallas, TX, USA
- Daniel G. Rogers**, Department of Psychology, University of Mississippi, MS, USA
- John L. Romano**, Department of Educational Psychology, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Karen S. Rook**, Department of Psychological Science, University of California, Irvine, CA, USA
- Ann Rosenthal**, Medical College of Wisconsin, Milwaukee, WI, USA
- J. Megan Ross**, Florida International University, Miami, FL, USA
- Tania L. Roth**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Alexander J. Rothman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Ronald H. Rozensky**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Kristen L. Rudd**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Kirstie K. Russell**, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada
- Arseny A. Ryazanov**, Department of Psychology, University of California San Diego, La Jolla, CA, USA

- Napapon Sailasuta**, Centre for Addiction and Mental Health, Toronto, ON, Canada
Stella Sakhon, Department of Psychology, The University of Arizona, Tucson, AZ, USA
Lauren E. Salminen, Imaging Genetics Center, University of Southern California, Marina del Rey, CA, USA
Christian Salvatore, Scuola Universitaria Superiore IUSS Pavia, Piazza della Vittoria, Pavia, Italy
DeepTrace Technologies S.R.L., Via Conservatorio, Milan, Italy
Peter M. Sandman, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
Keith Sanford, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
David A. Sbarra, Department of Psychology, University of Arizona, Tucson, AZ, USA
Michael F. Scheier, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
Adam T. Schmidt, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Elana Schwartz, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
Esther N. Schwartz, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
Seth J. Schwartz, Department of Public Health Sciences, University of Miami Miller School of Medicine, Miami, FL, USA
Ralf Schwarzer, Health Psychology, Freie Universität Berlin, Berlin, Germany
Health Psychology, Australian Catholic University, Melbourne, VIC, Australia
Richard J. Seime, Mayo Clinic Minnesota, Rochester, MN, USA
Claire E. Sexton, Department of Psychiatry, University of Oxford, Oxford, UK
Louise Sharpe, School of Psychology, Faculty of Science, The University of Sydney, Sydney, NSW, Australia
Lindsay Sheehan, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
Paschal Sheeran, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
Daniel Sheinbein, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
James A. Shepperd, Psychology Department, University of Florida, Gainesville, FL, USA
Brittany N. Sherrill, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
Molin Shi, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Emily W. Shih, Department of Psychology, University of California, Riverside, CA, USA
Ben W. Shulman, Psychology Department, University of California, Los Angeles, CA, USA
Elizabeth Silberman, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
Department of Neurology, Oregon Health and Science University, Portland, OR, USA
Roxane Cohen Silver, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
Colleen A. Sloan, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA

Rachel Smallman, Psychology Department, Texas A&M University System, College Station, TX, USA

Kathryn E. Smith, Neuropsychiatric Research Institute, Fargo, ND, USA
Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA

Patrick J. Smith, Duke University Medical Center, Durham, NC, USA

Todd A. Smitherman, Department of Psychology, University of Mississippi, University, MS, USA

Joshua M. Smyth, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA

Dara H. Sorkin, Department of Psychological Science, University of California, Irvine, CA, USA

R. L. Spencer, University of Colorado, Boulder, CO, USA

Melissa Spina, Psychology Department, University of Missouri System, Columbia, MO, USA

Bonnie Spring, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA

Annette L. Stanton, Department of Psychology, University of California, Los Angeles, Los Angeles, CA, USA

Ellen Stephenson, Department of Psychology, University of British Columbia, Vancouver, BC, Canada

Robert A. Stern, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Department of Neurosurgery and Anatomy & Neurobiology, Boston University School of Medicine, Boston, MA, USA

Angela K. Stevens, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Courtney J. Stevens, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA

Elizabeth M. Stewart, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia

Danu Anthony Stinson, Psychology Department, University of Victoria, Victoria, BC, Canada

Michelle L. Stock, Department of Psychological and Brain Sciences, George Washington University, Washington, DC, USA

Arthur A. Stone, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA

Department of Psychology, Dana and David Dornsife College of Letters Arts and Sciences, University of Southern California, Los Angeles, CA, USA

Jeff Stone, Psychology Department, University of Arizona, Tucson, AZ, USA

John A. Sturgeon, Center for Pain Relief, University of Washington, Seattle, WA, USA

Kieran T. Sullivan, Department of Psychology, Santa Clara University, Santa Clara, CA, USA

Amy Summerville, Psychology Department, Miami University, Oxford, OH, USA

Jessie Sun, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia

Department of Psychology, University of California, Davis, CA, USA

Steve Sussman, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

- Trevor James Swanson**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Allison M. Sweeney**, Department of Psychology, University of South Carolina, Columbia, SC, USA
- Kate Sweeny**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Sergey Tarima**, Medical College of Wisconsin, Milwaukee, WI, USA
- Carly A. Taylor**, Boston Medical Center, Boston, MA, USA
- George T. Taylor**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Interfakultäre Biomedizinische Forschungseinrichtung (IBF) der Universität Heidelberg, Heidelberg, Germany
- A. Janet Tomiyama**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Victoria A. Torres**, Department of Psychology, College of Liberal Arts, University of Mississippi, Oxford, MS, USA
- David R.M. Trotter**, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
- Bert N. Uchino**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- John A. Updegraff**, Department of Psychological Sciences, Kent State University, Kent, OH, USA
- Jay Urbain**, Milwaukee School of Engineering, Milwaukee, WI, USA
- Amy E. Ustjanauskas**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- UC San Diego Moores Cancer Center, San Diego, CA, USA
- Jason Van Allen**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Erika D. Van Dyke**, Department of Sport Sciences, College of Physical Activity and Sport Sciences, West Virginia University, Morgantown, WV, USA
- Department of Psychology, Springfield College, Springfield, MA, USA
- Judy L. Van Raalte**, Department of Psychology, Springfield College, Springfield, MA, USA
- Maryke Van Zyl-Harrison**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Rachel Vanderkruik**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Elizabeth J. Vella**, Department of Psychology, University of Southern Maine, Portland, ME, USA
- Birte von Haaren-Mack**, German Sport University Cologne, Köln, Nordrhein-Westfalen, Germany
- David Von Nordheim**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Mikhail Votinov**, Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany
- Institute of Neuroscience and Medicine (INM-10), Jülich, Germany
- Rebecca K. Vujnovic**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA
- Amy Wachholtz**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA

- Eric F. Wagner**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Ryan J. Walker**, Psychology Department, Miami University, Oxford, Ohio, USA
- Rachel A. Wamser-Nanney**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Britney M. Wardecker**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa Ward-Peterson**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Lisa Marie Warner**, Health Psychology, Freie Universitat Berlin, Berlin, Germany
- Neil D. Weinstein**, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
- Suzanne E. Welcome**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Kristen J. Wells**, San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- Amy Wenzel**, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, PA, USA
- Private Practice, Rosemont, PA, USA
- Summer L. Williams**, Department of Psychology, Westfield State University, Westfield, MA, USA
- Cynthia J. Willmon**, Reno VA Sierra Nevada Health Care System, Reno, NV, USA
- Sara M. Witcraft**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Stephen A. Wonderlich**, Neuropsychiatric Research Institute, Fargo, ND, USA
- Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA
- E.R. Woodruff**, University of Colorado, Boulder, CO, USA
- Patrick W. Wright**, Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Robert C. Wright**, Department of Psychology, University of California, Riverside, CA, USA
- Shawna Wright**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Tuppett M. Yates**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Julie D. Yeterian**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Lok-Kin Yeung**, Columbia University Medical Center, New York, NY, USA
- Alex J. Zautra**, Psychology Department, Arizona State University, Tempe, AZ, USA
- Colin A. Zestcott**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Peggy M. Zoccola**, Psychology Department, Ohio University, Athens, OH, USA

Foreword

Until the 1970s, there were no books, journals, or university courses on health psychology. Although the field's intellectual roots stretch back to the beginnings of psychology more than a century ago, its formal emergence depended on a convergence of influences (Friedman & Silver, 2007), including psychosomatic medicine, social-psychological and socio-anthropological perspectives on medicine, epidemiology, and medical and clinical psychology. Today, health psychology is a principal area of significant social science research and practice, with vital implications for the health and well-being of individuals and societies. Understanding the explosive trajectory of health psychology is useful to appreciating the strengths of the field and to approaching this new encyclopedia, the *Wiley Encyclopedia of Health Psychology*.

What is the nature of health? That is, what does it mean to be healthy? The way that this question is answered affects the behaviors, treatments, and resource allocations of individuals, families, health practitioners, governments, and societies. For example, if it is thought that you are healthy unless and until you contract a disease or suffer an injury, then attention and resources are primarily allocated toward “fixing” the problem through medications or surgical repairs. This is the traditional biomedical model of disease (sometimes called the “disease model”). Indeed, in the United States, the overwhelming allocation of attention and resources is to physicians (doing treatments) and to pharmaceuticals (prescription drugs and their development). In contrast, health psychology developed around a much broader and more interdisciplinary approach to health, one that is often termed the biopsychosocial model.

The biopsychosocial model (a term first formally proposed by George Engel in 1968) brings together core elements of staying healthy and recovering well from injury or disease (Stone et al., 1987). Each individual—due to a combination of biological influences and psychosocial experiences—is more or less likely to thrive. Some of this variation is due to genetics and early life development; some depends on the availability and appropriate applications of medical treatments; some involves nutrition and physical activity; some depends on preparations for, perceptions of, and reactions to life's challenges; and some involves exposure to or seeking out of healthier or unhealthier environments, both physically and socially. When presented in this way, it might seem obvious that health should certainly be viewed in this broader interdisciplinary way. However, by misdirecting its vast expenditures on health care, the United States gives its residents mediocre health at high cost (Kaplan, 2019). To approach these matters in a thorough manner, this encyclopedia includes four volumes, with Volume 1 focusing on the biological bases of health and health behavior, Volume 2 concentrating on the social bases, Volume 3 centering around the psychological and clinical aspects, and Volume 4 focused more broadly on crosscutting and applied matters.

Key parts of health depend on biological characteristics and how they interact with our experiences and environments. So, for example, in Volume 1, there are articles on injury to the brain, alcohol effects on the brain, nutrition, drug abuse, psychophysiology, and the tools and key findings of neuroscience. Note that even individuals with the same genes (identical twins) can and do have different health and recovery outcomes, and the articles delve into such complexities of health. Of course humans are also social creatures, and people's growth, development, and health behaviors take place in social contexts. So, in Volume 2, there are articles on such fundamental matters as social support, coping, spirituality, emotion, discrimination, communication, psychosocial stress, and bereavement.

Because our approaches to and conceptions of health are heavily influenced by society's institutions and structures revolving around medical care, much of health psychology derives from or intersects with clinical psychology and applied behavioral medicine. Volume 3 covers such clinical topics as psycho-oncology, depression, drug abuse, chronic disease, eating disorders, and the psychosocial aspects of coronary heart disease. Finally and importantly, there are a number of special and cross-cutting matters that are considered in Volume 4, ranging from relevant laws and regulations to telehealth and health disparities. Taken together, the articles triangulate on what it truly means to be healthy.

What are the most promising directions for the future that emerge from a broad and deep approach to health? That is, to where do these encyclopedia articles point? One key clue arises from a unique opportunity to enhance, extend, and analyze the classic Terman study of children who were followed and studied throughout their lives (Friedman & Martin, 2012). These studies revealed that there are lifelong trajectories to health, thriving, and longevity. Although anyone can encounter bad luck, a number of basic patterns emerged that are more likely to lead to good health. That is, for some individuals, certain earlier life characteristics and circumstances help propel them on *pathways* of healthier and healthier behaviors, reactions, relationships, and experiences, while others instead face a series of contingent stumbling blocks. There are multipart but nonrandom pathways across time linking personalities, health behaviors, social groups, education, work environments, and health and longevity. The present encyclopedia necessarily is a compendium of summaries of the relevant elements of health and thriving, but one that would and can profitably be used as a base to synthesize the long-term interdependent aspects of health.

In sum, this encyclopedia is distinctive in its explicit embrace of the biopsychosocial approach to health, not through lip service or hand-waving but rather through highly detailed and extensive consideration of the many dozens of topics crucial to this core interdisciplinary understanding.

References

- Friedman, H. S., & Martin, L. R. (2012). *The longevity project: Surprising discoveries for health and long life from the landmark eight-decade study*. New York: Penguin Plume Press.
- Friedman, H. S., & Silver, R. C. (Eds.) (2007). *Foundations of health psychology*. New York: Oxford University Press.
- Kaplan, R. M. (2019). *More than medicine: The broken promise of American health*. Cambridge, MA: Harvard University Press.
- Stone, G., Weiss, M., Matarazzo, J. D., Miller, N. E., Rodin, J., Belar, C. D., ... Singer, J. E. (Eds.) (1987). *Health psychology: A discipline and a profession*. University of Chicago Press.

Howard S. Friedman
University of California, Riverside
August 15, 2019

Preface

The field of health psychology is a specialty area that draws on how biology, psychology, behavior, and social factors influence health and illness. Despite the fact that the formal recognition of the field is only about a half of a century old, it has established itself as a major scientific and clinical discipline. The primary reason for this is that there have been a number of significant advances in psychological, medical, and physiological research that have changed the way we think about health, wellness, and illness. However, this is only the tip of the iceberg.

We have the field of health psychology to thank for much of the progress seen across our current healthcare system. Let me provide some examples that illustrate the diversity of these important contributions. Starting with a broad public health perspective, health psychologists have been involved in how our communities are planned and how urban development has a significant and direct impact on our health behaviors. That is, if we live close to where we work, play, and shop, we are more likely to walk or bike to our destination rather than drive or take public transportation. Focusing on a smaller, but no less important, source of data, health psychologists are involved in using information obtained from our genes to help counsel individuals to make good, well-informed health decisions that could have an impact on the individual immediately or in the future. Being well-informed can direct a person toward the best possible path toward wellness, whether it be measures aimed at prevention, further monitoring, or intervention.

Of course, there are many other contributions. For instance, data indicates that several of the leading causes of death in our society can be prevented or delayed (e.g., heart disease, respiratory disease, cerebrovascular disease, and diabetes) via active participation in psychological interventions. Knowing that we can improve health status by changing our behaviors seems like an easy “fix,” but we know that behavior change is tough. As such, it is not surprising that health psychologists have been involved in trying to improve upon treatment success by examining patient compliance. When we better understand what motivates and discourages people from engaging in treatment or pro-health behaviors, we can improve upon compliance and help individuals adopt more healthy lifestyles. Health psychologists have also played a role in shaping healthcare policy via identifying evidence-based treatments. This work has direct effects on how individuals receive healthcare as well as what treatments are available/reimbursable by insurance companies. Finally, there are also factors beyond medical care to consider (e.g., economic, educational) that can lead to differential health outcomes. Thus, health psychologists likewise examine ways to reduce health disparities, ensuring that the public and government officials are made aware of the impact of the social determinants of health.

I could go on and on, however, examples like this illustrate the significant impact this broad and exciting field has had (and will continue to have) on our understanding of health and wellness. Given the constantly changing nature of the field, it is not possible to be all inclusive; however, the aim of these four volumes is to provide readers an up-to-date overview of the field. Each entry is written to stand alone for those who wish to learn about a specific topic, and if the reader is left wanting more, suggested readings are provided to expand one's knowledge. Volume I, *Biological Bases of Health Behavior*, includes entries that cover topics in the broad areas of neuroscience and biopsychology relevant to health behavior. General topics include degenerative and developmental conditions, emerging methodologies available in clinical research, functional anatomy and imaging, and gene×environment interactions. Volume II, *Social Bases of Health Behavior*, addresses topics related to theories and concepts derived from social psychology. Specifically, topics related to the self, social cognition, social perception, attitudes and attitude change, perception, framing, and pro-health behaviors are included. Volume III, *Clinical Health Psychology and Behavioral Medicine*, covers the applied aspects of the field of health psychology including practical topics that clinical health psychologists face in the workplace, behavioral aspects of medical conditions, the impact of unhealthy behaviors, and issues related to the comorbidity of psychiatric disorders and chronic health concerns. Finally, Volume IV, *Special Issues in Health Psychology*, contains a wide array of topics that are worthy of special consideration in the field. Philosophical and conceptual issues are discussed, along with new approaches in delivering treatment and matters to consider when working with diverse and protected populations.

It is my sincere hope that the *Wiley Encyclopedia of Health Psychology* will serve as a comprehensive resource for academic and applied psychologists, other health care professionals interested in the relationship of psychological and physical well-being, and students across the health professions. I would very much like to thank and acknowledge all those who have made this work possible including Michelle McFadden, the Wiley editorial and production teams, the volume editors, and of course all of the authors who contributed their outstanding work.

Lee M. Cohen, PhD

Preface Volume 3: Clinical Health Psychology and Behavioral Medicine

The theory developments, research outcomes, and practice guidelines for the fields of clinical health psychology and behavioral medicine have expanded dramatically during the last 30 years. Thousands of interesting journal articles and hundreds of relevant books have been published during that time interval. Numerous well-regarded journals focus entirely or in part on these clinical health psychology topics, such as the journals *Health Psychology*, *Annals of Behavioral Medicine*, *Journal of Consulting and Clinical Psychology*, *Journal of the American Medical Association*, *New England Journal of Medicine*, and over 100 specialty journals on focused topics such as specific diseases, psychological disorders comorbid with chronic health problems, translational issues in health care, psychosocial interventions in health psychology and behavioral medicine, and so forth. The topics covered in this third volume of the *Wiley Encyclopedia of Health Psychology* is focused on some of the recent theory, research, and practice issues relevant to the field of clinical health psychology.

This volume covers some exciting and important issues in the area. Specifically, entries touch on both broad and specific topics covering (a) methodological sophistication, (b) chronic diseases, (c) diversity and inclusiveness, (d) biological issues, (e) translation of health psychology knowledge, (f) chronic psychopathology that is comorbid with health problems and diseases, and (g) social justice. In sum, we have attempted to include a wide array of entries on what we consider to be the most relevant topics and trends, although we could not include all possible relevant topics and trends. Nevertheless, we believe that readers will find a wide range of interesting and important entries in this volume.

We should say something about our recruitment and editing guidelines for Volume 3. We tried to recruit well-regarded experts—in some cases with national and international reputations—in each of the entry topic areas. Many of the entries are multiauthored, so most of these experts collaborated with other experts in their specific areas. We also tried to recruit a broad sampling across the numerous trends and topic areas in the field, rather than having most or all of the entries focused on just a few topics. Further, we worked diligently to provide the authors feedback that we believed would make the entries stronger in terms of standard scholarship goals such as accuracy, clarity, thoroughness, and adherence to standard professional writing styles in clinical health psychology. It should be noted, however, that virtually all of the entries were already in excellent shape, when we first received them, which dovetails with the outstanding reputations of our authors.

We believe that Volume 3, *Clinical Health Psychology and Behavioral Medicine*, will be a helpful resource book on an important topic. We also believe that readers will find this volume

an excellent addition to their professional library, along with the other volumes in the Wiley *Encyclopedia of Health Psychology*. In closing, we would like to acknowledge the excellent work of our authors, the helpful expertise of the staff at Wiley, the ongoing help of our home universities, and the wonderful support from our families.

C. Steven Richards, PhD
Lee M. Cohen, PhD

Acknowledgments

We would like to acknowledge the excellent work of our authors, the helpful expertise of Michelle McFadden and the staff at Wiley, the ongoing support of our home institutions, Texas Tech University and the University of Mississippi, and the wonderful support from our families—Carol, Dawn, Dave, and Jill (Steve) and Michelle, Ross, Rachel, and Becca (Lee).

C. Steven Richards and Lee M. Cohen
Editors

Editor-in-Chief Acknowledgments

I would like to publicly recognize and thank my family, Michelle, Ross, Rachel, and Becca for being supportive of my work life while also providing me with a family life beyond my wildest dreams. I love you all very much. I would also like to note my great appreciation and thanks to the faculty and staff of the Doctoral Training Program in Clinical Psychology at Oklahoma State University for providing me with an opportunity to expand my education and for providing extraordinary training. In particular, I would like to thank my late mentor Dr. Frank L. Collins Jr., Dr. Larry Mullins, Dr. John Chaney, and Patricia Diaz Alexander. Further, I am grateful to the University of Mississippi and Texas Tech University (and the colleagues and students I have had the privilege to work alongside) for affording me excellent working environments and the support to do the work I am honored to be a part of. Finally, I would like to recognize and thank the volume editors for their vision and perseverance to this project as well as to each of the contributors for their excellent entries and their dedication to this very important field.

Lee M. Cohen, Ph.D.
Editor-in-Chief

Body Image

Tyler B. Mason^{1,2}, Kathryn E. Smith^{2,3},
Jason M. Lavender⁴, and Stephen A. Wonderlich^{2,3}

¹*Department of Psychology, University of Southern California, Los Angeles, CA, USA*

²*Neuropsychiatric Research Institute, Fargo, ND, USA*

³*Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA*

⁴*Department of Psychiatry, University of California, San Diego, CA, USA*

Body image involves a person's subjective appraisal of his or her own physical characteristics and qualities (e.g., Pruzinsky, 2004; Sarwer & Steffen, 2015). Body image concerns are highly prevalent in Western societies and are more prominent among women (Fiske, Fallon, Blissmer, & Redding, 2014), although they occur among both men and women. Additionally, body image concerns appear to be common across the lifespan (Fiske et al., 2014). Evidence suggests that poor body image can negatively affect psychosocial functioning (Patalay, Sharpe, & Wolpert, 2015), health behaviors (Neumark-Sztainer, Paxton, Hannan, Haines, & Story, 2006), and overall quality of life (Mond et al., 2013). Conversely, positive body image is protective against numerous mental health problems and serves to increase overall psychological well-being (Gillen, 2015). Further, several mental disorders associated with mortality and reduced quality of life are marked by disturbances in body image (e.g., body dysmorphic disorder, eating disorders). Of particular relevance to the field of clinical health psychology, poor body image occurs in a variety of medical conditions and clinical populations (e.g., individuals with obesity, patients with cancer). Understanding and routinely assessing body image concerns may have utility in alleviating health problems and reducing further physical and mental health complications. In this entry, we describe body image in the field of clinical health psychology including risk factors and correlates of body image concerns.

Body Image and Clinical Health Psychology

Body image concerns are important health symptoms to consider in clinical health psychology and occur in a myriad of medical conditions and diseases. The most prominent medical condition associated with poor body image is obesity. This relationship is in part propagated by a pervasive cultural emphasis on thinness and the associated stigmatization of individuals who are overweight or obese (Puhl & Latner, 2007). Thus, body image concerns may be a problem among any medical and psychiatric patients who present with co-occurring obesity. Further, while weight loss usually improves overall body image, body image concerns, specifically related to excess skin, may be present among individuals who lose a great deal of weight, notably those who receive bariatric surgery (Song *et al.*, 2006). However, body contouring after large weight loss has been shown to be related to improvements in body image (Song *et al.*, 2006).

Body image concerns are also relatively common in patients with various types of cancer and other medical conditions. For example, disturbances in body image have been observed in patients with breast cancer (Lasry *et al.*, 1987), cervical cancer (Hawighorst-Knapstein *et al.*, 2004), and head and neck cancers (Fingeret *et al.*, 2013). Further, side effects of cancer treatment such as alopecia have also been associated with increases in body image concerns (Münstedt, Manthey, Sachse, & Vahrson, 1997). Other medical conditions and diseases that may involve body image concerns are inflammatory bowel disease and Crohn's disease (Saha *et al.*, 2015), endometriosis (Melis *et al.*, 2015), lupus (Jolly *et al.*, 2012), and systemic sclerosis (Ennis, Herrick, Cassidy, Griffiths, & Richards, 2012). Children and adolescents with various medical conditions may experience a similar pattern of concerns. For instance, in a meta-analysis examining relationships between body image and chronic illness among children and adolescents, Pinquart (2013) found that children and adolescents with obesity, cystic fibrosis, scoliosis, asthma, growth hormone deficits, spina bifida, cancer, and diabetes rated their bodies less positively than healthy comparison groups.

While body image concerns may be associated with particular diseases, certain characteristics of diseases and associated treatments may also be related to the manifestation of body image concerns. For example, disease activity and chronicity, treatment type, treatment complications, and presenting symptoms in various medical conditions may affect individuals' body image in various patient populations (e.g., Bullen *et al.*, 2012; Ennis *et al.*, 2012; Fingeret *et al.*, 2013; Rhondali *et al.*, 2013). Of note, treatment and improvement in disease activity are not always associated with improvements in body image (Saha *et al.*, 2015). In addition, treatment for medical conditions may worsen body image—for example, increased body dissatisfaction has been reported after mastectomy in breast cancer patients (Lasry *et al.*, 1987). On the other hand, some medical treatments have been shown to reduce body image concerns more so than others (e.g., ileal pouch–anal anastomosis in ulcerative colitis patients; Larson *et al.*, 2008; and robotic thyroidectomy among patients with papillary thyroid carcinoma; Lee *et al.*, 2014).

Risk Factors for Body Image Concerns

A host of risk factors for body image concerns has been identified. Demographic, sociocultural, and psychosocial variables are among the most widely studied and will be the focus of this section of the chapter. First, demographic characteristics including gender, race/ethnicity, and sexual orientation are associated with body image concerns. Women, Caucasian individuals,

and men who identify as gay or bisexual are generally more at risk for body image concerns; however, differences in these groups appear to be narrowing in some respects (Field et al., 2014; Morrison, Morrison, & Sager, 2004; Roberts, Cash, Feingold, & Johnson, 2006). Second, numerous sociocultural variables (i.e., characteristics of society that includes cultural norms for beauty and attractiveness) have been linked to body image concerns. For instance, greater media consumption and exposure (e.g., magazines, television, social media, Internet-based media) has been found to be associated with increased body image concerns (Grabe, Ward, & Hyde, 2008). Relatedly, internalization of societal norms regarding thinness and perceived sociocultural pressure for thinness are strongly related to increased body image concerns (Stice & Whitenton, 2002). Further, social comparisons, which involve tendencies to compare oneself with other people, are also highly predictive of body image concerns, especially in response to comparisons in which the other person is perceived as more attractive (Myers & Crowther, 2009).

Various psychosocial aspects also have been found to be related to elevated body image concerns. For example, certain personality characteristics are related to poor body image, including neuroticism and other forms of trait negative affectivity as well as perfectionism (Pennesi & Wade, 2016). Lifetime and recent stressful events including weight teasing and discrimination are also related to body image concerns (Eisenberg, Neumark-Sztainer, Haines, & Wall, 2006). Furthermore, greater internalizing problems, notably anxiety, depression, and low self-esteem, are related to increased body image concerns (Patalay et al., 2015; van den Berg, Mond, Eisenberg, Ackard, & Neumark-Sztainer, 2010). Finally, interpersonal and social deficits including attachment insecurity (Abbate-Daga, Gramaglia, Amianto, Marzola, & Fassino, 2010), poor peer relationships (Schutz & Paxton, 2007), and decreased social support (Stice & Whitenton, 2002) are also associated with increased body image concerns.

Correlates and Consequences of Body Image Concerns

Studies of clinical populations have primarily shown body image concerns to be related to reduced disease-specific and overall quality of life, as well overall psychological well-being (Rhondali et al., 2013; Yagil et al., 2015). Furthermore, Bullen et al. (2012) reported that preexisting disturbances in body image predicted later psychopathology in patients with colorectal disease. Thus, body image concerns related to disease and medical problems, as well as preexisting body image concerns, may impact mental and physical health outcomes. Additionally, in the broader body image literature, poor body image has been found to be related to numerous domains of mental and physical health, maladaptive behavior, and overall functioning. Below, we briefly review relationships between body image and each of these domains.

Psychological Functioning and Mental Health

Poor body image is broadly associated with poorer overall mental health (Mond et al., 2013), as well as lower self-esteem and increased internalizing symptoms among men and women (Field et al., 2014; Patalay et al., 2015). In addition, body image concerns are related to interpersonal and social problems and difficulties (Gupta & Gupta, 2013; Mond et al., 2013). Other research has also found increased body image concerns to be associated with suicidal ideation (Gupta & Gupta, 2013) and sleep problems (Gupta, Gupta, & Knapp, 2015).

Eating- and Weight-Related Behaviors

Body image concerns are related to a number of eating- and weight-related behaviors, including dietary restriction, use of diet pills, diuretics, and laxatives, and self-induced vomiting (Andrew, Tiggemann, & Clark, 2016; Neumark-Sztainer et al., 2006). Additionally, among men in particular, body image concerns in the form of a desire for greater muscularity are associated with use of steroids and other appearance- and performance-enhancing drugs (Murray, Griffiths, Mond, Kean, & Blashill, 2016). In turn, maladaptive eating patterns, such as binge eating and overeating, have been shown to be related to increased body image concerns (Neumark-Sztainer et al., 2006).

Psychiatric Conditions

In addition to being broadly associated with eating- and weight-control behaviors, body image concerns are a central feature in certain psychiatric disorders and commonly occur in others. For example, the experience of persistent, intrusive thoughts about one or more perceived flaws in one's appearance is the hallmark feature of body dysmorphic disorder. In addition, body image concerns are most notably associated with eating disorders and are included in nearly all models of disordered eating (Pennesi & Wade, 2016). Specifically, the overvaluation of body shape and weight is a defining characteristic of anorexia nervosa (AN) and bulimia nervosa (BN) and is also commonly observed in subclinical eating disorders and binge eating disorder (BED), the latter of which is characterized by a high prevalence of overweight and obesity.

Other Health Behaviors

While research has typically assessed relationships between body image concerns and eating- and weight-related behaviors, there is evidence that body image concerns are associated with other health-related behaviors as well. For example, studies have found associations between lower body satisfaction and lower physical activity, increased screen-based media use, and increased smoking among males and females (Farhat, Iannotti, & Caccavale, 2014; Neumark-Sztainer et al., 2006). In addition, in an online sample of women, body dissatisfaction was related to less skin screening behaviors and more alcohol consumption, whereas body appreciation was related to more sun protection behaviors (Andrew et al., 2016). Among males, elevated concerns over muscularity among males have been associated with a number of problematic behaviors, including binge drinking and drug use (Field et al., 2014; Jampel, Safren, & Blashill, 2015). Finally, one study of body image concerns and physical health found that chronic weight dissatisfaction predicted elevated risk for type 2 diabetes, over and above weight status (Wirth, Blake, Hébert, Sui, & Blair, 2014).

Sexual Health

Body image concerns have also been shown to be related to multiple domains of sexual functioning (Woertman & van den Brink, 2012). For example, women with body image concerns report more appearance concerns during sexual interactions, lower levels of desire and arousal, and decreased pleasure and sexual satisfaction (Woertman & van den Brink, 2012). In addition, a recent meta-analysis showed that increased body dissatisfaction was related to less condom use self-efficacy (Blashill & Safren, 2015). Further, body image concerns

were associated with risky sexual behavior among women (Merianos, King, & Vidourek, 2013). Conversely, body appreciation was related to better sexual functioning among women (Satinsky, Reece, Dennis, Sanders, & Bardzell, 2012).

Conclusions

Body image concerns are prominent in numerous clinical populations, both medical and psychiatric. Body image concerns are generally associated with reduced quality of life in patients and may lead to the development of additional mental and physical health problems. In addition, poor body image may be associated with engaging in numerous maladaptive behaviors including unhealthy eating- and weight-related behaviors, alcohol and drug use, and risky sexual behaviors. Poor body image may also be associated with less positive health behaviors, notably cancer screening behaviors (Andrew et al., 2016) and reduced physical activity. Due to the wide range of negative outcomes related to body image concerns, screening for body image concerns in clinical health settings is imperative in order to provide optimal medical care and reduce disease burden.

Author Biographies

Tyler B. Mason, PhD, is an assistant professor in the Department of Preventative Medicine at the University of Southern California. He received his PhD in applied psychological science in 2015 and BS in psychology in 2010 from Old Dominion University. His primary research interests include etiology and treatment of binge eating and obesity.

Kathryn E. Smith, PhD, is a T32 postdoctoral research fellow at the Neuropsychiatric Research Institute. She received her BA in psychology from Macalester College and her PhD in clinical psychology from Kent State University. Her primary interests include emotion regulation and co-occurring psychopathology in eating disorders and obesity.

Jason M. Lavender, PhD, is an assistant research scientist in the Department of Psychiatry at the University of California, San Diego. He completed his undergraduate education at Duke University and his PhD in clinical psychology at the University at Albany, SUNY. His research addresses emotion, neurocognition, and personality in eating disorders.

Stephen A. Wonderlich, PhD, is the Chester Fritz distinguished professor and associate chairperson in the Department of Psychiatry and Behavioral Science at the University of North Dakota, chair of eating disorders and co-director of the Eating Disorder and Weight Management Center at Sanford, and president and scientific director of the Neuropsychiatric Research Institute.

References

- Abbate-Daga, G., Gramaglia, C., Amianto, F., Marzola, E., & Fassino, S. (2010). Attachment insecurity, personality, and body dissatisfaction in eating disorders. *The Journal of Nervous and Mental Disease*, 198, 520–524.
- Andrew, R., Tiggemann, M., & Clark, L. (2016). Positive body image and young women's health: Implications for sun protection, cancer screening, weight loss and alcohol consumption behaviours. *Journal of Health Psychology*, 21, 28–39.

- Blashill, A. J., & Safren, S. A. (2015). Body dissatisfaction and condom use self-efficacy: A meta-analysis. *Body Image, 12*, 73–77. doi:10.1016/j.bodyim.2014.10.002
- Bullen, T. L., Sharpe, L., Lawsin, C., Patel, D. C., Clarke, S., & Bokey, L. (2012). Body image as a predictor of psychopathology in surgical patients with colorectal disease. *Journal of Psychosomatic Research, 73*, 459–463. doi:10.1016/j.jpsychores.2012.08.010
- Eisenberg, M. E., Neumark-Sztainer, D., Haines, J., & Wall, M. (2006). Weight-teasing and emotional well-being in adolescents: Longitudinal findings from Project EAT. *Journal of Adolescent Health, 38*, 675–683. doi:10.1016/j.jadohealth.2005.07.002
- Ennis, H., Herrick, A. L., Cassidy, C., Griffiths, C. E., & Richards, H. L. (2012). A pilot study of body image dissatisfaction and the psychological impact of systemic sclerosis-related telangiectases. *Clinical and Experimental Rheumatology, 31*, 12–17.
- Farhat, T., Iannotti, R. J., & Caccavale, L. J. (2014). Adolescent overweight, obesity and chronic disease-related health practices: Mediation by body image. *Obesity Facts, 7*, 1–14.
- Field, A. E., Sonnevile, K. R., Crosby, R. D., Swanson, S. A., Eddy, K. T., Camargo, C. A., ... Micali, N. (2014). Prospective associations of concerns about physique and the development of obesity, binge drinking, and drug use among adolescent boys and young adult men. *JAMA Pediatrics, 168*, 34–39. doi:10.1001/jamapediatrics.2013.2915
- Fingeret, M. C., Hutcheson, K. A., Jensen, K., Yuan, Y., Urbauer, D., & Lewin, J. S. (2013). Associations among speech, eating, and body image concerns for surgical patients with head and neck cancer. *Head & Neck, 35*, 354–360. doi:10.1002/hed.22980
- Fiske, L., Fallon, E. A., Blissmer, B., & Redding, C. A. (2014). Prevalence of body dissatisfaction among United States adults: Review and recommendations for future research. *Eating Behaviors, 15*, 357–365. doi:10.1016/j.eatbeh.2014.04.010
- Gillen, M. M. (2015). Associations between positive body image and indicators of men's and women's mental and physical health. *Body Image, 13*, 67–74.
- Grabe, S., Ward, L. M., & Hyde, J. S. (2008). The role of the media in body image concerns among women: A meta-analysis of experimental and correlational studies. *Psychological Bulletin, 134*, 460–476. doi:10.1037/0033-2909.134.3.460
- Gupta, M. A., & Gupta, A. K. (2013). Cutaneous body image dissatisfaction and suicidal ideation: Mediation by interpersonal sensitivity. *Journal of Psychosomatic Research, 75*, 55–59.
- Gupta, M. A., Gupta, A. K., & Knapp, K. (2015). Dissatisfaction with cutaneous body image is directly correlated with insomnia severity: A prospective study in a non-clinical sample. *Journal of Dermatological Treatment, 26*, 193–197.
- Hawighorst-Knapstein, S., Fusshoeller, C., Franz, C., Trautmann, K., Schmidt, M., Pilch, H., ... Vaupel, P. (2004). The impact of treatment for genital cancer on quality of life and body image—Results of a prospective longitudinal 10-year study. *Gynecologic Oncology, 94*, 398–403. doi:10.1016/j.ygyno.2004.04.025
- Jampel, J. D., Safren, S. A., & Blashill, A. J. (2015). Muscularity disturbance and methamphetamine use among HIV-infected men who have sex with men. *Psychology of Men & Masculinity, 16*, 474.
- Jolly, M., Pickard, A. S., Mikolaitis, R. A., Cornejo, J., Sequeira, W., Cash, T. F., & Block, J. A. (2012). Body image in patients with systemic lupus erythematosus. *International Journal of Behavioral Medicine, 19*, 157–164. doi:10.1007/s12529-011-9154-9
- Larson, D. W., Davies, M. M., Dozois, E. J., Cima, R. R., Piotrowicz, K., Anderson, K., ... Pemberton, J. H. (2008). Sexual function, body image, and quality of life after laparoscopic and open ileal pouch-anal anastomosis. *Diseases of the Colon & Rectum, 51*, 392–396. doi:10.1007/s10350-007-9180-5
- Lasry, J. C. M., Margolese, R. G., Poisson, R., Shibata, H., Fleischer, D., Lafleur, D., ... Taillefer, S. (1987). Depression and body image following mastectomy and lumpectomy. *Journal of Chronic Diseases, 40*, 529–534.
- Lee, S., Kim, H. Y., Lee, C. R., Park, S., Son, H., Kang, S. W., ... Park, C. S. (2014). A prospective comparison of patient body image after robotic thyroidectomy and conventional open thyroidectomy in patients with papillary thyroid carcinoma. *Surgery, 156*, 117–125.

- Melis, I., Litta, P., Nappi, L., Agus, M., Melis, G. B., & Angioni, S. (2015). Sexual function in women with deep endometriosis: Correlation with quality of life, intensity of pain, depression, anxiety, and body image. *International Journal of Sexual Health*, 27, 175–185. doi:10.1080/19317611.2014.952394
- Merianos, A. L., King, K. A., & Vidourek, R. A. (2013). Body image satisfaction and involvement in risky sexual behaviors among university students. *Sexuality & Culture*, 17, 617–630. doi:10.1007/s12119-013-9165-6
- Mond, J., Mitchison, D., Latner, J., Hay, P., Owen, C., & Rodgers, B. (2013). Quality of life impairment associated with body dissatisfaction in a general population sample of women. *BMC Public Health*, 13, 920. doi:10.1186/1471-2458-13-920
- Morrison, M. A., Morrison, T. G., & Sager, C. L. (2004). Does body satisfaction differ between gay men and lesbian women and heterosexual men and women?: A meta-analytic review. *Body Image*, 1, 127–138. doi:10.1016/j.bodyim.2004.01.002
- Münstedt, K., Manthey, N., Sachsse, S., & Vahrson, H. (1997). Changes in self-concept and body image during alopecia induced cancer chemotherapy. *Supportive Care in Cancer*, 5, 139–143.
- Murray, S. B., Griffiths, S., Mond, J. M., Kean, J., & Blashill, A. J. (2016). Anabolic steroid use and body image psychopathology in men: Delineating between appearance-versus performance-driven motivations. *Drug and Alcohol Dependence*, 165, 198–202.
- Myers, T. A., & Crowther, J. H. (2009). Social comparison as a predictor of body dissatisfaction: A meta-analytic review. *Journal of Abnormal Psychology*, 118, 683–698.
- Neumark-Sztainer, D., Paxton, S. J., Hannan, P. J., Haines, J., & Story, M. (2006). Does body satisfaction matter? Five-year longitudinal associations between body satisfaction and health behaviors in adolescent females and males. *Journal of Adolescent Health*, 39, 244–251. doi:10.1016/j.jadohealth.2005.12.001
- Patalay, P., Sharpe, H., & Wolpert, M. (2015). Internalising symptoms and body dissatisfaction: Untangling temporal precedence using cross-lagged models in two cohorts. *Journal of Child Psychology and Psychiatry*, 56, 1223–1230. doi:10.1111/jcpp.12415
- Pennesi, J. L., & Wade, T. D. (2016). A systematic review of the existing models of disordered eating: Do they inform the development of effective interventions? *Clinical Psychology Review*, 43, 175–192. doi:10.1016/j.cpr.2015.12.004
- Pinquart, M. (2013). Body image of children and adolescents with chronic illness: A meta-analytic comparison with healthy peers. *Body Image*, 10, 141–148.
- Pruzinsky, T. (2004). Enhancing quality of life in medical populations: A vision for body image assessment and rehabilitation as standards of care. *Body Image*, 1, 71–81.
- Puhl, R. M., & Latner, J. D. (2007). Stigma, obesity, and the health of the nation's children. *Psychological Bulletin*, 133, 557–580.
- Rhondali, W., Chisholm, G. B., Daneshmand, M., Allo, J., Kang, D. H., Filbet, M., ... Bruera, E. (2013). Association between body image dissatisfaction and weight loss among patients with advanced cancer and their caregivers: A preliminary report. *Journal of Pain and Symptom Management*, 45, 1039–1049. doi:10.1016/j.jpainsymman.2012.06.013
- Roberts, A., Cash, T. F., Feingold, A., & Johnson, B. T. (2006). Are black-white differences in females' body dissatisfaction decreasing? A meta-analytic review. *Journal of Consulting and Clinical Psychology*, 74, 1121–1131. doi:10.1037/0022-006X.74.6.1121
- Saha, S., Zhao, Y. Q., Shah, S. A., Degli Esposti, S., Lidofsky, S., Shapiro, J., ... Samad, Z. (2015). Body image dissatisfaction in patients with inflammatory bowel disease. *Inflammatory Bowel Diseases*, 21, 345–352. doi:10.1097/MIB.0000000000000270
- Sarwer, D. B., & Steffen, K. J. (2015). Quality of life, body image and sexual functioning in bariatric surgery patients. *European Eating Disorders Review*, 23, 504–508.
- Satinsky, S., Reece, M., Dennis, B., Sanders, S., & Bardzell, S. (2012). An assessment of body appreciation and its relationship to sexual function in women. *Body Image*, 9, 137–144.
- Schutz, H. K., & Paxton, S. J. (2007). Friendship quality, body dissatisfaction, dieting and disordered eating in adolescent girls. *British Journal of Clinical Psychology*, 46, 67–83.

- Song, A. Y., Rubin, J. P., Thomas, V., Dudas, J. R., Marra, K. G., & Fernstrom, M. H. (2006). Body image and quality of life in post massive weight loss body contouring patients. *Obesity, 14*, 1626–1636. doi:10.1038/oby.2006.187
- Stice, E., & Whitenton, K. (2002). Risk factors for body dissatisfaction in adolescent girls: A longitudinal investigation. *Developmental Psychology, 38*, 669–678.
- van den Berg, P. A., Mond, J., Eisenberg, M., Ackard, D., & Neumark-Sztainer, D. (2010). The link between body dissatisfaction and self-esteem in adolescents: Similarities across gender, age, weight status, race/ethnicity, and socioeconomic status. *Journal of Adolescent Health, 47*, 290–296. doi:10.1016/j.jadohealth.2010.02.004
- Wirth, M. D., Blake, C. E., Hébert, J. R., Sui, X., & Blair, S. N. (2014). Chronic weight dissatisfaction predicts type 2 diabetes risk: Aerobic center longitudinal study. *Health Psychology, 33*, 912–919. doi:10.1037/hea0000058
- Woertman, L., & van den Brink, F. (2012). Body image and female sexual functioning and behavior: A review. *Journal of Sex Research, 49*, 184–211.
- Yagil, Y., Geller, S., Sidi, Y., Tirosh, Y., Katz, P., & Nakache, R. (2015). The implications of body-image dissatisfaction among kidney-transplant recipients. *Psychology, Health & Medicine, 20*, 955–962.

Suggested Reading

- Grogan, S. (2006). Body image and health contemporary perspectives. *Journal of Health Psychology, 11*, 523–530. doi:10.1177/1359105306065013
- Pruzinsky, T. (2004). Enhancing quality of life in medical populations: A vision for body image assessment and rehabilitation as standards of care. *Body Image, 1*, 71–81.

Caregivers and Clinical Health Psychology

Allison Marziliano and Anne Moyer

Department of Psychology, Stony Brook University, Stony Brook, NY, USA

An informal caregiver is defined as “any relative, partner, friend, or neighbor who has a significant personal relationship with, and provides a broad range of assistance for an older person or an adult with a chronic or disabling condition” (Family Caregiver Alliance, 2016). The American Association of Retired Persons (AARP) and the National Alliance for Caregiving (NAC), a nonprofit coalition of national organizations, collaborate every 5 years to produce a research report on unpaid family caregivers in the United States, the most recent of which was published in June of 2015 (National Alliance for Caregiving, 2016a, 2016b). According to this publication, Caregiving in the U.S. 2015, it is estimated that 34.2 million adults in the United States have provided care to another adult or adults over the age of 50 years old within the last 12 months, representing approximately 14.3% of the American adult population. The majority of caregivers are relatives of the care recipient (85%) and female (60%), and most (82%) care for one other individual rather than multiple individuals. The average age of caregivers in general is 49.2 years, compared with the average age when limiting the sample to spousal caregivers, 62.3 years old.

In addition to providing useful information about caregivers, the research report, Caregiving in the U.S. 2015, also provides descriptions of the individuals for whom they provide care, or care recipients (National Alliance for Caregiving, 2016a, 2016b). Care recipients are often older individuals with functional disabilities who had a prior relationship with the person who has come to assume the role as their unpaid caregiver. Nearly half (47%) of care recipients live in their own homes, about 1/3 (35%) live with their caregivers, and 18% reside in long-term care or senior housing. The average age of those individuals receiving care is 69.4 years old, with nearly half (47%) of care recipients above the age of 74 years old.

Caregiver Burden

Caregiver burden is multifaceted. In a systematic review of the literature on assessment tools to measure the impact of family caregiving, caregiver burden is described as a negative response to the impact of providing care on the caregiver's physical, psychological, emotional, social, and financial condition. Burden is often the result of the caregivers' belief that demands placed upon them surpass their own physical or mental capacity.

Some research has addressed the financial burden associated with caregiving. Providing care to a loved one is costly in and of itself; however, caregiving may interfere with time previously devoted to employment for both the patient and caregiver, which may lead to reductions in the flow of income, further compounding financial strain. Caregivers often report less time for themselves and competing financial demands as a result of their role. In one study, schedule burden was cited as the most prevalent form of burden experienced by caregivers, reported by 58% of end-of-life caregivers and 32% of caregivers of patients with chronic illness (Sautter et al., 2014). In one study, 60–80% of caregivers of patients with Parkinson's disease reported disruptions in their financial status (Sanyal et al., 2015). In a sample of caregivers of patients with head and neck cancer, financial stress was significantly related to high levels of unmet needs (Balfe et al., 2016). In caregivers of individuals suffering from schizophrenia, only approximately 1/3 (31.9%) endorsed "agree" or "strongly agree" when responding to whether they believed they have the financial capacity to provide adequate care to their loved one (Gupta, Isherwood, Jones, & Van Impe, 2015).

Particular attention has been devoted to financial burden in the context of female caregivers. Research using data from 2,093 women over the age of 52 with at least one living parent or parent-in-law participating in the Health and Retirement Surveys in 2006, 2008, and 2010 supported a reciprocal relationship between parental caregiving and lower household income (Lee, Tang, Kim, & Albert, 2014). In a study of female caregivers of patients with Alzheimer's disease, the authors suggested that the greatest financial challenge was the high cost of the care they deliver, with females bearing six times the cost of care than men do (Yang & Levey, 2015).

In addition to financial burden, physical and health-related burden has been reported by caregivers. In a cross-sectional study of family caregivers of individuals with schizophrenia, approximately 2/3 of the sample (65.5%) believed themselves to be in poor physical health (Thunyadee, Sitthimongkol, Sangon, Chai-Aroon, & Hegadoren, 2015). Not only do caregivers often perceive themselves as being in ill-health, but also there is some research to suggest that their health is indeed affected by their caregiving role. Spousal caregivers are at a 600% increased risk of developing dementia (Wennberg, Dye, Streetman-Loy, & Pham, 2015), and caregivers more generally are at risk of experiencing sleep trouble, fatigue, insomnia, weight gain, pain, headaches, and heartburn (Gupta et al., 2015; Skalla, Lavoie Smith, Li, & Gates, 2013).

Research has identified correlates and predictors of burden in caregivers. A large body of literature suggests that caregiver burden is related to certain patient-related factors such as higher levels of patient cognitive impairment (Corallo et al., 2016; Vaingankar et al., 2016) and patient motor disability (Sanyal et al., 2015). Other correlates and predictors of caregiver burden include less use of hospice care, female gender, student status, spousal relationship to care recipient, the use of emotion-focused coping strategies, duration of caregiving, hours spent caregiving, social isolation, and lack of choice in assuming the caregiving role (Adelman, Tmanova, Delgado, Dion, & Lachs, 2014; Duggleby et al., 2016; Gupta et al., 2015; Sanyal et al., 2015; Thunyadee et al., 2015).

Negative Psychosocial Sequelae

Although caregiver burden is not a classifiable syndrome, such burdens can result in clinical diagnoses such as depression and anxiety and even lead to suicide (Adelman et al., 2014). The prevalence of depressive disorders has been found to be as high as 22% among caregivers of dementia patients (Cuipers, 2005). The prevalence of depressive symptoms for caregivers of critically ill patients is similar but is reported to be even higher during the critical care period (Haines, Denehy, Skinner, Warrillow, & Berney, 2015). Although the evidence regarding this is more sparse, clinically significant anxiety is estimated to occur in about a quarter of caregivers for people with dementia (Cooper, Balamurali, & Livingston, 2007). In the context of life-threatening diseases such as cancer, one of the most common physical conditions for which patients receive informal caregiving (Girgis, Lambert, Johnson, Waller, & Currow, 2013), a reciprocal relationship has been found between caregiver and patient distress (Northouse, Katapodi, Schafenacker, & Weiss, 2012). In addition, cancer diagnosis and treatment follow a dynamic course; as a cancer patient's condition declines or nears death, caregivers' depression, anxiety, and distress worsen, and a caregiver's depression and anxiety may also have consequences for the patient (Williams & McCorkle, 2011). Estimates of the prevalence of anxiety in partners and caregivers of people with cancer have been found to range from 16 to 56%, and even PTSD has been documented in 4% (Girgis et al., 2013).

Positive Psychosocial Sequelae

The picture is not all bleak, however. Evidence suggests that the capacity exists for caregivers to experience, as a result of their role, positive psychosocial sequelae (Moore et al., 2011), such as posttraumatic growth (PTG), the cognitive process through which those who are faced with a traumatic event positively reinterpret and find meaning in the event (Tedeschi & Calhoun, 1996), and benefit finding (BF), one's perception that positive changes have occurred as a result of experiencing difficult life events. Furthermore, in some studies, caregivers score higher in certain facets of PTG, such as personal strength, than the patients for whom they provide care (Cormio et al., 2014). Research has identified common correlates of increased caregiver PTG and benefit finding as increased social support, self-affirmation, having a positive outlook on life, positive affect, and the use of positive religious coping strategies (Balfe et al., 2016; Thombre, Sherman, & Simonton, 2010). Some research has focused on the relationship between caregiver PTG and patient-related factors, indicating a positive relationship between cancer caregivers' report of patients' quality of life at end of life and caregiver PTG (Hatano, Fujimoto, Hosokawa, & Fukui, 2015). While BF is an important construct in and of itself, its importance also lays in that some research has found caregiver BF to positively predict caregiver quality of life (Navarta-Sanchez et al., 2016).

Albeit less of a focus than PTG and BF, other research has examined the positive constructs of resilience and accomplishment in relation to caregiving. In a study of caregivers of children with cancer, correlates of parent resilience include coping strategies, social support, positive provider interactions, and lack of psychosocial distress (Rosenberg, Baker, Syrjala, Back, & Wolfe, 2013). In another study of caregivers of individual with multiple sclerosis (MS), positive perception of accomplishment was related to older age of the patient and greater number of

hours per week the caregiver provided care, while negative perception of accomplishment was related to caregiver perception that caregiving is an emotionally draining role, higher levels of caregiver education, and a spousal relationship between patient and caregiver (Buchanan & Huang, 2012). Although a great deal of literature has demonstrated the negative effects of caregiving, fortunately, recent research has focused on routes to amplifying the positive psychosocial sequelae associated with caring for a loved one.

Support for Caregivers

The challenges associated with caregiving mean that assistance, including finding ways to take care of themselves, is important for caregivers. These types of assistance include support in the legal, financial, emotional, social, educational, physical, and mental health areas of functioning. National organizations provide supportive resources in the form of information and education for both caregivers and healthcare and policy professionals, online and in-person support groups, first-person stories, connections to clinical trials and research studies, and advocacy. For instance, the Family Caregiver Alliance provides a national registry to assist caregivers of people who are living at home or in a residential facility to locate local public, nonprofit, and private programs, such as government health and disability programs, legal resources, and disease-specific organizations (Family Caregiver Alliance, 2016). Examples of local resources include information, assistance, individual counseling, support groups, caregiver training, respite care, and supplemental services, such as transportation and home modifications (New York State Office of the Aging, 2016). The NAC conducts authoritative research on trends in the needs of caregivers with a focus on reaching a broad, racially and ethnically diverse population sample and works to increase public awareness of family caregiving issues (National Alliance for Caregiving, 2016a, 2016b).

Practical and tangible forms of support such as respite, or time away from the duties of caregiving, are considered particularly valuable by caregivers, although the influence of social norms (e.g., related to a sense of familial duty) may make caregivers reluctant to let go of their caregiving responsibilities. Making use of respite services can allow caregivers to continue their employment or education, social activities, or hobbies integral to their identity; engage in self-care activities like recreation, exercise, and good nutrition; and seek legal and financial counsel. To be effective, however, respite needs to be regular or substantial (i.e., at a frequency of at least twice a week) and involves large blocks of time. In response to evidence that caregivers often feel their respite time goes to waste or is unfulfilling, frustrating, or even increased their stress, some research has begun to investigate how to improve the ways that respite time can best meet individual needs, through goal setting that reflects personal priorities and forming strategies to optimize goal attainment (Lund et al., 2014). Practical ways that healthcare providers can support the caregivers of the patients they treat are to assess what types of difficulty they might be having, encourage them to engage in self-care, provide information about their care recipients' illness, and provide referrals to assistance available to them (Adelman et al., 2014).

Technological forms of assistance can also be useful. These include emergency response systems, webcams or mobility trackers for monitoring, lifts, medication reminder systems, and socialization via Skype (Adelman et al., 2014). Education and counseling can be readily provided by web- and video-based platforms, and decision-making support can be facilitated via telephone and videoconferencing.

Psychosocial Interventions for Caregivers

A number of interventions have been developed for caregivers of individuals with dementia. A large ($N = 1,222$) multisite study (the Resources for Enhancing Alzheimer's Caregiver Health [REACH] initiative) tested a group of site-specific, theory-driven interventions that were tailored for the ethnic groups served (Wisniewski et al., 2003). The interventions were diverse and based on different theoretical frameworks, but they were similar in being based on a basic stress–health model that addressed stressors themselves, their appraisal, and/or caregivers' response to them. These included *care-recipient-focused behavior management skill training and caregiver-focused, problem-solving training* (involving didactic instruction, role-playing, modeling, feedback, manuals, and videotapes, home visits, and therapeutic telephone calls), a 24-hr/day *telephone-linked computer intervention* (involving a voicemail support network, information on specific care recipients' behavioral problems, access to a geriatric nurse specialist, and providing distracting conversation), *information and referral* delivered within primary care clinics (involving commercially available pamphlets regarding dementia), *behavioral care* (involving targeted one-on-one information and counseling sessions related to care recipients' behavioral problems supplemented by telephone calls), *enhanced care* (involving specific cognitive and behavioral strategies to reduce caregiver distress taught in person and supported by telephone calls), a *family-based structural multisystem in-home intervention* (involving in-home family therapy sessions to enhance family functioning and reduce caregiver distress) with and without a computerized telephone integration system, a *coping with caregiving class* (involving mood-management skills to reduce stress and distress), an *enhanced support group* (involving professional leadership and a commitment to meet regularly for 1 year), and an *environmental skill building program* (involving in-home individualized environmental strategies delivered by an occupational therapist). Across all interventions, burden and depression scores were lower for active conditions compared with control conditions at 6 months, but this was only statistically significant for burden and the effect was small (Gitlin et al., 2003). Nonetheless, the interventions were rated as beneficial, helpful, and valuable by participants, and there were significant subgroup effects for those who were female and had lower education for burden and for those who were Hispanic, non-spouses, and had lower education for depression (Gitlin et al., 2003).

As alluded to by the design of some of the interventions in the REACH study, because there is a potentially synergistic relationship between the behavior of the person with dementia and their informal caregiver's level of burden and the strategies they use to manage the behavior of the person with dementia, interventions are often directed at both individuals in the dyad. For instance, for the person with dementia, such things as behavioral problems, cognitive functioning, mood, independence in daily activities, sleep, and quality of life may be the focus, and for the caregiver, mood, burden, competence, and quality of life may be the focus. These dyadic interventions include components such as information, training in daily life activities, walking or exercise, and adaptations to the physical environment for the person with dementia and information, psychoeducation, skills training, and coping strategies for the caregiver. A systematic review of 20 of these programs showed them to be promising in terms of effectiveness for one or both members of the dyad (Van't Leven et al., 2013).

An approach that differs from psychosocial interventions is meditative-based interventions that involve mindfulness or concentration-based techniques, some of which are designed to be undertaken at home. A systematic review of eight studies suggests that they hold some promise in alleviating burden and depression in dementia caregivers (Hurley, Patterson, & Cooley,

2014). Interventions involving relaxation and yoga have been found to be most likely to be effective specifically for anxiety in caregivers, an often untargeted outcome (Cooper, Balamurali, Selwood, & Livingston, 2007).

Barriers to Utilizing Psychosocial Interventions

There is evidence to suggest that family caregivers underutilize the mental health services and interventions that may be available to them. In one study (Mosher et al., 2013) of caregivers reporting anxiety and depressive symptoms, only a quarter of participants reported using mental health services, and 39% reported using complementary and alternative medicine in the 3-month period in which the research took place. These findings are in contrast to those who expressed an interest in support services; 29% of caregivers who did not receive mental health services desired them. Thus, these findings suggest that it may not be a lack of empirically validated interventions for caregivers, but rather the presence of barriers that inhibits caregivers from seeking the psychosocial support they need.

A commonly reported barrier to seeking mental health services for both caregivers and patients alike is the fear of stigmatization (Gulliver, Griffiths, & Christensen, 2010). In one study, caregivers of individuals with mental health illnesses indicated their concerns for their loved one seeking treatment as hindering their employment opportunities, being viewed as weak or crazy, being embarrassed or ashamed, or being unable to afford services (Dockery et al., 2015). Correlates of reporting stigma related to patients' seeking of mental health services included being a female caregiver and being an African American caregiver. Caregivers also cite stigma as a concern preventing them from seeking their own mental health support services (Boots, Wolfs, Verhey, Kempen, & de Vugt, 2015), which may negatively affect their capability to care for not only themselves but also the care recipient.

In addition to stigma, research on caregivers has identified other barriers preventing them from utilizing mental health services and interventions. Commonly cited barriers reported by caregivers of patients with various diagnoses also include struggling with acknowledging their own needs, low acceptance of their new role, negative view of mental health professionals, desire for independent management of their issues, perceived conflict between their role as a caregiver and obtaining treatment for themselves, anticipated negative self-perception, feeling unequipped to manage their situation, and reluctance to allow help (Boots et al., 2015; Mosher et al., 2013). It is critical to consider these barriers during intervention design in order to maximize the efficacy of mental health treatment for caregivers.

Post-caregiving

Some research refers to the period following the cessation of care, post-caregiving, as a stage well within the caregiving life course that requires, but often lacks, unique attention (Hash, 2006). Research demonstrates the potential for cognitive and psychological changes that may begin during caregiving but last into the post-caregiving stage. For example, in a study of caregivers of palliative care patients who completed neuropsychological assessments as caregivers and at least 6 months from the death of the care recipient, results showed an improvement in self-monitoring and regulating attentional resources and a decline in spatial working memory and delayed episodic memory (Mackenzie, Smith, Hasher, Leach, & Behl, 2007). In another study, individuals who care or cared for chronically ill same-sex partners commonly

reported loneliness and depression in the post-caregiving period (Hash, 2006). During this stage, then, caregivers continuously encounter difficult transitions such as coping with grief and mourning, rebuilding, or returning to their pre-caregiving life.

Conclusion

In sum, informal caregivers report financial, schedule, and health-related burden, as well as experience depressive disorders, anxiety, and distress. Fortunately, research on growth and finding benefit in the caregiving experience is increasing and demonstrates the potential for positive psychosocial sequelae associated with this role. Although support for caregivers comes in various forms, from promotion of respite to psychosocial and meditative interventions, research suggests that mental health services and interventions are underutilized due to specific barriers such as fear of stigmatization. Future research would be wise to focus on overcoming the barriers to accessing mental health services that caregivers often face and providing such support into the post-caregiving period.

Author Biographies

Allison Marziliano is a doctoral candidate in social and health psychology at Stony Brook University and a predoctoral fellow in the Department of Psychiatry and Behavioral Sciences at Memorial Sloan Kettering Cancer Center. Her research interests include the study of psychosocial issues surrounding cancer patients, their families, and their caregivers, as well as grief and bereavement.

Anne Moyer is an associate professor of social and health psychology at Stony Brook University. Her research focuses on psychosocial factors surrounding cancer and cancer risk, medical decision making, research methodology and research synthesis, and the psychological aspects of human research participation.

References

- Adelman, R. D., Tmanova, L. L., Delgado, D., Dion, S., & Lachs, M. S. (2014). Caregiver burden: A clinical review. *JAMA*, *113*, 1052–1059.
- Balfé, M., O, Brien, K., Timmons, A., Butow, P., O, Sullivan, E., Gooberman-Hill, R., & Sharp, L. (2016). What factors are associated with posttraumatic growth in head and neck cancer carers? *European Journal of Oncology Nursing*, *21*, 31–37.
- Boots, L. M., Wolfs, C. A., Verhey, F. R., Kempen, G. I., & de Vugt, M. E. (2015). Qualitative study on needs and wishes of early-stage dementia caregivers: The paradox between needing and accepting help. *International Psychogeriatrics*, *27*, 927–936.
- Buchanan, R. J., & Huang, C. (2012). Caregiver perceptions of accomplishment from assisting people with multiple sclerosis. *Disability and Rehabilitation*, *34*, 53–61.
- Cooper, C., Balamurali, T. B., & Livingston, G. (2007). A systematic review of the prevalence and covariates of anxiety in caregivers of people with dementia. *International Psychogeriatrics*, *19*, 175–195.
- Cooper, C., Balamurali, T. B., Selwood, A., & Livingston, G. (2007). A systematic review of intervention studies about anxiety in caregivers of people with dementia. *International Journal of Geriatric Psychiatry*, *22*, 181–188.

- Corallo, F., DeCola, M. C., Buono, V. L., Di Lorenzo, G., Bramanti, P., & Marino, S. (2016). Observational study of quality of life of Parkinson's patients and their caregivers. *Psychogeriatrics*, 17(2), 97–102.
- Cormio, C., Romito, F., Viscanti, G., Turaccio, M., Lorusso, V., & Mattioli, V. (2014). Psychological well-being and posttraumatic growth in caregivers of cancer patients. *Frontiers in Psychology*, 5, 1342–1350.
- Cuipers, P. (2005). Depressive disorders in caregivers of dementia patients: A systematic review. *Aging & Mental Health*, 9, 325–330.
- Dockery, L., Jeffery, D., Schauman, O., Williams, P., Farrelly, S., Bonnington, O., ... MIRIAD study group. (2015). Stigma- and non-stigma-related treatment barriers to mental healthcare reported by service users and caregivers. *Psychiatry Research*, 228, 612–619.
- Duggleby, W., Williams, A., Ghosh, S., Moquin, H., Ploeg, J., Markle-Reid, M., & Peacock, S. (2016). Factors influencing changes in health related quality of life of caregivers of persons with multiple chronic conditions. *Health and Quality of Life Outcomes*, 27, 81–90.
- Family Caregiver Alliance. (2016). Definitions. Retrieved from <https://www.caregiver.org/definitions-0>.
- Girgis, A., Lambert, S., Johnson, C., Waller, A., & Currow, D. (2013). Physical, psychosocial, relationship, and economic burden of caring for people with cancer: A review. *Journal of Oncology Practice*, 9, 197–202.
- Gitlin, L. N., Burgio, L. D., Mahoney, D., Burns, R., Zhang, S., Schulz, R., ... Ory, M. G. (2003). Effect of multicomponent interventions on caregiver burden and depression: The REACH multisite initiative at 6-month follow-up. *Psychology and Aging*, 18, 361–374.
- Gulliver, A., Griffiths, K. M., & Christensen, H. (2010). Perceived barriers and facilitators to mental health help seeking in young people: A systematic review. *BMC Psychiatry*, 10, 113–122.
- Gupta, S., Isherwood, G., Jones, K., & Van Impe, K. (2015). Assessing health status in informal schizophrenia caregivers compared with health status in non-caregivers and caregivers of other conditions. *BMC Psychiatry*, 15, 162–173.
- Haines, K. J., Denehy, L., Skinner, E. H., Warrillow, S., & Berney, S. (2015). Psychosocial outcomes in informal caregivers of the critically ill: A systematic review. *Critical Care in Medicine*, 43, 1112–1120.
- Hash, K. (2006). Caregiving and post-caregiving experiences of midlife and older gay men and lesbians. *Journal of Gerontological Social Work*, 47, 121–138.
- Hatano, Y., Fujimoto, S., Hosokawa, T., & Fukui, K. (2015). Association between “Good Death” of cancer patients and post-traumatic growth in bereaved caregivers. *Journal of Pain and Symptom Management*, 50, e4–e6.
- Hurley, R. V. C., Patterson, T. G., & Cooley, S. J. (2014). Meditation-based interventions for family caregivers of people with dementia: A review of the empirical literature. *Aging & Mental Health*, 18, 281–288.
- Lee, Y., Tang, F., Kim, K. H., & Albert, S. M. (2014). The vicious cycle of parental caregiving and financial well-being: A longitudinal study of women. *Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, 70, 425–431.
- Lund, D. A., Utz, R. L., Caserta, M. S., Wright, S. D., Llanque, S. M., Lindfelt, C., ... Montoro-Rodriguez, J. (2014). Time for living and caring: An intervention to make respite more effective for caregivers. *International Journal of Aging and Human Development*, 79, 157–178.
- Mackenzie, C. S., Smith, M. C., Hasher, L., Leach, L., & Behl, P. (2007). Cognitive functioning under stress: Evidence from informal caregivers of palliative patients. *Journal of Palliative Medicine*, 10, 749–758.
- Moore, A. M., Gamblin, T. C., Geller, D. A., Youssef, M. N., Hoffman, K. E., Gemmell, L., ... Steel, J. L. (2011). A prospective study of posttraumatic growth as assessed by self-report and family caregiver in the context of advanced cancer. *Psychooncology*, 20, 479–487.
- Mosher, C. E., Champion, V. L., Hanna, N., Jalal, S. I., Fakiris, A., Birdas, T. J., ... Ostroff, J. S. (2013). Support service use and interest in support services among distressed family caregivers of lung cancer patients. *Psychooncology*, 22, 1549–1556.

- National Alliance for Caregiving. (2016a). About the Alliance. Retrived from <http://www.caregiving.org/about/about-the-alliance/>.
- National Alliance for Caregiving. (2016b). Caregiving in the United States 2015 Research Report. Retrived from http://www.caregiving.org/wp-content/uploads/2015/05/2015_Caregivinginthe_US_Final-Report-June-4_WEB.pdf.
- Navarta-Sanchez, M. V., Senosiain, G., Riverol, M., Ursua, S., Diaz de Cerio, A., Anaut, B., ... Portillo, M. C. (2016). Factors influencing psychosocial adjustment and quality of life in Parkinson's patients and informal caregivers. *Quality of Life Research*, 25, 1959–1968.
- New York State Office of the Aging. (2016). Programs and Services. Retrieved from <http://www.aging.ny.gov/NYSOFA/Services/Index.cfm?id=CSVCS>.
- Northouse, L. L., Katapodi, M. C., Schafenacker, A. M., & Weiss, D. (2012). The impact of caregiving on the psychological well-being of family caregivers and cancer patients. *Seminars in Oncology Nursing*, 28, 236–245.
- Rosenberg, A. R., Baker, K. S., Syrjala, K. L., Back, A. L., & Wolfe, J. (2013). Promoting resilience among parents and caregivers of children with cancer. *Journal of Palliative Medicine*, 16, 645–652.
- Sanyal, J., Das, S., Ghosh, E., Banerjee, T. K., Bhaskar, L. V. K. S., & Raghavendra Rao, V. (2015). Burden among Parkinson's disease care givers for a community based study from India. *Journal of Neurological Sciences*, 358, 276–281.
- Sautter, J. M., Tulsy, J. A., Johnson, K. S., Olsen, M. K., Burton-Chase, A. M., Hoff Lindquist, J., ... Steinhauser, K. E. (2014). Caregiver experience during advanced chronic illness and last year of life. *Journal of the American Geriatric Society*, 62, 1082–1090.
- Skalla, K. A., Lavoie Smith, E. M., Li, Z., & Gates, C. (2013). Multidimensional needs of caregivers for patients with cancer. *Clinical Journal of Oncology Nursing*, 17, 500–506.
- Tedeschi, R. G., & Calhoun, L. G. (1996). The Posttraumatic Growth Inventory: Measuring the positive legacy of trauma. *Journal of Traumatic Stress*, 9, 455–471.
- Thombre, A., Sherman, A. C., & Simonton, S. (2010). Religious coping and posttraumatic growth among family caregivers of cancer patients in India. *Journal of Psychosocial Oncology*, 28, 173–188.
- Thunyadee, C., Sithimongkol, Y., Sangon, S., Chai-Aroon, T., & Hegadoren, K. M. (2015). Predictors of depressive symptoms and physical health in caregivers of individuals with schizophrenia. *Nursing and Health Sciences*, 17, 12–19.
- Vaingankar, J. A., Chong, S. A., Abdin, E., Picco, L., Jeyagurunathan, A., Zhang, Y. J., ... Subramaniam, M. (2016). Care participation and burden among informal caregivers of older adults with care needs and associations with dementia. *International Psychogeriatrics*, 28, 221–231.
- Van't Leven, N., Prick, A. E., Groenewoud, J. G., Roelofs, P. D., de Lange, J., & Pot, A. M. (2013). Dyadic interventions for community-dwelling people with dementia and their family caregivers: A systematic review. *International Psychogeriatrics*, 25, 1581–1603.
- Wennberg, A., Dye, C., Streetman-Loy, B., & Pham, H. (2015). Alzheimer's patient familial caregivers: A review of burden and interventions. *Health and Social Work*, 40, e162–e169.
- Williams, A. L., & McCorkle, R. (2011). Cancer family caregivers during the palliative, hospice, and bereavement phases: A review of the descriptive psychosocial literature. *Palliative and Supportive Care*, 9, 315–325.
- Wisniewski, S. R., Belle, S. H., Marcus, S. M., Burgio, L. D., Coon, D. W., Ory, M. G., ... Schulz, R. (2003). The resources for enhancing Alzheimer's caregiver health (REACH): Project design and baseline characteristics. *Psychology and Aging*, 18, 375–384.
- Yang, Z., & Levey, A. (2015). Gender differences: A lifetime analysis of the economic burden of Alzheimer's Disease. *Women's Health Issues*, 25-5, 436–440.

Close Relationships

Kieran T. Sullivan

Department of Psychology, Santa Clara University, Santa Clara, CA, USA

Close relationships are strongly related to physical health. People who are married live longer and are less likely to experience a host of serious health problems, including heart attacks and chronic illnesses (Johnson, Backlund, Sorlie, & Loveless, 2000). This health advantage holds true when comparing married people with all unmarried groups: divorced, widowed, and never married people (though with recent increases in cohabitation, the gap is narrowing between married and never married people). People in dissatisfying, difficult marriages, however, do not enjoy these health benefits; in fact, they actually fare worse than unmarried people; high conflict and having a critical or demanding spouse undermines health as surely as supportive interactions protect it (Umberson, Williams, Powers, Liu, & Needham, 2006).

How Relationships Affect Health

There are several mechanisms by which intimate relationships influence health. The role and responsibilities of being a long-term intimate partner entail an internal sense of obligation to remain healthy for partners and any children. Indeed, there is ample evidence that simply being married is a significant deterrent of health-compromising behaviors. Beyond the mere presence of a partner and related role obligations, spouses, partners, and close others provide social support, which may entail emotional support or practical (instrumental support) or both. In fact, spouses and intimate partners are by far the most likely person to provide social support when needed. Social support positively affects biological processes (e.g., leads to a stronger immune system), affect, psychological functioning, health behavior, and the ability to cope with illness. Conversely, a lack of social support in close relationships is associated with anxiety and depression, which, in addition to affecting physiological processes, are associated with behavioral patterns that increase risk for disease and mortality.

Social Support

The health benefits of social support are due in part to the mitigation of stress reactions and assistance in coping with stress. The stress-buffering hypothesis suggests that social support provided by intimate partners in the face of stress provides a buffer from the negative effects of stress by positively shaping the stress appraisal response. Individuals who know they have close others who will help them cope with stressors (e.g., provide resources, provide emotional support) appraise that stressor differently than those without such close others. Support may also buffer the effects of stress on physiological processing by attenuating or eliminating the stress reaction or by directly influencing physiological processes (Cohen & Wills, 1985). Indeed, people with high levels of social support, such as those in intimate relationships, who experience stress are less likely to succumb to bacterial and viral infections, are less likely to develop mental health problems as a consequence of stress (such as depression), and are less likely to incur damage caused by neuroendocrine, immune, and cardiovascular responses to stress. Matching the type of support with the type of stressor (i.e., a solution for a practical problem or emotional support for an emotional issue) is key for the buffering effect to occur.

Many studies investigating social support and health have focused on couples dealing with serious or chronic illnesses, a useful context for capturing the practical and emotional support provided by close others. Social support increases patient adherence to medical regimens prescribed by physicians following an illness; the odds of adherence are more than twice as high among patients with greater levels of social support. When distinguishing practical and emotional support, studies indicate that the odds of adherence are more than three and a half times greater for patients receiving practical support (e.g., assistance, reminders, and support for a specific behavior) and almost twice as high for patients receiving emotional support (e.g., nurturance) compared with patients without these types of support (DiMatteo, 2004). Psychosocial interventions with patients and their families designed to increase social support suggest a modestly positive though mixed effect of family involvement; overall evidence indicates that family involvement leads to decreases in depression and mortality (Martire, Lustig, Schulz, Miller, & Helgeson, 2004). Mixed findings regarding social support and illness may be due to variation in the match between desired and received support and the extent to which received or enacted support is perceived as support by the receiver.

Intimate partners also strive to prevent future health problems by deliberating attempting to promote health-enhancing behaviors and inhibit health-compromising behaviors. Such attempts may take the form of social support (helping close others achieve or maintain health through emotional and practical support) and/or social control (deliberate attempts to influence close others' health behaviors).

Social Control

Like social support, social control can have a positive effect on partner health, but it can also impede health behavior change and have negative effects on partners and relationships. According to the dual-effects hypothesis of social control, deliberate attempts by close others to influence health behavior do sometimes improve health behavior but may also lead to psychological distress, including feelings of irritation, hostility, sadness, and guilt (Lewis &

Rook, 1999). Early research guided by this hypothesis, based on general measures of social control, reported mixed results and contributed, in part, to leading researchers to make a distinction between positive and negative social control. Subsequent studies clarified that negative social control (involving tactics such as disapproval, guilt induction, and pressuring) is much less likely to lead to health behavior change and more likely to lead to psychological distress. Negative social control is also much more likely to backfire—resulting in partners hiding behaviors and negatively affecting the relationship. Positive social control (involving tactics such as reinforcing behavior change, modeling, and persuasion) is more likely to positively influence behavior and less likely to lead to psychological or relationship distress. Further, positive social control may even lead to increases in well-being (Craddock, vanDellen, Novak, & Ranby, 2015).

Types of attempts have been delineated beyond positive and negative categories. Researchers have examined the effects of bilateral attempts, such as discussing the issue with the spouse, versus unilateral attempts, such as throwing out unhealthy food, and the effects of direct attempts, such as trying to persuade a partner to make a change, versus indirect attempts, such as inducing guilt. Bilateral and direct attempts are more likely to be associated with health-enhancing behavior compared with unilateral and indirect attempts, though the findings have been mixed (Lewis & Butterfield, 2007). Some evidence suggests that direct attempts do not affect partner behavior immediately, but do affect behavior over time. Conversely, indirect attempts help in the short run, but do not last over time.

The differential effect of types of attempts can be accounted for, at least in part, by the emotions partners experience in response to social control attempts. Early findings that positive and negative aspects of social control are independent gave rise to the domain-specific model, which posits that positive social control is related to positive affect but not related to negative affect, whereas negative social control is related to negative affect but not positive affect; evidence for this model is mixed and a recent meta-analysis suggests that positive social control is positively related to positive affect and negatively related to negative affect. The same holds for negative social control, that is, that negative social control is positively related to negative affect and negatively related to positive affect (Craddock et al., 2015).

According to the meditational model of social control, proposed by Tucker and colleagues, variance in behavioral response to social control strategies is significantly reduced when the affective response to attempts is accounted for, indicating that the way a partner feels about a social control attempt is at least partly responsible for how the partner responds behaviorally (Tucker & Anders, 2001). This model, which has been well supported across many studies, explains why the same attempt (e.g., “Honey, let’s go for a walk”) can have a positive or a negative effect—it all depends on how the partner feels about the suggestion. Positive emotional responses to social control attempts are more likely to lead to behavior change, whereas negative emotional responses are more likely to backfire—engendering resistance and/or hiding of health-compromising behavior.

The expanded meditational model proposes that the effect of social control on health behavior can be better explained if motivation to change the health behavior and reactance (resistance to partner’s attempts to influence health behavior) are taken into account, along with emotional response. Preliminary evidence suggests that health behavior change is more likely when positive social control attempts elicit positive affect *and* predispose the partner to make the change. Further, reactance has a direct negative effect on the likelihood of making a health behavior change. This effect may be in the form of ignoring the attempts and/or hiding the unhealthy behavior (Logic, Okun, & Pugliese, 2009).

Cognitions and Readiness to Change

Social support and social control are distinct concepts, but they are related to one another. Distinguishing between the two—and when an attempt might be construed as positive and negative—can be challenging, as the same type of attempt to influence health behavior may be considered supportive by one person and controlling by another. Researchers have proposed that the attributions that individuals make about their partners' change tactics (e.g., concern about health vs. concern about appearance) may account for variance in how change tactics are received (Lewis & Butterfield, 2005). Cognition is also a feature of the readiness model, which proposes that an individual's readiness to make a change determines whether an attempt is perceived as supportive or controlling and, subsequently, how the individual responds emotionally (Sullivan, Pasch, Bejanyan, & Hanson, 2010). Initial evidence suggests that readiness to make a change causes changes in how an attempt is perceived and the emotional reaction to the attempt and that those perceptions and emotions affect how likely the individual will be to make a change. Perceptions of attempts as supportive and subsequent positive emotional responses lead to greater intentions to make a behavior change, and perceptions as controlling and subsequent negative emotional reactions lead to lower intentions to make a health behavior change.

Predictors and Consequences of Using of Social Control

Much of the research in the area of social control and health focuses on the recipients of control attempts, how they feel, and whether they respond behaviorally. Less is known about the antecedents of social control attempts and the personal and relationship consequences of attempting to influence a partner's health behavior. In a seminal study, Butterfield and Lewis (2002) examined characteristics of the partner trying to influence the other (agent) and characteristics of the partner being influenced (target), as well as characteristics of the relationship and the situation to clarify predictors of social tactic use. In this and a subsequent study, they found that social control approaches vary based on agents' cognitions and emotions. That is, agents used all types (i.e., bilateral, direct, etc.) of tactics more frequently when they were more ready for the partner to make a change. Further, agents' perceptions of how difficult it is for their partners to make the change led to more frequent use of negative and indirect tactics. Finally, agents' learned helplessness regarding their ability to influence partners' health behavior was positively associated with less frequent use of bilateral tactics and more frequent use of negative tactics. Targets' characteristics affected agents' tactic use as well; the more ready a target was to make a change, the more the agent used positive, direct, and bilateral tactics. In terms of consequences of attempts to influence partner behavior, agents experience more stress and more tense interactions when they use social control tactics, but less stress and more enjoyable marital interactions when they employ support tactics.

Context

The contextual model emphasizes the importance of including situational variables in models of social support and social control and understanding the process by which contextual variables affect partner influence on health behavior. The contextual variables that have received attention to date include relationship satisfaction, gender, ethnicity, personality, history of control attempts, and type of behavior (i.e., health enhancing vs. health compromising).

All but the first two variables have received attention only very recently, and the information provided is based on only one or two studies.

Relationship Satisfaction

Relationship satisfaction may moderate the effect of social control on health behavior such that partners experiencing social control attempts are much more likely to benefit from those attempts when their relationship satisfaction is high. Partners who are less satisfied with their relationships are less likely to change their behavior in response to social control attempts, have lower levels of positive affect and higher levels of negative affect in response to partner control attempts, and are more likely to hide unhealthy behavior in response to positive and negative social control attempts. In contrast, couples high in satisfaction are much less likely to experience psychological distress and hide unhealthy behaviors only in response to negative social control. Finally, relationship length is positively associated with behavior change in dating relationships. It is important to note, however, that findings regarding relationship satisfaction are mixed, with some studies reporting no effect of relationship satisfaction.

Gender

Gender appears to be related to the receipt and provision of social control; men are more likely to be the recipients of social control, and women are more likely to attempt to control the health of their partners. There is also evidence of gender differences in affective reactions to and effectiveness of social control attempts. Attempts to influence health are more likely to lead to positive and negative affective responses for women compared with men. Regarding the effectiveness of social control attempts, there is evidence that female partners' change attempts are more effective than male partners' change attempts. Further, male partners' change attempts are more likely to backfire and have the opposite effect of what was intended than women's change attempts. As with relationship satisfaction, it is important to note that findings regarding gender effects are mixed, with some studies reporting no gender differences in receipt/provision of social control or the affective reactions to and effectiveness of social control attempts. Gender may also interact with equity in affecting social control attempts, for example, husbands have been shown to use unilateral and positive tactics more frequently when their wives view the relationship as unequal.

Ethnicity

Few studies have examined ethnic differences in social support or social control of partner health behaviors. One study of dietary behavior in people with diabetes found that partner social support and social control were associated with good dietary behavior in Mexican Americans, but only social support was associated with good dietary behavior in non-Hispanic whites.

Personality

Again, studies here are sparse, but there is evidence that partners who are high in neuroticism experience more social control attempts than partners lower in neuroticism. Further, those high in neuroticism have more negative affective responses to change attempts and are less likely to change their health behavior.

History of Control Attempts

Based on initial evidence, it appears that a history of control attempts by an intimate partner on an individual is associated with an individual's increased use of indirect tactics when attempting to influence the intimate partner's health.

Type of Behavior

Partners use negative tactics less frequently when trying to initiate a health-enhancing behavior compared with when they are trying to terminate a health-compromising behavior. Further, health-compromising behaviors and behaviors with more severe consequences elicit more social control attempts by intimate partners compared with health-enhancing behaviors and behaviors with less serious consequences.

Future Directions

There are a number of avenues by which the understanding of the relationship between intimate relationships and health behavior can be further illuminated: The first is research on the buffering effect of social support and how it may vary based on type of stressors and characteristics of the situation, the support provider, and the recipient. The second is the continued examination of contextual variables and the role they play in the frameworks provided by major models in the field. Because there is so little work on contextual variables, replication is needed for variables presented here, and additional variables need to be examined (e.g., age, previous history of health behavior, whether the health behavior is a shared problem, and health barriers). The third is further research on the characteristics of the partners, including readiness and motivation to change, as well as the attributions regarding control attempts and the affect they have on psychological well-being and health behavior change.

Author Biography

Kieran T. Sullivan is a professor of psychology at Santa Clara University. She has published widely in the area of intimate relationships. She is a consulting editor for the *Journal of Family Psychology*. Her current research focuses on support, control, and health behavior in intimate relationships.

References

- Butterfield, R. M., & Lewis, M. A. (2002). Health-related social influence: A social ecological perspective on tactic use. *Journal of Social and Personal Relationships, 19*, 505–526.
- Cohen, S., & Wills, T. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*(2), 310–357.
- Craddock, E., vanDellen, M. R., Novak, S. A., & Ranby, K. W. (2015). Influence in relationships: A meta-analysis on health-related social control. *Basic and Applied Social Psychology, 37*, 118–130.
- DiMatteo, M. R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology, 23*(2), 207–218.
- Johnson, N. J., Backlund, E., Sorlie, P. D., & Loveless, C. A. (2000). Marital status and mortality: The national longitudinal mortality study. *Annals of Epidemiology, 10*, 224–238.

- Lewis, M. A., & Butterfield, R. M. (2005). Antecedents and reactions to health-related social control. *Personality and Social Psychology Bulletin*, 31, 416–427.
- Lewis, M. A., & Butterfield, R. M. (2007). Social control in marital relationships: Effect of one's partner on health behaviors. *Journal of Applied Social Psychology*, 37, 298–318.
- Lewis, M. A., & Rook, K. S. (1999). Social control in personal relationships: Impact on health behaviors and psychosocial distress. *Health Psychology*, 18(1), 63–71.
- Logic, M., Okun, M. A., & Pugliese, J. A. (2009). Expanding the mediational model of the effects of health-related social control. *Journal of Applied Social Psychology*, 39, 1373–1396.
- Martire, L. M., Lustig, A. P., Schulz, R., Miller, G. E., & Helgeson, V. S. (2004). Is it beneficial to involve a family member? A meta-analysis of psychosocial interventions for chronic illness. *Health Psychology*, 23, 599–611.
- Sullivan, K. T., Pasch, L. A., Bejanyan, K., & Hanson, K. (2010). Social support, social control and health behavior change in spouses. In K. T. Sullivan & J. Davila (Eds.), *Support processes in intimate relationships* (pp. 219–239). New York, NY: Oxford Press.
- Tucker, J. S., & Anders, S. L. (2001). Social control of health behaviors in marriage. *Journal of Applied Social Psychology*, 31(3), 467–485.
- Umberson, D., Williams, K., Powers, D. A., Liu, H., & Needham, B. (2006). You make me sick: Marital quality and health over the life course. *Journal of Health and Social Behavior*, 47, 1–16.

Suggested Readings

- Agnew, C. R., & South, S. C. (Eds.) (2014). *Interpersonal relationships and health: Social and clinical psychological mechanisms*. New York, NY: Oxford University Press.
- Loving, T. J., & Sbarra, D. A. (2016). Relationships and health. In M. Mikulincer, P. Shaver, R. Phillip, J. A. Simpson, J. F. Dovidio, & F. John (Eds.), *APA handbook of personality and social psychology, vol. 3: Interpersonal relations*. Washington, DC: American Psychological Association.
- Newman, M. L., & Roberts, N. A. (Eds.) (2013). *Health and social relationships: The good, the bad, and the complicated*. Washington, DC: American Psychological Association.

Infertility, Miscarriage, and Neonatal Loss

Amy Wenzel

*Department of Psychiatry, University of Pennsylvania School of Medicine,
Philadelphia, PA, USA*

Private Practice, Rosemont, Pennsylvania, PA, USA

For many people, becoming a parent is a life task that carries great significance and meaning. Although the prospect of becoming pregnant and having a child is associated with joy and eager anticipation for many couples, it can be associated with sorrow and despair for couples who experience difficulties. Some couples experience *infertility*, or the inability to conceive after 12 months of regular sexual activity free of birth control. Other couples conceive but lose the baby in various stages of pregnancy. *Miscarriage* and *neonatal loss* occur when the embryo or fetus dies before it can live on its own. The term, miscarriage, is typically used when the loss occurs before the 20th week of gestation (sometimes 24th week of gestation in select studies). In contrast, the term neonatal loss (or *stillbirth*) is typically used when the loss occurs at or after the 20th week of gestation, including instances in which a fetus is carried to term but dies during childbirth. Together, miscarriage and neonatal loss are understood as *perinatal loss*. This article describes the phenomenology of, emotional consequences of, and interventions for infertility and perinatal loss.

Infertility

Infertility is typically designated in two ways. *Primary infertility* occurs when a woman has never conceived, and *secondary infertility* occurs when a woman had been previously pregnant, even if the pregnancy resulted in a loss. Although the definition of infertility presented earlier indicates that it is diagnosed after 12 months of trying unsuccessfully to conceive without using birth control, it is often diagnosed after 6 months of trying unsuccessfully to conceive in women over the age of 35. The rationale underlying the use of a different criterion for women

over the age of 35 is that the likelihood of conceiving declines rapidly after the age of 30, so implementing fertility treatment sooner rather than later in women of advanced maternal age increases the likelihood of a successful pregnancy. Research shows that among women who have attempted to achieve pregnancy, the rate of infertility varies between 16 and 28%. In women over age 40, between 3 and 6% are involuntarily childless due to infertility, and an additional 4–6% have fewer children than desired (Schmidt, 2006).

The causes of infertility are multifaceted. Approximately 80% of infertility cases can be explained by female factors, male factors, or both, leaving the remaining 20% of cases unexplained by medical causes. Causes of female infertility include factors related to ovulation, the cervix or uterus, and/or the fallopian tubes. Women who infrequently ovulate or do not ovulate are at high risk for infertility; these difficulties can be caused by hormonal factors such as polycystic ovary syndrome (PCOS), altered levels of follicle-stimulating hormone (FSH), or luteinizing hormone (LH) due to stress or sudden weight gain or loss, or excess prolactin. Uterine and cervical causes of infertility include fibroids, an abnormally shaped uterus, and cervical stenosis (i.e., narrowing of the cervix). Women who have endometriosis, a condition in which the uterine lining extends outside the uterus, can have difficulty conceiving because resultant scarring can block the fallopian tubes. Moreover, the fallopian tubes can also be blocked by pelvic inflammatory disease, which is an infection of the uterus and fallopian tubes caused by sexually transmitted infections. Male infertility is typically caused either by a low sperm count or poor-quality sperm that are not mobile enough to reach the partner's egg. Some research demonstrates that anxiety and stress are associated with poorer outcomes in infertility treatment (e.g., Boivin & Schmidt, 2005); however, emotional distress is rarely viewed as the sole cause of infertility.

The emotional consequences of infertility can be devastating for those who experience it, as becoming a parent is often central to people's expectations for the course they believe their lives will follow. It is common for people struggling with infertility to report a sense of failure, defectiveness, and incompetence, as well as jealousy of and isolation from others who seem to achieve parenthood easily. Female infertility is commonly associated with a sense of betrayal by one's body. Male infertility is commonly associated with a threat to virility. Although the prevalence of mental health diagnoses is not elevated in people who struggle with infertility, they often report elevated levels of emotional distress on self-report inventories of depression, anxiety, and life satisfaction (Greil, Schmidt, & Peterson, 2016). Women tend to report higher levels of infertility-related depression, anxiety, and stress than do men. In contrast, people who are characterized by resilience, or the capacity to respond adaptively to negative life events, report comparatively lower levels of infertility-related stress, higher quality of life, and more adaptive coping strategies (Sexton, Byrd, & von Kluge, 2010).

Over the past 30 years, technology has allowed for the development of sophisticated approaches to medically assisted reproduction. Assisted reproductive technology (ART) is a category of medical procedures used to achieve pregnancy. Perhaps the most well-known ART is in vitro fertilization (IVF), a procedure in which (a) a woman takes hormones to increase the number of eggs; (b) her eggs are removed and placed in a petri dish along with sperm; (c) fertilization occurs in the petri dish; and (d) one or more embryos are placed in her uterus. Some women choose to freeze embryos and later thaw one or more embryos and have them transferred to the uterus, a procedure called frozen/thawed embryo transfer cycle (FET). Fertilization can also occur using donor eggs, such that after the eggs are fertilized, they are transferred into a woman's uterus. Non-ART medically assisted reproduction interventions include ovulation induction using pharmacotherapy and intrauterine insemination (IUI) using the husband/partner's or a donor's sperm. In developed countries, approximately 56% of people who struggle with infertility seek some form of medically assisted reproduction (Boivin, Bunting, Collins, & Nygren, 2007). The live birth rate in women

under age 40 after undergoing ART is between 15 and 25%, depending in the specific intervention (Schieve, Devine, Boyle, Petrini, & Warner, 2009).

Many people experience a great deal of stress when they undergo medically assisted reproduction interventions, as these interventions can be unpredictable, ambiguous, and time consuming. In many cases, treatment assumes a central focus in their lives, and it can be difficult to know when to discontinue treatment. Some research suggests that infertility treatment confers stress above and beyond that conferred by infertility itself (Greil, Lowry, McQuillan, & Shreffler, 2011). As might be expected, infertility-related emotional distress decreases with successful treatment, and it often persists when treatment is unsuccessful. In addition, infertility and infertility treatment can put great strain on the partner or marital relationship. However, a subset of couples who remain involuntarily childless report that their relationship strengthened because of the shared experience (Peterson, Pirritano, Block, & Schmidt, 2011).

When women become pregnant through medically assisted reproduction technology, they view pregnancy as more stressful and report more anxiety than women who did not experience infertility. Moreover, women who became pregnant through infertility treatment report more emotional distress following a pregnancy loss than women who did not become pregnant through infertility treatment. However, there is no evidence that women whose children were born through infertility treatment parent any differently or report differing levels of parenting stress than women whose children were conceived naturally, although parents who had children using medically assisted reproduction technologies endorse stronger feelings toward their children and a high level of gratitude (Sundby, Schmidt, Heldaas, Bugge, & Tanbo, 2007). There are few long-term consequences of infertility-related emotional distress in couples who eventually have a successful pregnancy or who adopt children; however, emotional distress persists in a subset of people who remain involuntarily childless.

Although the need for infertility-specific mental health services for people experiencing infertility has been recognized, there is a paucity of research that has examined the efficacy of specific interventions for infertility-related emotional distress. Interventions based on cognitive behavioral therapy (CBT) typically include components such as relaxation, cognitive restructuring (i.e., a process by which people evaluate the accuracy and helpfulness of negative thoughts associated with emotional distress), stress management, and psychoeducation about infertility and its emotional consequences. Research shows that such CBT approaches are more efficacious than routine care (Domar et al., 2000) and pharmacotherapy (Faramarzi et al., 2008) in reducing depression, anxiety, and stress in people who struggle with infertility. In contrast, evidence is mixed regarding the degree to which mental health intervention targeting people struggling with infertility actually improves pregnancy rates.

Miscarriage and Neonatal Loss

Because the achievement of parenthood is an important developmental task for many adults, a loss can be devastating on many levels. According to perinatal loss experts David and Martha Diamond,

In addition to the loss of the fetus or baby, which can be a devastating event in and of itself, there are lost opportunities for progress in adult development, for the repair of old wounds, and for the redefinition of relationships with others. The grief, loss, and trauma may re-evoke unresolved grief from the past. Preexisting psychopathology can be exacerbated, and new disorders can be precipitated (Diamond & Diamond, 2016, p. 488).

The often-abrupt shift from the expectation that there will be a new addition to the family to the stark realization that this will not occur prompts a profound sense of disappointment and despair in many people who experience it. Perinatal loss is a particularly difficult event for women to endure, given that they are already in a vulnerable state due to fluctuating hormones, shifting their identity to that of being a parent, and perhaps grappling with hurts from their own parentings of which they were reminded during their pregnancy. However, it is important not to underestimate the toll that perinatal loss takes on men as well.

The overall incidence of miscarriage is between 15 and 20%, and it increases substantially with age, with as many as 75% of women experiencing a miscarriage after age 45 (Hemminki & Forssas, 1999). Most miscarriages occur in the first 12 weeks of gestation, which is why many people refrain from sharing news of the pregnancy until they have completed the first trimester. Symptoms of miscarriage are cramping and vaginal bleeding. An ultrasound verifies that the pregnancy is no longer viable. Many women are advised to simply allow the miscarriage to proceed naturally; however, in instances in which this does not occur, medical intervention may be necessary. Some women undergo *dilation and curettage* (often referred to as a D&C), in which a medical team dilates a woman's cervix and uses surgical intervention to remove the contents of the uterus. Other women are given medicine to induce contractions. The incidence of neonatal loss—loss that occurs at or after 20 weeks of gestation—is approximately 0.6% (MacDorman & Gregory, 2015). Women whose fetus died in utero typically go through the process of labor and delivery. Other women deliver live infants that die soon after birth.

There are many factors that account for perinatal loss. Between 50 and 70% of first trimester miscarriages can be attributed to genetic problems, such as chromosomal abnormalities. Additional causes of miscarriage include infections, maternal medical conditions like diabetes or thyroid disease, hormone problems, immune system responses, uterine abnormalities, and instances in which the egg did not implant properly. Risk factors for miscarriage include advanced maternal age (i.e., age 35 or older), maternal medical conditions like diabetes or thyroid disease, a history of three or more miscarriages, smoking, drinking alcohol, use of illicit drugs, toxins, and obesity. Causes of neonatal loss include placental abruption, premature rupture of membranes (i.e., water breaks prematurely), preeclampsia (i.e., a condition that typically occurs after 20 weeks of gestation, characterized by high blood pressure and potential damage to an organ system like the kidneys), birth defects like structural abnormalities, growth restriction, infection, and umbilical cord abnormalities.

Many people experience a pronounced grief reaction in the time following a perinatal loss. However, the grief is focused on what their child would have been like and what life would have been like with the child, rather than on memories. Grieving a perinatal loss is particularly challenging because there are few culturally sanctioned grieving practices. Many women report significant feelings of guilt and shame, a sense of failure, and self-blame. Women who miscarried in the first trimester often grieve in silence, as they had not yet disclosed the pregnancy to others. Women who were visibly pregnant and suffer a neonatal loss must contend with announcing the news to others and fielding questions from acquaintances and co-workers who ask pregnancy. Men also report elevated grief after perinatal loss, although it is typically less pronounced than in women and more profound in response to a neonatal loss, rather than to a miscarriage. One explanation for this is that men have not experienced quickening (i.e., the perception of fetal movements) and the bodily changes that women typically experience. It is likely that results from studies finding gender differences in grieving perinatal loss can be explained by gender differences in the general expression of grief and emotional distress, with men being more likely to remain stoic and use problem-solving strategies to cope with loss and women being more likely to express their emotions openly.

Complicated grief occurs when the grief reaction becomes chronic and is associated with life interference and significant personal distress. Approximately 10–20% of women who experience perinatal loss struggle with complicated grief, although this range is not appreciably different from the percentage of complicated grief reactions in response to other losses (Brier, 2008). Factors that put women at risk for complicated grief reactions to perinatal loss include poor psychological functioning prior to the loss, poor social support, and a history of other perinatal losses.

A body of research has examined specific symptoms associated with mental health disorders reported by people who have experienced perinatal loss. Research indicates that elevated symptoms of depression in women persist 6 months to 1 year following a perinatal loss and that the women at greatest risk for depression are those who are childless, are highly invested in the pregnancy, and/or have concerns about infertility (Robinson, 2014). Men also report elevated depressive symptoms in the first few months following a perinatal loss, but in contrast to symptoms reported by women, their symptoms drop significantly after 3 months. Moreover, women who have experienced perinatal loss typically report high levels of anxiety during subsequent pregnancies, with the understandable worry that they will experience another loss. In fact, there is evidence that women who have experienced multiple perinatal losses have more emergency department visits and hospitalizations than women without such a history (Kinsey, Baptiste-Roberts, Zhu, & Kjerulff, 2015). Women also endorse rumination and intrusive thoughts up through at least 6 months' post-loss.

Experts have been increasingly recognizing the traumatic nature of perinatal loss. The definition of trauma in the *Diagnostic and Statistical Manual, Fifth Edition* (DSM-5) indicates that a person must have exposure to actual or threatened death, which indeed is the case with a perinatal loss. Many women who experience perinatal loss contend with a great deal of blood and pain. There is a profound sense that something is going horribly wrong, coupled with a sense of uncontrollability. The woman has literally lost an extension of herself in her child, as well as an integral part of a dream for parenthood. For these reasons, Diamond and Diamond (2016) have described perinatal loss as being one type of *reproductive trauma*. Rates of post-traumatic stress symptoms in women who have experienced perinatal loss range from 10 to 25% at 1-month post-loss (Diamond & Diamond, 2016).

In addition, other family members often experience emotional distress associated with the perinatal loss. Children who knew about their mother's pregnancy can experience grief, along with confusion over what happened to the baby. Even when children do not know about the pregnancy, they can often sense when their parents are upset. Grandparents are also affected tremendously by perinatal loss, both in terms of the loss of a grandchild and concern for what their own child is enduring (Robinson, 2014).

Although there is little consensus on the best interventions to deliver to parents who experience perinatal loss, most experts agree that it is important for healthcare providers to acknowledge the meaning associated with the loss, as well as to provide parents with some latitude to choose the best way to proceed with logistical issues (such as how to dispose of the remains, whether to have a memorial service). There is insufficient evidence to support the provision of counseling in the immediate aftermath of a perinatal loss. However, psychotherapy and counseling should be offered as an option when people experience a perinatal loss, with the understanding that it is their choice as to whether they take advantage of it. Psychotherapy or counseling should be recommended at follow-up visits when it becomes clear that their emotional distress is at a level that causes life interference. A topic of debate is whether women who experience advanced neonatal loss should hold their babies. Some parents find that doing so provides a sense of closure. However, some research shows that there is an increase in

depressive, anxiety, and posttraumatic stress symptoms in parents who held their baby, relative to parents who did not (Hughes, Turton, Hopper, & Evans, 2002).

Mental health professionals who work with people who have experienced perinatal loss are encouraged to provide the utmost empathy and compassion. They should create an atmosphere in which the client experiences, rather than avoids, the painful affect associated with the loss. Psychoeducation can be helpful in normalizing grief reactions, and previous unresolved losses that have been activated by the current loss should be addressed (Diamond & Diamond, 2016). CBT interventions have demonstrated efficacy in reducing prolonged grief, depression, anxiety, and posttraumatic stress in an open trial (Nakano, Akechi, Furukawa, & Sugiura-Ogasawara, 2013) and a randomized controlled trial (Kersting et al., 2013).

Author Biography

Amy Wenzel, PhD, ABPP, is the founder and president of Wenzel Consulting, LLC, a clinical assistant professor of psychology in psychiatry at the University of Pennsylvania School of Medicine, an adjunct faculty member at the Beck Institute of Cognitive Behavior Therapy, and an affiliate of The Postpartum Stress Center. She has authored or edited 20 books, many of topics within perinatal psychology.

References

- Boivin, J., Bunting, L., Collins, J. A., & Nygren, K. (2007). An international estimate of infertility prevalence and treatment-seeking: Potential need and demand for infertility care. *Human Reproduction*, 22, 1506–1512.
- Boivin, J., & Schmidt, L. (2005). Infertility-related stress in men and women predicts treatment outcome 1 year later. *Fertility and Sterility*, 83, 1745–1752.
- Brier, N. (2008). Grief following miscarriage: A comprehensive review of the literature. *Journal of Women's Health*, 17, 451–464.
- Diamond, D. J., & Diamond, M. O. (2016). Understanding and treating the psychosocial consequences of pregnancy loss. In A. Wenzel (Ed.), *Oxford Handbook of Perinatal Psychology* (pp. 487–523). New York, NY: Oxford University Press.
- Domar, A. D., Clapp, D., Slawsby, E., Kessel, B., Orav, J., & Freizinger, M. (2000). The impact of group psychological interventions on distress in infertile women. *Health Psychology*, 19, 568–575.
- Faramarzi, M., Alipor, A., Esmaelzadeh, S., Kherikhahm, E., Poladi, K., & Pash, H. (2008). Treatment of depression and anxiety in infertile women: Cognitive behavioral therapy versus fluoxetine. *Journal of Affective Disorders*, 108, 159–164.
- Greil, A. L., Lowry, M., McQuillan, J., & Shreffler, K. M. (2011). Infertility treatment and fertility-specific distress: A longitudinal analysis of a population-based sample of U.S. women. *Social Science & Medicine*, 73, 87–94.
- Greil, A. L., Schmidt, L., & Peterson, B. D. (2016). Understanding and treating the psychosocial consequences of infertility. In A. Wenzel (Ed.), *Oxford Handbook of Perinatal Psychology* (pp. 524–547). New York, NY: Oxford University Press.
- Hemminki, E., & Forssas, E. (1999). Epidemiology of miscarriage and its relation to other reproductive events in Finland. *American Journal of Obstetrics and Gynecology*, 18, 396–401.
- Hughes, P., Turton, P., Hopper, E., & Evans, C. D. (2002). Assessment of guidelines for good practice in psychosocial care of mothers after stillbirth: A cohort study. *The Lancet*, 360, 114–118.
- Kersting, A., Dölemeyer, R., Steinig, J., Walter, F., Kroker, K., ... Wanger, B. (2013). Brief Internet-based intervention reduces posttraumatic stress in parents after the loss of a child during pregnancy: A randomized controlled trial. *Psychotherapy and Psychosomatics*, 82, 372–381.

- Kinsey, C. B., Baptiste-Roberts, K., Zhu, J., & Kjerulff, K. H. (2015). Effect of multiple previous miscarriages on health behaviors and health care utilization during subsequent pregnancy. *Women's Health Issues, 25*, 155–161.
- MacDorman, M. F., & Gregory, E. C. W. (2015). *Fetal and perinatal mortality: United States, 2013*. Atlanta, GA: Centers for Disease Control and Prevention. Retrieved from https://www.cdc.gov/nchs/data/nvsr/nvsr64/nvsr64_08.pdf
- Nakano, Y., Akechi, T., Furukawa, T. A., & Sugiura-Ogasawara, M. (2013). Cognitive behavior therapy for psychological distress in patients with recurrent miscarriage. *Psychology Research and Behavior Management, 6*, 37–43.
- Peterson, B. D., Pirritano, M., Block, J. M., & Schmidt, L. (2011). Marital benefit and coping strategies in men and women undergoing unsuccessful fertility treatments over a 5-year period. *Fertility and Sterility, 95*, 1759–1763.
- Robinson, G. E. (2014). Pregnancy loss. *Best Practice & Research Clinical Obstetrics & Gynaecology, 28*, 169–178.
- Schieve, L. A., Devine, O., Boyle, C. A., Petrini, J. R., & Warner, L. (2009). Estimation of contribution of non-assisted reproductive technology ovulation stimulation fertility treatments to US singleton and multiple births. *American Journal of Epidemiology, 170*, 1396–1407.
- Schmidt, L. (2006). Infertility and assisted reproduction in Denmark: Epidemiology and psychosocial consequences. *Danish Medical Bulletin, 53*, 390–417.
- Sexton, M. B., Byrd, M. R., & von Kluge, S. (2010). Measuring resilience in women experiencing infertility using the CD-RISC: Examining infertility-related stress, general distress, and coping styles. *Journal of Psychiatric Research, 44*, 236–241.
- Sundby, J., Schmidt, L., Heldaas, K., Bugge, S., & Tanbo, T. (2007). Consequences of IVF among women: 10 years posttreatment. *Journal of Psychosomatic Obstetrics & Gynecology, 28*, 115–120.

Suggested Reading

- Covington, S. D., & Burns, L. H. (Eds.) (2006). *Infertility counseling: A comprehensive handbook for clinicians* (2nd ed.). New York, NY: Cambridge University Press.
- Diamond, D. J., & Diamond, M. O. (2016). Understanding and treating the psychosocial consequences of pregnancy loss. In A. Wenzel (Ed.), *Oxford Handbook of Perinatal Psychology* (pp. 487–523). New York, NY: Oxford University Press.
- Greil, A. L., Schmidt, L., & Peterson, B. D. (2016). Understanding and treating the psychosocial consequences of infertility. In A. Wenzel (Ed.), *Oxford Handbook of Perinatal Psychology* (pp. 524–547). New York, NY: Oxford University Press.
- Robinson, G. E. (2014). Pregnancy loss. *Best Practice & Research Clinical Obstetrics & Gynaecology, 28*, 169–178.
- Wenzel, A. (2014). *Coping with infertility, miscarriage, and neonatal loss: Finding perspective and creating meaning*. Washington, DC: APA Books (LifeTools Division).

Prevention and Health Across the Lifespan

John L. Romano

*Department of Educational Psychology, University of Minnesota Twin Cities,
Minneapolis, MN, USA*

Throughout history and across different disciplines, there has been a strong consensus that it is better to prevent a problem than to correct it after it is manifested. This axiom has especially been applied in matters of health, as we are regularly reminded about a host of behaviors and actions that we should take to ward off health problems. These include, among many, eating a healthy diet, being physically active, reducing stress, and getting flu shots. These actions are designed to reduce premature illness and disability and promote the quality of life into older age. However, although there is strong agreement among professionals and the lay public alike about the importance of prevention, there is reluctance and difficulty following preventive actions that science tells us will reduce disease and extend life. In addition to exploring the reluctance and lack of motivation to follow preventive recommendations, this chapter will summarize other areas of prevention as they pertain to matters of health across the lifespan. These areas of prevention include recent definitions of prevention, American Psychological Association (APA) Guidelines for Prevention in Psychology, protective factors to promote health and well-being, prevention in the US Affordable Care Act (ACA), psychological interventions for prevention, and prevention and health in the twenty-first century.

Definitions of Prevention

While the word prevention has an intuitive meaning to most people, the word has generated discussion and nuances among scholars. Primary, secondary, and tertiary prevention were described by Caplan (1964) as activities and interventions designed as appropriate for an entire population (primary), for those at risk for developing a problem (secondary), and to reduce the impact of an existing problem (tertiary). These terms have been in use for many years.

However, Gordon (1987) conceptualized prevention interventions differently, describing prevention interventions as universal, selective, and indicated. Gordon argued that once a problem was manifested (tertiary), interventions for them are not prevention. Gordon's universal prevention is similar to primary prevention, while selective and indicated refer to interventions for groups who are at less (selective) and greater (indicated) risk for developing a disorder. The US Institute of Medicine's Committee on Prevention of Mental Disorders adopted Gordon's definitional terms for prevention in their seminal volume "Reducing Risks for Mental Disorders" (Mrazek & Haggerty, 1994). Romano and Hage (2000) expanded this definition by incorporating the strengthening of individual protective factors and institutional change strategies as important components of a comprehensive definition of prevention. In a later volume, the National Research Council and Institute of Medicine (2009) also incorporated reduction of risk and promotion of protective factors in its conceptualization of prevention. In a recent volume, Conyne (2015) described everyday wellness and prevention as actions taken to improve "personal wellness and to avert significant problems, while contributing to the health and well-being of others" (p. 19). This definition incorporates individual empowerment and systemic change as important prevention strategies. As the professional specialty of prevention has expanded, definitions of prevention have become more focused and inclusive, not only attending to individuals and groups but also recognizing that systemic and institutional preventive interventions are needed to address major societal problems. As will be discussed in this chapter, the US ACA: Affordable Care Act (Federal Law, also called "Obama Care," 2010), while controversial, is one example of legislation that incorporates prevention services as important to overall healthcare, and as described in the following section, APA's Guidelines for Prevention in Psychology reinforce the importance of prevention in psychology.

Guidelines for Prevention in Psychology

It has been well documented that historically prevention has not held a very visible or prominent position in the field of psychology (Romano & Hage, 2000). Prevention is generally not a core course within psychology graduate programs, and prevention has not been considered as a specific competency or domain in accreditation of graduate training programs, or as an educational requirement for license as a psychologist. While community psychology, with a strong prevention and social justice focus, was organized as a specialty in the mid-1960s and other organizations such as the Society for Prevention Research and the Prevention Section of APA's Society of Counseling Psychology (Division 17) were created over the last 25 years, the impact of these organizations on the broader profession of psychology has been modest.

To advance the cause of prevention, science and its visibility in psychology, a group of APA-member psychologists, began the arduous task (over multi-years) of developing an APA set of guidelines for prevention in psychology. The guidelines, based on an earlier "*Best Practices in Psychology*" manuscript (Hage et al., 2007), were vetted by all major psychology stakeholders (e.g., professionals and consumers), revised accordingly, and eventually approved by APA Council and published in the *American Psychologist* (APA, 2014).

The *Guidelines for Prevention in Psychology* are very relevant to all of psychology, including health psychology. The Guidelines, which are aspirational, consist of nine guidelines of best practices for prevention psychology. The Guidelines recommend the following: (a) prevention interventions that are theory driven and supported by evidence, (b) prevention science that attends to socially and culturally appropriate practices, (c) prevention that addresses risk as well as protective factors to enhance well-being, (d) prevention activities include research and

evaluation as integral to assess prevention practices, (e) prevention research and practices that include consideration of ethical issues as they pertain to prevention, (e) prevention specialists who attend to social disparities that may inform science and practice, (f) psychologists encouraged to seek out prevention training through various avenues throughout their career, (g) psychologists engaged in systemic change interventions to strengthen organizations and units of society to reduce distress and disability, and (h) psychologists encouraged to contribute to public policy discourses to inform decision makers about topics relevant to prevention science.

The APA Prevention Guidelines are important to health psychology as they offer a set of aspirational recommendations to psychologists and other mental health workers to advance the cause of prevention in their clinical and research work. The Guidelines also reinforce the importance of not only stopping and reducing problematic behaviors but also strengthening factors that can be protective to individual persons as well as groups and communities. For example, health psychologists can work with schools and workplaces to promote healthy lifestyles, which serve as protections against future disease. The Guidelines also address the relevance of focusing on institutional and public policies to reinforce prevention of health problems and health promotion at the macro levels of society. In the United States, these macrolevel initiatives have reduced tobacco addiction (although it took many years and needs to be regularly reinforced), have produced dietary guidelines to promote good health, and passed laws to reduce traffic fatalities (e.g., use of seat belts, alcohol use, distracted driver penalties), to name a few examples. Community policies and laws to promote health are often contentious and controversial, and thus, at systemic levels, prevention and health promotion initiatives usually become very much a question about individual and community values, beliefs, and attitudes, as exemplified by efforts to enact legislation prohibiting cigarette smoking in bars and restaurants and laws to control access to guns to reduce crime.

Protective Factors to Promote Health and Well-Being

The promotion of protective factors is important to an overall framework of prevention initiatives. Protective factors are characteristics of an individual, small group, institution, or a larger community that can serve as protections against problematic behaviors now or in the future. Scholars have conceptualized protective factors in different ways. For example, Lösel and Farrington (2012) identified six types of protective factors that can serve as buffers to violent behaviors, that is, biological, personality, family, school, peers, and community. While Lösel and Farrington directly applied these factors to the prevention of youth violence, they offer a comprehensive framework for prevention of other problems and health promotion across the lifespan. For example, in healthcare settings, providers can assess protective factors related to health problems of an elder patient, such as support from family to follow prescribed care to reduce the risks of an emerging health problem; in younger adults, providers can assess genetic influences that may be related to a potential health problem. In addition, providers can assess personality characteristics (e.g., conscientiousness) and motivations to make health-enhancing changes to reduce risk. Too often, patients hear what not to do (e.g., stop smoking, eat better, exercise, etc.), but for many these represent long-term habits (e.g., addictions) and are very difficult to change without specific direction and guidance from professionals. Therefore, it is important that health providers discuss with patients how they might go about making lifestyle changes to improve health and provide referrals and other recommendations that offer specific guidance for making positive changes in lifestyle behaviors. The change process will be strengthened if protective factors are enhanced as part of the strategy to reduce problem behaviors.

Others have also address protective factors through specific frameworks. For example, Romano (1997) articulated the importance of promoting student well-being in schools by focusing on strengthening students' knowledge, skills, attitudes, and behaviors that promote well-being in school, at home, and in the community. Building resiliency in youth and young adults has also enriched research and practice to strengthen protective factors (e.g., APA Task Force on Resilience and Strength in Black Children and Adolescents, 2008; Luthar, Cicchetti, & Becker, 2000). Promoting protective factors in older adults has also been examined. These interventions can take the form of providing psycho-educational programs related to healthy lifestyles, developing social support networks to reduce social isolation, and following medical regimens (Reynolds et al., 2012). In addition, positive psychology (Seligman & Csikszentmihalyi, 2000), with its emphasis on the study of strengths and well-being, has received much attention in clinical and research practices, and thus, positive psychology offers another perspective through which to focus on protective factors in prevention science.

Finally, at the macro level, it is important to also promote community and societal protections and buffers to problem behaviors to reduce risks. Vera and Shin (2006) refer to the importance of reducing toxic environments, communities, and institutions that allow discrimination and disenfranchisement to exist among particular groups within a locale or larger society. Advocating for protections that provide adequate healthcare, housing, employment, and education for all members of a community is extremely important to support an overall health and wellness agenda across all population groups. However, suggestions on how best to achieve such community-wide initiatives are usually burdened with political controversies. One notable example is the US ACA, which became the law in 2010, but the ACA merits have been hotly debated ever since passage. However, as discussed in the next section, ACA includes many benefits to support preventive interventions across the lifespan.

Prevention and the Affordable Care Act

The US ACA has generated much controversy since it was passed in 2010 as policy for delivering and providing healthcare across the United States. Despite the political controversies surrounding the law, ACA provides for many benefits that support prevention, and psychologists are well positioned to provide leadership and services. Specifically, ACA provides for the Prevention and Public Health Fund that supports grants and state funding for a range of prevention programming, including school-based health services, maternal health and early childhood home visit programs, and cultural competence training. In addition, ACA supports preventive patient healthcare across the lifespan, including adolescent alcohol and drug screening and assessment, developmental screening for children under age three with continued surveillance throughout childhood, and adolescent screening and counseling for obesity. In addition, ACA provides for screenings and counseling for adults coping with alcohol misuse, diet counseling for those at risk for chronic disease, and tobacco use screening and interventions. The law also provides for annual wellness visits for seniors through Medicare and preventive services for those on Medicaid. Psychologists are encouraged to review the benefits of the ACA law and educate their clients about services that can be received under the law.

In addition, psychologists can be an excellent resource for primary care physicians and other health professionals as they are often the first ones who learn that their patients are at risk for mental or physical health problems. (See special issue of the *American Psychologist* on primary care and psychology [McDaniel & deGruy, 2014] for several papers on this topic.) Working in collaboration, psychologists and primary care providers can form a powerful

partnership to prevent health problems and reduce patient risk. Integrated models of care between psychologists and physicians have been advanced to strengthen these collaborations (Vogel, Kirkpatrick, Collings, Cederna-Meko, & Grey, 2012). Psychologists not only can assist in direct patient care but also can serve as partners and consultants in developing public health media campaigns and prevention materials that are usually available in healthcare settings. One adolescent survey conducted by Taveras et al. (2007) found that while adolescents reported to health providers during physical exams about their lifestyle behaviors that placed them at risk for different problems, they were not offered counseling to change risk behaviors. With benefits available through ACA, perhaps more patients across the lifespan will receive prevention counseling to change problematic lifestyle behaviors and habits that place them at risk for future disease and disability. The ACA offers opportunities for prevention-oriented psychologists to engage with healthcare professionals to offer benefits and services to improve health across the lifespan.

Psychological-Based Theoretical Interventions for Prevention

Patients are often told to change a habit of lifestyle to improve health and well-being by healthcare professionals. These directives may include, for example, improve diet, lose weight, stop smoking, consume less alcohol, control anger, etc. However, much less often are patients in primary care settings given the tools to make these behavioral changes, and, in addition, most primary care physicians and other health professionals do not have the time and expertise to engage in counseling and coaching with their patients on how best to make these changes. Therefore, behavioral specialists, for example, counselors, psychologists, or other mental health and behavioral specialists, are the most appropriate professionals to engage patients in the process to make necessary behavioral changes, thus, another important reason to advance integrated models of care (Kelly & Coons, 2012). Mental health and behavioral professionals are most often educated and trained in theories of counseling and psychotherapy that have been popularized through research and practice in settings that focus on crisis intervention and remediation. Common theories taught in applied psychology and counseling graduate programs include psychodynamic theories, cognitive behavioral theory, person-centered theory (PCT), and behavioral theories. While these theories may also be applied in preventive counseling and coaching situations, there are other psychological theories and perspectives that are neglected in many graduate training programs. However, less commonly taught theories can provide additional theoretical frameworks for the preventive counseling practitioner and researcher. These theories will be briefly summarized in this section.

Romano (2015) presented a summary of several theories that have been applied in prevention research and practice. They will briefly be reviewed here, and readers who are unfamiliar with these theories are encouraged to learn more about them.

Transtheoretical Model of Behavior Change (TTM)

TTM is a stage model of behavioral change introduced by Prochaska and colleagues (Prochaska, DiClemente, & Norcross, 1992; Prochaska, Johnson, & Lee, 2009). TTM asserts that behavioral changes occur in stages, although the stages are not necessarily linear. The six stages of change are precontemplation, contemplation, preparation, action, maintenance, and termination. This model is useful because it acknowledges that before change can occur, clients must be motivated to make a change, prepare a plan of action and carry out the plan, and provide a

system for maintaining the change. For many behavioral changes of lifestyle, for example, smoking cessation, improvements in diet, maintenance continues, and termination is not relevant. TTM is useful in behavioral change counseling because it acknowledges that clients may not be ready or motivated for change (action stage), and if the counselor begins with preparation and action stages, without considering the client's readiness for change (precontemplation and contemplation), the counseling effort may go for naught and result in early termination of counseling. Prochaska and colleagues have developed a well-thought-out and evidence-based theory that is useful when assisting clients to make behavioral changes.

Theory of Reasoned Action and Planned Behavior (TRA/PB)

The TRA was first proposed by Fishbein (1967) and subsequently extended by Ajzen (1991) into the TRA and PB. TRA offers a framework to understand the relationship between beliefs, attitudes, and behaviors, and Ajzen's component (theory of PB) addressed the extent that a person believes that a behavior is under his/her control. TRA/PB is a cognitive model of behavior change in which change is influenced by a person's beliefs and attitudes about the behavior change, and these variables are further influenced by the opinions of others (social norms) about the behavior change and the person's belief that he/she can make the change. Thus, within a prevention framework and applying this theory, it is important to assess the client's attitudes and beliefs about making a change and the extent that the person believes that change is possible for him/her. Applying TRA/PB to alcohol use prevention with adolescents, for example, a provider would assess the adolescent's beliefs and attitudes about not using alcohol and how nonuse would be viewed by others important to him/her (e.g., peers, parents, coaches, etc.). In addition, the provider would discuss the youth's self-perception of being able to avoid alcohol use. In this example, the adolescent may believe that avoiding alcohol will make him/her less popular with peers, but abstinence from alcohol would receive strong approval from parents and other adults important to the adolescent.

Thus, in the TRA/PB framework, assessing beliefs and attitudes is necessary to bring about behavior change. One contribution of TRA/PB is elicitation research, which is a process to assess perceived beliefs among a group targeted for behavior change prior to initiating prevention activities associated with the desired change. For example, a medical clinic with a large Hmong client population may use elicitation research to initially assess with a pilot group of similar Hmong patients the most important beliefs about a desired behavior change (e.g., regular health screenings) prior to initiating a larger-scale program to encourage change among the Hmong population. Elicitation research data can be collected in different ways, for example, focused groups, questionnaires. Elicitation research recognizes that beliefs about a behavior change are not universal and that the most effective interventions will be population specific.

Health Belief Model (HBM)

HBM was developed by US social psychologists in the 1950s to better understand why some people do not participate in health screenings to prevent illness (Rosenstock, 1974). HBM is a cognitive theory based on a person's beliefs about the importance of health screenings. HBM is based on four perceived personal beliefs about his/her risk for contracting an illness or disease, and these beliefs influence whether or not a person participates in preventive health screenings and actions. The four perceived beliefs are susceptibility, severity, benefits, and barriers. Personal beliefs about susceptibility and severity of an illness as well as perceived benefits and barriers, according to HBM, are determinants and motivations for participating

in health screenings. For example, a woman with a family history of breast cancer is likely to be more diligent scheduling regular mammograms, and her motivation to receive the screenings will be increased if the procedure is paid for by health insurance. While HBM was originally developed to better understand the lack of participation in some preventive health screenings, the model can also be considered as a preventive theory to address nonmedical problems, for example, assessment of risk associated with drinking and driving. The model may be especially useful with children and adolescents who underestimate their susceptibility and risk associated with problem behaviors, for example, experimenting with cigarette smoking will not lead to addiction (“I can quit anytime”). It can also be helpful to practitioners to assess perceived personal beliefs among clientele from different ethnic groups to better understand differences between a provider’s beliefs about engaging in a preventive behavior and beliefs of clients from different ethnic groups.

Motivational Interviewing (MI)

MI has received considerable attention in the literature, especially in recent years (Miller & Rollnick, 2002). MI has its foundation in the work of Carl Rogers and his PCT. Although MI is more directive than PCT, MI recognizes the value and importance of the therapeutic relationship in behavior change. Four principles guide MI: (a) *express empathy*, as the counselor communicates empathetic understanding toward the client; (b) *develop discrepancy*, in which the counselor confronts the client about motivations to maintain or change behavior; (c) *roll with resistance*, as the counselor nonjudgmentally accepts client resistance to change; and (d) *support self-efficacy*, as the counselor assists the client to gain awareness about skills that he/she possesses to make behavioral changes. While MI was initially developed to help people stop addictive behaviors, it has been applied across a wide range of behaviors. MI would be most useful to assist individuals who are at risk for problematic behaviors (selective and indicated prevention). For example, MI might be used in a college setting to assist students who have low academic achievement or students who have been cited for problematic behavior related to alcohol use. In a medical setting, MI can be a framework to assist individuals whose lifestyle behaviors place them at risk for developing a future medical disease, such as obesity as a risk for diabetes. MI has been applied in brief counseling interventions with supplemental activities to change behaviors (e.g., Murphy et al., 2012), and thus, MI offers an advantage in medical settings where time may be limited for longer interventions.

The theoretical frameworks summarized in this section are examples of models that can form a foundation for health providers to assist people make behavioral changes. There are other theories that can also be helpful to clinicians, and it is also understood that theoretical approaches do not necessarily function in isolation from each other. A practitioner may use more than one theory to facilitate client change during the counseling/coaching process, depending on the nature of the desired behavioral change, client demographics and readiness for change, and the provider’s comfort applying the theoretical framework.

Prevention and Health in the Twenty-First Century

This manuscript has presented background on prevention science and the specialty’s contribution to strengthening the overall health of a population. Prevention science can be applied across the lifespan, and as psychologists and other mental health professionals gain increased opportunities to collaborate with medical personnel, the collaboration offers

tremendous opportunities to assist people make necessary behavioral changes to improve health. Prevention offers much promise to reduce noncommunicable diseases (contributed to by behaviors of lifestyle) and extend the quality of life, especially with an aging US population. The role of psychologists as members of integrated healthcare teams is increasing, and applying prevention in these settings is critically important (see McDaniel & deGruy, 2014 and recently released APA video [www.apa.org/health/psychologists-integrated-care.aspx]).

Psychologists are encouraged to become familiar with the US government's major initiative in setting and monitoring health goals for the nation, *Healthy People 2020* (www.healthypeople.gov). *Healthy People* was first published in 1979 and at subsequent 10-year intervals, to measure progress, set goals, and make recommendations to improve the overall health and well-being of the population. The mission of *Healthy People 2020* is to identify national health improvement priorities; increase public awareness and understanding about the determinants of health, disease, and disability; apply measurable objectives and goals to monitor progress; engage multiple sectors of the society to improve health; and identify critical research, evaluation, and data collection needs to assess progress. The goals of *Healthy People 2020* are to promote longer life that is free of preventable disease, promote health equity and eliminate health disparities, create social and physical environments that support health, and promote quality of life and healthy development and healthy behaviors across the lifespan.

Healthy People 2020 is the single best resource to review and collect information about the health status of the US population and become knowledgeable about health promotion and disease prevention in the United States. Researchers and practitioners can search for research literature, evidence-based interventions, and professional tools that support health improvements/promotions and disease prevention across many health conditions and within different sectors of the society.

In addition, to provide direct health promotion and disease prevention programs and activities with at-risk patients, psychologists are encouraged to consider prevention interventions that offer assistance to those who are in relationship with people with illnesses. For example, psychologists can provide assistance and support to caregivers of the elderly who have developed cognitive disorders. Attending to caregivers of the elderly will become increasingly important with an aging US population. As the population ages, chronic disease and disability will also increase. Therefore, offering programs and support for those who care for this population, either in institutions or at home, is critically important in the twenty-first century. For example, personnel in nursing homes and assisted living settings will need support to help them understand the needs of disabled elders and also to prevent burnout and emotional stress that can accompany caring for disabled elderly. In addition, relatives who care for family members also need assistance to help them understand the disease and disability of their loved one and also to help them manage their own stress levels related to caregiving. Psychologists and other mental health professionals can also assist with end-of-life processes and issues that arise with terminally ill patients and their loved ones. Hospice programs are increasingly utilized across the United States to support quality of life at the end of life for the terminally ill. Hospice programs funded through Medicare/Medicaid and most private insurance policies offer psychological support and medical assistance during this difficult time to patients and their caregivers to manage patient pain and to educate and provide support during the dying process. As written in the Medicare legislation, direct mental health and social services in hospice programs are usually provided by social workers. However, psychologists may serve as bereavement counselors and can support the programs by applying their expertise through training and education of hospice personnel as well as family members of the dying patient to prevent excessive stress and burnout of professional personnel and family members.

Summary

This paper has highlighted some of the ways that health and prevention science intersect. In a previous volume, Romano (2015) summarized research and literature germane to the work of psychologists that has been conducted in the medical sector such as prevention of depression in geriatric patients and public health campaigns to improve health and prevent disease. There are many opportunities for psychology to apply the theories and practices of the profession to advance the health and well-being of the population through prevention science research and applications. The time is ripe to apply scientific advances in prevention science to reduce illness and disability and enhance the quality of life across the life cycle.

Author Biographies

Dr. John L. Romano is an emeritus professor of educational psychology at the University of Minnesota. Romano received his bachelor, masters, and doctoral degrees from Le Moyne College (Syracuse, NY), Pennsylvania State University, and Arizona State University, respectively. He has authored prevention-focused journal articles and books including the books *Prevention Psychology: Enhancing Personal and Social Well-Being* (APA, 2015) and coedited *The Cambridge Handbook of International Prevention Science* (Cambridge, 2017). He served as cochair and chair of the work group that produced the *APA Guidelines for Prevention in Psychology*, and he has been the recipient of several US Department of Education grants to promote safe and drug-free schools. Romano is a fellow of the American Psychological Association (APA). He has received several awards including the Prevention Section (APA's Society of Counseling Psychology) Inaugural Lifetime Achievement Award, Distinguished Contributions to Counseling Psychology (APA Society of Counseling Psychology), APA's International Psychology Division International Mentoring Award, and the American Counseling Association Research Award. He has been a visiting professor, consultant, and external examiner at universities in several countries.

References

- Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, 50, 179–211.
- APA. (2014). Guidelines for prevention in psychology. *The American Psychologist*, 69, 285–296.
- APA Task Force on Resilience and Strength in Black Children and Adolescents. (2008). *Resilience in African American children and adolescents: A vision for optimal development*. Washington, DC: Author. Retrieved from <http://www.apa.org/pi/cyf/resilience>
- Caplan, G. (1964). *Principles of preventive psychiatry*. New York, NY: Basic Books.
- Conyne, R. K. (2015). *Counseling for wellness and prevention: Helping people become empowered in systems and settings* (3rd ed.). New York, NY: Routledge.
- Fishbein, M. (Ed.) (1967). *Readings in attitude theory and measurement*. New York, NY: Wiley.
- Gordon, R. (1987). An operational definition of disease prevention. In J. A. Sternberg & M. M. Silverman (Eds.), *Preventing mental disorders* (pp. 20–26). Rockville, MD: U.S. Department of Health and Human Services.
- Hage, S. M., Romano, J. L., Conyne, R. K., Kenny, M., Matthews, C., Schwartz, J. P., & Waldo, M. (2007). Best practice guidelines on prevention practice, research, training, and social advocacy. *The Counseling Psychologist*, 35, 493–566.

- Kelly, J., & Coons, H. L. (2012). Integrated health care and professional psychology: Is the setting right for you? *Professional Psychology: Research & Practice*, 43, 586–595.
- Lösel, F., & Farrington, D. P. (2012). Direct protective and buffering protective factors in the development of youth violence. *American Journal of Preventive Medicine*, 43(Suppl.1), 8–23.
- Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, 71, 543–562.
- McDaniel, S. H., & deGruy III, F. V. (2014). Special issue scholarly lead: An introduction to primary care and psychology. *American Psychologist*, 69, 325–331.
- Miller, W. R., & Rollnick, S. (2002). *Motivational interviewing* (2nd ed.). New York, NY: Guilford Press.
- Mrazek, P. J., & Haggerty, R. J. (Eds.) (1994). *Reducing risks for mental disorders: Frontiers for preventive intervention research*. Washington, DC: National Academy Press.
- Murphy, J. G., Denhart, A. A., Skidmore, J. R., Borsari, B., Barnett, N. P., Colby, S. M., & Martens, M. P. (2012). A randomized controlled trial of a behavioral economic supplement to brief motivational interventions for college drinking. *Journal of Consulting and Clinical Psychology*, 80, 876–886.
- National Research Council and Institute of Medicine. (2009). *Preventing mental, emotional, and behavioral disorders among young people: Progress and possibilities*. Washington, DC: National Academies Press.
- Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change: Applications to addictive behaviors. *American Psychologist*, 47, 1102–1114.
- Prochaska, J. O., Johnson, S., & Lee, P. (2009). The transtheoretical model of behavior change. In S. A. Shumaker, J. K. Ockene, & K. A. Riekert (Eds.), *The handbook of behavior change* (3rd ed., pp. 59–83). New York, NY: Springer.
- Reynolds, C. F., Cuijpers, P., Patel, V., Cohen, A., Dias, A., Chowdhary, N., ... Albert, S. M. (2012). Early intervention to reduce the global health and economic burden of major depression in older adults. *Annual Review of Public Health*, 33, 123–135.
- Romano, J. L. (1997). School personnel training for the prevention of tobacco, alcohol, and other drug use: Issues and outcomes. *Journal of Drug Education*, 27, 245–258.
- Romano, J. L. (2015). *Prevention psychology: Enhancing personal and social well-being*. Washington, DC: American Psychological Association.
- Romano, J. L., & Hage, S. M. (2000). Prevention and counseling psychology: Revitalizing commitments for the 21st century. *The Counseling Psychologist*, 28, 733–763.
- Rosenstock, J. M. (1974). Historical origins of the health belief model. *Health Education Monographs*, 2, 328–335.
- Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist*, 55, 5–14.
- Taveras, E. M., Sobol, A. M., Hannon, C., Finkelstein, D., Wiecha, J., & Glottmaker, S. L. (2007). Youths' perceptions of over-weight related prevention counseling at a primary care visit. *Obesity*, 15, 831–836.
- Vera, E. M., & Shin, R. Q. (2006). Promoting strengths in a socially toxic world: Supporting resilience with systematic interventions. *The Counseling Psychologist*, 34, 80–89. Int.
- Vogel, M. E., Kirkpatrick, H. A., Collings, A. S., Cederna-Meko, C. L., & Grey, M. J. (2012). Integrated care: Maturing the relationship between psychology and primary care. *Professional Psychology: Research & Practice*, 43, 271–280.

Suggested Reading

- Israelashvili, M., & Romano, J. L. (Eds.) (2016, September). *The Cambridge handbook of international prevention science*. Cambridge University Press.
- McDaniel, S. H., & de Gruy, F. V. 2014, May/June Special Issue: Primary Care and Psychology. *American Psychologist*, 69(4).
- Romano, J. L. (2015). *Prevention psychology: Enhancing personal and social well-being*. Washington, DC: American Psychological Association.

Self-Affirmation and Responses to Health Messages: An Example of a Research Advance in Health Psychology

Allison M. Sweeney¹ and Anne Moyer²

¹*Department of Psychology, University of South Carolina, Columbia, SC, USA*

²*Department of Psychology, Stony Brook University, Stony Brook, NY, USA*

People, especially those at risk, tend to resist persuasive appeals to change their behavior. When people are uninterested in changing a health behavior, they may try to discredit unwanted health information by forming counterarguments or alternative explanations (Ditto & Lopez, 1992). To the extent that people tend to prefer positive information that reflects well on the self, it is not surprising, then, that people scrutinize unfavorable information more than favorable information (Giner-Sorolila & Chaiken, 1997). To the extent that health information reminds people of their shortcomings, it can be a potent threat to their positive self-views. Ironically, the more personally relevant a health message is, the less likely an individual is to process it objectively (Lieberman & Chaiken, 1992).

One promising approach to overcoming resistance to health information involves leading people to think about their most important values or actions, a process known as self-affirmation (Steele, 1988). Self-affirmation theory proposes that highlighting important sources of self-worth restores or reinforces an individual's overall sense of self-integrity. By making salient aspects of the self that are important to one's identity, but are unrelated to the threat at hand, self-affirmation helps to offset any potential self-threat elicited by health information (Sherman & Cohen, 2006). As a result, self-affirmation should reduce any motivation to respond defensively to health information. Supporting this prediction, a number of studies have found that when people are self-affirmed, they are more likely to process health information in an objective manner (Reed & Aspinwall, 1998; Sherman, Nelson, & Steele, 2000).

Much of the early research on self-affirmation and health focused on the ability of self-affirmation to change people's responses to health information, including message acceptance (Sherman et al., 2000). Further efforts have been made to clarify whether increasing people's openness toward health information translates into a change in future health-related behaviors. Several studies indicate that self-affirmation facilitates behavior change across a diverse range of behaviors, including physical activity, fruit and vegetable consumption (Epton & Harris, 2008), alcohol consumption (Armitage, Harris, & Arden, 2011), and adherence to medication (Wileman et al., 2014). Such studies typically examine the influence of self-affirmation over the course of a week or longer, with some studies finding effects extending over several months (Harris et al., 2014; Wileman et al., 2014).

Self-affirmation has a small-sized effect on health behaviors (Epton, Harris, Kane, van Koningsbruggen, & Sheeran, 2015; Sweeney & Moyer, 2015). Although small, the effect size of self-affirmation is similar to those obtained in meta-analyses of other health behavior change interventions (e.g., Gallagher & Updegraff, 2012). Thus, self-affirmation holds considerable promise as a useful framework for explaining when people accept health information and, in turn, make changes in their behavior. In this chapter we will examine some of the recent developments and discoveries among studies of self-affirmation, including (a) the incorporation of diverse research methods, (b) the identification of moderating variables, and (c) efforts to combine self-affirmation with other intervention strategies. We will then highlight two major questions for future research: (a) how does self-affirmation influence health behaviors and (b) what is the trajectory of self-affirmation?

Applying Diverse Research Methods to Studies of Self-Affirmation

Many early studies on self-affirmation and health-related outcomes consisted of laboratory-based studies that measured immediate responses to health information. After completing a self-affirmation or a control task, people were presented with health information and reported on their responses to it, such as behavioral intentions and message processing (Reed & Aspinwall, 1998; Sherman et al., 2000). Within the last decade, however, researchers have begun to examine whether self-affirmation impacts outcomes beyond people's self-reported experiences. An emerging literature encompassing a diverse array of methods suggests that self-affirmation impacts the body's immediate and physiological response to threat. For example, relative to non-affirmed individuals, people who are self-affirmed have a lower cortisol response when exposed to a stressful situation (Creswell et al., 2005) and take less time for their mean arterial blood pressure to return to baseline after receiving threatening information (Schmeichel & Tang, 2015).

To date, two studies have examined the influence of self-affirmation on neural activity.

To examine the effect of self-affirmation on immediate sensitivity to threat, participants completed a speeded response time task while their neural activity was recorded with electroencephalography (EEG) (Legault, Al-Khindi, & Inzlicht, 2012). The response time task was a go/no-go task that involved making or withholding a response depending on the type of stimuli presented. Participants were instructed to respond as quickly as possible and, thus, were prone to errors. The researchers tested whether self-affirmation would impact people's sensitivity to making errors by examining their error-related negativity (ERN) response, an event-related potential found in past research to index motivational significance. Participants who self-affirmed prior to the go/no-go task displayed a greater ERN response, suggesting that they were more sensitive toward making mistakes during the response time task. To the

extent that errors are experienced as aversive, the authors interpreted this finding as suggesting that self-affirmation increases people's openness to processing threat.

Adopting a different approach, one recent study used functional magnetic resonance imaging (fMRI) to examine neural activity during the processing of health information (Falk et al., 2015). The results indicated that people who were self-affirmed showed greater activation in the ventromedial prefrontal cortex (VMPFC), an area of the brain associated with self-related processing when reading the health message. Furthermore, increased activation in the VMPFC was associated with greater health behavior change, suggesting that self-affirmation may engender health behavior change by allowing people to process otherwise threatening health information as self-relevant. Taken together, research by Legault et al. (2012) and Falk et al. (2015) provide initial evidence that applying a neurophysiological approach to self-affirmation may be a useful framework for further understanding the cognitive and motivational processes that underlie self-affirmation.

Another notable change among studies of self-affirmation has been an increased emphasis on measuring behavioral changes arising from self-affirmation. Attempts to examine the impact of self-affirmation on health behaviors have relied primarily on self-reports (e.g., Armitage et al., 2011; Epton & Harris, 2008). Recently, however, a few studies have begun to incorporate more objective measures of health behavior change. For example, one study found that self-affirmation led to a reduction in sedentary behavior, as assessed with accelerometers worn by participants for 1 month (Falk et al., 2015). Another study examined whether self-affirmation would encourage hemodialysis patients to adhere to recommendations (Wileman et al., 2014). By measuring patients' serum phosphate levels across 1 year, the researchers were able to show a positive effect of self-affirmation on medication adherence.

Moderators of Self-Affirmation's Effects: The Content of the Message and Characteristics of the Individual

As research on self-affirmation has accumulated, researchers have identified several factors that moderate the effectiveness of self-affirmation. For example, it may be worthwhile to consider the content of health messages and the way that this interacts with self-affirmation. One study examined the strength of a message related to the risk of fibrocystic disease (FBD) in those who consumed caffeinated beverages (Klein, Harris, Ferrer, & Zajac, 2011). The rationale was that if those who are self-affirmed are more likely to attend to messages and become sensitive to their strength, then self-affirmation should result in stronger inclinations to change behavior in response to strong messages. Messages of high strength about the link between caffeine consumption and fibrocystic disease cited reputable organizations and sources that supported the link; messages of low strength cited less well-known organizations and mentioned sources that did not support the link. Significant interactions between message strength and self-affirmation were found for feelings of vulnerability (i.e., worry) and intentions to reduce caffeine intake, such that these were higher in those who were self-affirmed and were exposed to the strong message. Furthermore, feelings of vulnerability were found to mediate the effect on behavior change intentions, suggesting that attention to message strength may be facilitated by self-affirmation's effect on worry.

An additional consideration in the effectiveness of self-affirmation is the characteristics (i.e., disposition, life history, or affect) of those who are exposed to health messages. Importantly, if individual differences do interact with self-affirmation's effects, examining only main effects may result in some important effects being obscured, if moderating effects are not

examined (Düring & Jessop, 2015). For instance, people are continually exposed to conflicting health information in their everyday lives, and this can result in confusion and feelings of fatalism about such things as the causes of cancer (Niederdeppe & Gurmankin Levy, 2007). For individuals who perceive high levels of ambiguity about a health topic, health messages may be especially threatening because, in addition to the disconcerting nature of the information, any additional information may only increase this sense of ambiguity (Klein, Hamilton, Harris, & Han, 2015). Self-affirmation may be particularly useful in reducing defensiveness and facilitating receptivity to health messages in people who feel confused by health recommendations. This idea was tested in a study that exposed women with varying levels of risk and perceptions of ambiguity about the causes of cancer to a clear, unambiguous, and authoritative article describing the connection between high alcohol consumption and increased breast cancer risk (Klein et al., 2015). Self-affirmation increased message acceptance in those who perceived high levels of ambiguity in extant cancer prevention recommendations.

Although self-affirmation can be experimentally induced, people are also thought to exhibit tendencies to spontaneously self-affirm (Pietersma & Dijkstra, 2012). These tendencies are assessed by items such as “When I feel threatened or anxious, I find myself thinking about my strengths” (Harris, Napper, Griffin, Schuz, & Stride, in press, cited in Ferrer et al., 2015). Such tendencies have been found to moderate the relationship between anticipated (but not current) negative affect and intentions to obtain genetic test results for diseases that did not have any medically actionable precautionary measures, such as Huntington disease (Ferrer et al., 2015). Similarly, spontaneous self-affirmation and optimism moderated the tendencies of those who tend to avoid information to have lowered intentions to learn results for such diseases (Taber et al., 2015).

In addition to individual differences in the tendency to spontaneously self-affirm, another potential moderating variable of self-affirmation’s effects is self-esteem. To the extent that those with low self-esteem have fewer resources to draw upon when presented with messages that threaten the sense of self, self-affirmation may prove especially beneficial. Accordingly, those with low self-esteem who were affirmed had more positive attitudes toward behavioral change (increasing exercise), stronger endorsement of intentions to exercise, and lower levels of message derogation, but were no different in terms of behavioral control or actual behavior change (Düring & Jessop, 2015).

So, considering moderating characteristics of individuals or health messages is promising, and looking beyond main effects of self-affirmation can prove illuminating. Some further considerations include whether self-affirmation’s effects for those who perceive high levels of ambiguity extend to receptivity to, for instance, more ambiguous messages (Klein et al., 2015). Weaker messages may also be more ambiguous; for example, in the study cited above, the strong message read: “The link between FBD and breast cancer is well-established and FBD has been recognized as a breast cancer risk factor by the Centers for Disease Control and Prevention,” whereas the weak message read: “At a recent meeting of the American Academy of Osteopathic Medicine, a small group of dieticians with links to the fast food industry issued a formal statement supporting the link between FBD and breast cancer as well as the link between caffeine and FBD” (p. 1239). Given the potential effects of perceptions of ambiguity, it could be important to disentangle the effects of message strength and level of ambiguity.

Combining Self-Affirmation With Other Intervention Strategies

Another approach to expanding research on self-affirmation has been to combine self-affirmation with other health behavior change techniques. For example, implementation intentions

(“if-then” plans that concretely specify how to reach a particular goal; Gollwitzer, 1999) are thought to be useful in channeling the motivation generated via self-affirmation into volition and actual behavior change (Jessop, Sparks, Buckland, Harris, & Churchill, 2014; van Dijk & Dijkstra, 2014). Accordingly, some research has begun investigating the effectiveness of incorporating this technique to enhance the effectiveness of self-affirmation.

Armitage and colleagues (Armitage et al., 2011; Armitage, Rowe, Arden, & Harris, 2014) developed a self-affirmation implementation intention intervention to reduce alcohol consumption in adolescents. Forming implementation intentions for alcohol consumption may help individuals plan appropriate behavioral responses when critical situations are encountered, such as being offered a drink. A goal in the design of this intervention was to affirm the self without essay writing or questionnaire elaboration, which depends on good verbal fluency, in order to make it more broadly implementable. Accordingly, participants completed the stem, “If I feel threatened or anxious, then I will...” with one of four options that involved thinking about things they valued in themselves, remembering things they have succeeded in, or thinking about things they stood for, before viewing a diagram depicting the body parts and conditions affected by alcohol consumption. In a first study, this briefer intervention was as effective as a more traditional self-affirmation manipulation and more effective than a control condition in reducing alcohol consumption 1 month later (Armitage et al., 2011), and, in a second study, more effective than a control condition 2 months later (Armitage et al., 2014).

Harris et al. (2014) examined the independent and synergistic effects of self-affirmation and implementation intentions with a 2×2 study design. There was a significant interaction between the two techniques at the 7-day but not the 3-month follow-up, such that self-affirmed participants who formed implementation intentions prior to being exposed to a message emphasizing the benefits of eating fruits and vegetables consumed significantly more. Conversely, however, in a series of two studies with a similar 2×2 design, combining self-affirmation and implementation intentions resulted in a *negative* effect on exercise behavior at 1 week (Jessop et al., 2014). The authors suggested that the counterintuitive finding may have stemmed from the combined techniques producing conflicting construal levels of the behavior or counteracting types of information processing.

Other research has examined whether self-affirmation interacts with aspects of the health message, such as the framing of health information in terms of gains or losses. One study tested the combined effects of self-affirmation and message framing on intentions to engage in indoor tanning (Mays & Zhao, 2016). Because indoor tanning, despite being a significant risk factor for skin cancer and melanoma, is believed to be related to the sense of self and feelings of attractiveness, self-affirmation was thought to be a particularly appropriate technique to prevent defensiveness to loss-framed messages. Loss-framed messages (that emphasize the costs of tanning) may be more threatening than gain-framed messages (that emphasize the benefits of not tanning). After completing a self-affirmation or a control task, participants viewed an image with an accompanying message that emphasized either the risks of indoor tanning or the benefits of avoiding indoor tanning.

For both intentions to indoor tan and intentions to quit indoor tanning, contrary to expectations, there was no interaction between the framing of the message and self-affirmation. In addition, although there were main effects for message framing, with loss-framed messages being more effective, the main effect for self-affirmation was only significant for intentions to tan, with those in the self-affirmation conditions reporting *stronger* intentions to tan. An explanation for this puzzling finding was suggested by the fact that self-affirmation led to perceptions of lower argument strength. The authors speculated that because indoor tanning is so tied to the self-concept, the self-affirmation manipulation could have increased the salience of the importance of indoor tanning and, thus, exacerbated defensiveness to the

messages. This finding also brings attention to the notion that the type of target behavior, and its relevance to sense of self, may be an important consideration in self-affirmation research.

In a rare example of using self-affirmation techniques in clinical samples, patients with cardiopulmonary disease (angioplasty, hypertension, and asthma) were exposed to a positive affect and self-affirmation intervention, in addition to a patient education workbook that the control groups also received, in three randomized trials (Mancuso et al., 2012; Ogedegbe et al., 2012; Peterson et al., 2012). The self-affirmation induction involved thinking of something participants had done that they were proud of and the suggestion that reflecting on these could help them overcome challenges in engaging in a new health behavior. In this instance, the self-affirmation intervention was intended, in combination with techniques aimed to increase positive affect, such as thinking about small things that made them feel good, to overcome barriers to behavioral change rather than to reduce defensiveness to a message. Related to the point made earlier, the trials with different populations had somewhat different target behaviors, which also yielded different effects: in the angioplasty patients the intervention resulted in increased physical activity relative to the control group; in the hypertensive patients the intervention resulted in increased medication adherence relative to the control group; and in the asthmatic patients the intervention and control groups were no different in terms of physical activity. It is also important to note that, where the interventions were effective, the extent to which the effects can be attributed to the self-affirmation manipulation versus the components intended to create positive affect is not clear.

In sum, the emerging evidence regarding the combination of self-affirmation with other intervention techniques is mixed, such that the research on even the most commonly studied added technique, implementation intentions, shows both increases and decreases in effectiveness. However, the literature is diverse, with different target behaviors, study populations, follow-up time points, and even types of self-affirmation inductions, which may contribute to this lack of uniformity. Considering aspects of the message itself (e.g., framing) may be especially worthwhile, as these vary in the literature also, and, as most messages are framed either positively or negatively, giving explicit attention to this is worthwhile. Finally, some authors have pointed out that self-affirmation may play only a small role in the larger behavior change process, among other techniques, such as self-monitoring and identifying barriers, so continuing to consider its role in combined interventions is useful, despite the mixed evidence that has emerged thus far (van Dijk & Dijkstra, 2014).

Having highlighted some of the recent developments among studies of self-affirmation and health-related outcomes, we will now turn to discussing some of the major research questions that remain for future research.

How Does Self-Affirmation Lead to Health Behavior Change?

Although numerous studies have indicated that self-affirmation increases acceptance of health information and facilitates behavior change, surprisingly little is known about *how* it impacts health outcomes. To date, researchers have focused primarily on people's self-reported responses to health information as a source of potential mediators. For example, one possibility is that self-affirmation changes people's attitudes toward health information (e.g., by increasing perceptions of risk), which, in turn, leads to changes in health behavior. Although there is some preliminary evidence that an increase in perceived vulnerability mediates the effect of self-affirmation on behavioral intentions (Klein et al., 2011), other studies have found

that neither attitudes toward the health behavior nor perceived threat (Armitage et al., 2014) mediates the effect of self-affirmation on health behavior.

Another possibility is that self-affirmation engenders a change in intentions, which, in turn, leads to a change in behavior. Supporting this prediction, one study found that among people who are at a higher risk for developing type 2 diabetes, intentions to take an online diabetes risk test mediated the effect of self-affirmation on risk test participation (van Koningsbruggen & Das, 2009). Other studies of self-affirmation, however, have failed to find a mediating effect of intentions on behavior change (Armitage et al., 2014). Further casting doubt on the viability of behavioral intentions as a mediator, several studies have found that self-affirmation does not always elicit a positive change in behavioral intentions (Harris et al., 2014; Reed & Aspinwall, 1998). Among studies of self-affirmation measuring both health intentions and behavior, one meta-analytic review found that intention effect sizes did not predict behavior effect sizes (Sweeney & Moyer, 2015), suggesting that a change in behavioral intentions does not translate to a comparable change in behavior. Taken together, limited research supports a causal intention–behavior relation among self-affirmation studies.

Whereas much of the extant research has focused on the role of single potential mediating variables, future research may examine whether self-affirmation influences health outcomes through multiple variables. Few self-affirmation studies have adopted a path analysis approach to examine whether a set of variables helps to explain the impact of self-affirmation on health behavior. One exception is by Armitage, Harris, Hepton, and Napper (2008) who found that health message acceptance mediated the relation between self-affirmation and intentions and intentions mediated the relation between message acceptance and behavior. Self-affirmation has been applied to health behaviors that vary across a number of dimensions, including the type of behavior (i.e., health promoting vs. health damaging) and the temporal impact of the behavior (i.e., immediate vs. distant consequences). Given this variability, in addition to the inherent complexities associated with long-term health behavior change, future research may consider adopting an analytic approach that examines a set of variables and takes into account variables specific to the targeted health behavior.

Another avenue for future research may involve testing mediating variables that extend beyond people's immediate self-reported responses to health information. Several studies have suggested that self-affirmation facilitates a broader perspective from which to view information (Critcher & Dunning, 2015; Sherman et al., 2013). For example, when people are self-affirmed, they are more likely to think about actions in terms of their abstract, superordinate aspects (e.g., why to improve one's health), rather than in terms of their concrete, subordinate aspects (e.g., how to improve one's health; Sherman et al., 2013).

In the context of health behaviors, a broader perspective may be helpful for several reasons. For example, threatening information may be experienced as less aversive when viewed from a distance (Sherman, 2013). Offering preliminary support for this prediction, one study tested whether thinking in broad (vs. narrow) terms would influence tanners' receptivity toward health information on the risks of tanning (Belding, Naufel, & Fujita, 2015). Tanners led to think that broad, superordinate categories were more motivated to reduce their risk of skin cancer than tanners led to think in narrow, subordinate categories. These authors suggest that thinking in broad terms promotes long-term self-change motivation, rather than short-term self-protection motivation. Relatedly, when a threat or temptation does not loom quite so largely, a broadened perspective may make it easier for people to exert self-control. Several studies have indicated that people are better at practicing self-control when led to think in broad (vs. narrow) terms, as evidenced, for example, by differences in preferences for healthy versus unhealthy foods (Fujita & Han, 2009).

Another possibility is that a broadened perspective helps people to connect their current actions to their long-term aspirations. Supporting this prediction, past research has found that construing information in broad (vs. narrow) terms facilitates attention toward one's long-term goals when faced with reminders of goal-related temptations (Fujita & Sasota, 2011). Such research suggests, for example, that a dieter who adopts a broader perspective may find it easier to bring to mind his or her dieting goal when faced with tempting food. Furthermore, other research has found that a broad (vs. narrow) perspective helps people to recognize the commonalities across their various life goals (Clark & Freitas, 2013). As a result, a broadened perspective may make it easier to connect a targeted health behavior (e.g., eating more fruits and vegetables) with one's long-term goals (e.g., living a long and happy life).

Having established that self-affirmation has the potential to be a useful tool for promoting health behavior change, an important next step for researchers is to develop an empirically supported mechanistic account of self-affirmation. Given that people's immediate self-reported responses to health information do not appear to mediate the effect of self-affirmation, future research may consider other ways in which self-affirmation impacts the self and the consequences this has for evaluation, motivation, and goal-directed action. Identifying the mechanisms that underlie self-affirmation effects may help to further refine self-affirmation theory as a whole and also offers important practical benefits, such as increasing understanding of the specific conditions under which self-affirmation is most effective.

What Is the Trajectory of Self-Affirmation?

Much of the research on self-affirmation and health has focused on the immediate changes that occur after self-affirming, such as changes in message acceptance. However, measures of people's deliberate responses to health information after self-affirming have yielded some inconsistent findings. As noted previously, for example, self-affirmation does not always lead to a positive change in behavioral intentions (e.g., Harris et al., 2014; Jessop et al., 2014). Similarly, other studies have failed to find a significant effect of self-affirmation on outcomes such as perceived behavioral control, perceived threat, message derogation, or self-efficacy (Armitage et al., 2008, 2014; Jessop et al., 2014). Although most studies have assessed whether self-affirmation elicits immediate changes in health-related cognitions, one recent study measured intentions and attitudes immediately after participants read a health message and again during a 1-week follow-up. There were no immediate differences in intentions and attitudes between the self-affirmation and control group; however, the self-affirmation group did express greater intentions and attitudes 1 week later.

Such findings may lead one to wonder whether self-affirmation elicits an immediate change or is the effect gradual? In a meta-analytic review Epton et al. (2015) found that self-affirmation exerts a smaller effect on outcomes that are measured immediately after encountering health information (i.e., message acceptance) than measures that typically involve some delay (i.e., behavior). Importantly, however, the time of measurement of a health behavior (e.g., days vs. weeks) has not been found to moderate the effect of self-affirmation on behavior (Epton et al., 2015; Sweeney & Moyer, 2015). Furthermore, as reviewed previously, a recent study using fMRI found that when people were self-affirmed, they engaged in greater self-related processing, as reflected through activity in the VMPFC, while reading health information (Falk et al., 2015). Additionally, neural activity during message processing predicted changes in health behavior that were distinct from changes predicted from participants' self-reports of behavioral intentions and attitudes. These findings suggest that self-affirmation

enacts immediate effects on the self (as reflected through participant's neural activity) that relate to subsequent behavior change.

Taken together, self-affirmation appears to exert some immediate influences, as evidenced, for example, by studies showing that self-affirmation produces an immediate change in physiological responses to threat (e.g., Creswell et al., 2005) and in neural activity (e.g., Falk et al., 2015). However, the observed heterogeneity in people's self-reported responses suggests that self-affirmation may not elicit a change in health-related cognitions that can be reliably captured at a conscious level. That is, some of the influence of self-affirmation may occur outside of people's conscious awareness.

Increased attention is being given to understanding how health-related decisions are shaped by both conscious and unconscious processes (Sheeran, Gollwitzer, & Bargh, 2013). To date, relatively few studies have examined the implicit effects of self-affirmation in relation to health outcomes. One exception is Klein and Harris (2009) who used a dot probe task to examine whether self-affirmation enhances people's tendency to direct their attention toward threatening health information. After completing a self-affirmation or control task and reading a message about the risks of alcohol consumption, female participants completed a task in which a neutral and a threatening word from the health message were presented simultaneously and followed by a dot. Participants were asked to quickly identify whether the dot appeared on the left or right side of the screen. Self-affirmed participants were faster to identify the location of the dot when it appeared after a threat-related word, suggesting that self-affirmed participants displayed a stronger attentional bias for threat-related words than non-affirmed participants. Whereas other studies have used self-report measures to assess differences in perceived threat, this methodology allowed for a novel test of the extent to which self-affirmation elicits differences in implicit processing of health information.

Another exception is van Koningsbruggen, Das, and Roskos-Ewoldsen (2009) who used a lexical decision-making task to examine whether self-affirmation increases responsiveness toward health information at an implicit level. After completing a self-affirmation or control task and reading a message about the risks of caffeine, coffee drinkers and non-coffee drinkers completed a response time task that required them to distinguish between non-words, neutral words, and threat-related words (e.g., "heart disease"). Among coffee drinkers, those who self-affirmed were faster at recognizing threat-related words than non-affirmed individuals, suggesting self-affirmation increased accessibility of threat-related cognitions among people for whom the health message was most relevant.

These two studies provide initial support for the potential usefulness of implicit measures in studies of self-affirmation and health. In light of recent research suggesting that people may not always be able to reliably report on conscious changes that arise after self-affirmation, future research may consider adopting a framework that encompasses both conscious and unconscious processes. Increasingly health behavior change models are adopting a dual-systems approach that incorporates both reflective and automatic health-related processes (Sheeran et al., 2013). Whereas much of the extant research has focused on conscious changes arising from self-affirmation, future research is needed to further elucidate the nonconscious influences of self-affirmation.

Conclusions

Research on self-affirmation and health outcomes has grown extensively in recent decades. Building upon studies that highlighted the potential of self-affirmation as a tool for reducing

defensive processing, numerous studies have been devoted to understanding the different types of health-related cognitions that are influenced by self-affirmation and any implication this may have for health behavior change. In this chapter, we highlighted three recent developments in this area of research including the adoption of a wide array of methodological approaches, emphasis on identifying moderating variables, and efforts to combine self-affirmation with other behavior change techniques. By drawing attention to the need for developing a mechanistic account of self-affirmation and the need to further our understanding the trajectory of self-affirmation, we hope this chapter will encourage future investigations of self-affirmation as a tool for health behavior change.

Author Biographies

Allison M. Sweeney is a postdoctoral researcher at the University of South Carolina. Integrating theories and methods across health, social, and cognitive psychology, her research focuses on developing theory-based interventions for promoting health behavior change.

Anne Moyer is an associate professor of social and health psychology at Stony Brook University. Her research focuses on psychosocial factors surrounding cancer and cancer risk, medical decision making, research methodology and research synthesis, and the psychological aspects of human research participation.

References

- Armitage, C. J., Harris, P. R., & Arden, M. A. (2011). Evidence that self-affirmation reduces alcohol consumption: Randomized exploratory trial with a new, brief, means of self-affirming. *Health Psychology, 30*, 633–641.
- Armitage, C. J., Harris, P. R., Hepton, G., & Napper, L. (2008). Self-affirmation increases acceptance of health-risk information among United Kingdom adult smokers with low socioeconomic status. *Psychology of Addictive Behaviors, 22*, 88–95.
- Armitage, C. J., Rowe, R., Arden, M. A., & Harris, P. R. (2014). A brief psychological intervention that reduces adolescent alcohol consumption. *Journal of Consulting and Clinical Psychology, 82*, 546–550.
- Belding, J. N., Naufel, K. Z., & Fujita, K. (2015). Using high-level construal and perceptions of change-ability to promote self-change over self-protection motives in response to negative feedback. *Personality and Social Psychology Bulletin, 41*, 822–838.
- Clark, S., & Freitas, A. (2013). Construing action abstractly and perceiving consonance among goal pursuits: Implications for activity substitutability and the accessibility of activity-goal links. *Motivation and Emotion, 37*, 537–549.
- Creswell, J. D., Welch, W. T., Taylor, S. E., Sherman, D. K., Gruenewald, T. L., & Mann, T. (2005). Affirmation of personal values buffers neuroendocrine and psychological stress responses. *Psychological Science, 16*, 846–851.
- Critcher, C. R., & Dunning, D. (2015). Self-affirmations provide a broader perspective on self-threat. *Personality and Social Psychology Bulletin, 41*, 3–18.
- Ditto, P., & Lopez, D. (1992). Motivated skepticism: Use of differential decision criteria for preferred and nonpreferred conclusions. *Journal of Personality and Social Psychology, 63*, 568–584.
- Düring, C., & Jessop, D. C. (2015). The moderating impact of self-esteem on self-affirmation effects. *British Journal of Health Psychology, 20*, 274–289.
- Epton, T., & Harris, P. (2008). Self-affirmation promotes health behavior change. *Health Psychology, 27*, 746–752.

- Epton, T., Harris, P. R., Kane, R., van Koningsbruggen, G. M., & Sheeran, P. (2015). The impact of self-affirmation on health-behavior change: A meta-analysis. *Health Psychology, 34*, 187–196.
- Falk, E. B., O'Donnell, M. B., Cascio, C. N., Tinney, F., Kang, Y., Lieberman, M. D., ... Strecher, V. J. (2015). Self-affirmation alters the brain's response to health messages and subsequent behavior change. *Proceedings of the National Academy of Sciences, 112*, 1977–1982.
- Ferrer, R. A., Taber, J. M., Klein, W. M. P., Harris, P. R., Lewis, K. L., & Biessecker, L. G. (2015). The role of current affect, anticipated affect and spontaneous self-affirmation in decisions to receive self-threatening information. *Cognition and Emotion, 29*(8), 1456–1465.
- Fujita, K., & Han, H. A. (2009). Moving beyond deliberative control of impulses: The effect of construal levels on evaluative associations in self-control conflicts. *Psychological Science, 20*, 799–804.
- Fujita, K., & Sasota, J. A. (2011). The effects of construal levels on asymmetric temptation-goal cognitive associations. *Social Cognition, 29*, 125–146.
- Gallagher, K. M., & Updegraff, J. A. (2012). Health message framing effects on attitudes, intentions and behavior: A meta-analytic review. *Annals of Behavioral Medicine, 43*, 101–116.
- Giner-Sorolla, R., & Chaiken, S. (1997). Selective use of heuristic and systematic processing under defense motivation. *Personality and Social Psychology Bulletin, 23*, 84–97.
- Gollwitzer, P. M. (1999). Implementation intentions: Strong effects of simple plans. *American Psychologist, 54*, 493–503.
- Harris, P. R., Brearley, I., Sheeran, P., Barker, M., Klein, W. M. P., Creswell, J. D., ... Bond, R. (2014). Combining self-affirmation with implementation intentions to promote fruit and vegetable consumption. *Health Psychology, 33*, 729–736.
- Jessop, D. C., Sparks, P., Buckland, N., Harris, P. R., & Churchill, S. (2014). Combining self-affirmation and implementation intentions: Evidence of detrimental effects on behavioral outcomes. *Annals of Behavioral Medicine, 47*, 137–147.
- Klein, W. M. P., Hamilton, J. G., Harris, P. R., & Han, P. K. (2015). Health messaging to individuals who perceive ambiguity in health communications: The promise of self-affirmation. *Journal of Health Communication, 20*, 566–572.
- Klein, W. M. P., & Harris, P. R. (2009). Self-affirmation enhances attentional bias toward threatening components of a persuasive message. *Psychological Science, 20*, 1463–1467.
- Klein, W. M. P., Harris, P. R., Ferrer, R. A., & Zajac, L. E. (2011). Feelings of vulnerability in response to threatening messages: Effects of self-affirmation. *Journal of Experimental Social Psychology, 47*, 1237–1242.
- Legault, L., Al-Khindi, T., & Inzlicht, M. (2012). Preserving integrity in the face of performance threat: Self-affirmation enhances neurophysiological responsiveness to errors. *Psychological Science, 23*, 1455–1460.
- Liberman, A., & Chaiken, S. (1992). Defensive processing of personally relevant health messages. *Personality and Social Psychology Bulletin, 18*, 669–679.
- Mancuso, C. A., Choi, T. N., Westermann, H., Wenderoth, S., Hollenberg, J. P., Wells, M. T., ... Charlson, M. E. (2012). Increasing physical activity in patients with asthma through positive affect and self-affirmation: A randomized trial. *Archives of Internal Medicine, 173*, 337–343.
- Mays, D., & Zhao, X. (2016). The influence of framed messages and self-affirmation on indoor tanning behavioral intentions in 18- to 30-year-old women. *Health Psychology, 35*(2), 123–130.
- Niederdeppe, J., & Gurmankin Levy, A. (2007). Fatalistic beliefs about cancer prevention and three prevention behaviors. *Cancer Epidemiology, Biomarkers & Prevention, 16*, 998–1003.
- Ogedegbe, G. O., Boutin-Foster, C., Wells, M. T., Allegrante, J. P., Isen, A. M., Jobe, J. B., & Charlson, M. E. (2012). A randomized controlled trial of positive-affect intervention and medication adherence in hypertensive African Americans. *Archives of Internal Medicine, 172*, 322–326.
- Peterson, J. C., Charlson, M. E., Hoffman, Z., Wells, M. T., Wong, S. C., Hollenberg, J. P., ... Allegrante, J. P. (2012). A randomized controlled trial of positive-affect induction to promote physical activity after percutaneous coronary intervention. *Archives of Internal Medicine, 172*, 329–336.
- Pietersma, S., & Dijkstra, A. (2012). Cognitive self-affirmation inclination: An individual difference in dealing with self-threats. *British Journal of Social Psychology, 51*, 33–51.

- Reed, M. B., & Aspinwall, L. G. (1998). Self-affirmation reduces biased processing of health-risk information. *Motivation and Emotion*, 22, 99–132.
- Schmeichel, B. J., & Tang, D. (2015). Individual differences in executive functioning and their relationship to emotional processes and responses. *Current Directions in Psychological Science*, 24, 93–98.
- Sheeran, P., Gollwitzer, P. M., & Bargh, J. A. (2013). Nonconscious processes and health. *Health Psychology*, 32, 460–473.
- Sherman, D., & Cohen, G. (2006). The psychology of self-defense: Self-affirmation theory. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (pp. 183–242). San Diego, CA: Academic Press.
- Sherman, D., Nelson, L., & Steele, C. (2000). Do messages about health risks threaten the self? Increasing the acceptance of threatening health messages via self-affirmation. *Personality and Social Psychology Bulletin*, 26, 1046–1058.
- Sherman, D. K. (2013). Self-affirmation: Understanding the effects. *Social and Personality Psychology Compass*, 11, 834–845.
- Sherman, D. K., Hartson, K. A., Binning, K. R., Purdie-Vaughns, V., Garcia, J., Taborsky-Barba, S., ... Cohen, G. L. (2013). Deflecting the trajectory and changing the narrative: How self-affirmation affects academic performance and motivation under identity threat. *Journal of Personality and Social Psychology*, 104, 591–618.
- Steele, C. M. (1988). The psychology of self-affirmation: Sustaining the integrity of the self. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (pp. 261–302). San Diego, CA: Academic Press.
- Sweeney, A. M., & Moyer, A. (2015). Self-affirmation and responses to health messages: A meta-analysis on intentions and behavior. *Health Psychology*, 34, 149–159.
- Taber, J. M., Klein, W. M. P., Ferrer, R. A., Lewis, K. L., Harris, P. R., Sheppard, J. A., & Biesecker, L. G. (2015). Information avoidance tendencies, threat management resources, and interesting in genetic sequencing feedback. *Annals of Behavioral Medicine*, 49(4), 616–621.
- van Dijk, S., & Dijkstra, A. (2014). Adding power or losing strength? Searching for strategies on how self-affirmation may grow stronger: A comment on Jessop et al. *Annals of Behavioral Medicine*, 47, 131–132.
- van Koningsbruggen, G., & Das, E. (2009). Don't derogate the message! Self-affirmation promotes online type 2 diabetes risk test taking. *Psychology & Health*, 24, 635–649.
- van Koningsbruggen, G., Das, E., & Roskos-Ewoldsen, D. (2009). How self-affirmation reduces defensive processing of threatening health information: Evidence at the implicit level. *Health Psychology*, 28, 563–568.
- Wileman, V., Farrington, K., Chilcot, J., Norton, S., Wellsted, D. M., Almond, M. K., ... Armitage, C. J. (2014). Evidence that self-affirmation improves phosphate control in hemodialysis patients: A pilot cluster randomized controlled trial. *Annals of Behavioral Medicine*, 48, 275–281.

Suggested Reading

- Cohen, G. L., & Sherman, D. K. (2014). The psychology of change: Self-affirmation and social psychological intervention. *Annual Review of Psychology*, 65, 333–371.
- Harris, P. R., & Epton, T. (2009). The impact of self-affirmation on health cognition, health behaviour and other health-related responses: A narrative review. *Social and Personality Psychology Compass*, 3, 962–978.

Stigma of Disease and Its Impact on Health

Lindsay Sheehan and Patrick Corrigan

Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA

For people who experience chronic diseases and health conditions, stigma threatens to compound the physical and mental manifestations of illness, leading to unfavorable health and life outcomes. Particularly stigmatizing conditions such as HIV/AIDS (Farber, Shahane, Brown, & Campos, 2014), lung cancer (Chambers et al., 2015), irritable bowel syndrome (IBS; Taft, Ballou, & Keefer, 2013), drug addiction (Barry, McGinty, Pescosolido, & Goldman, 2014), and mental illness (Parcesepe & Cabassa, 2013) have been targets of research on the stigma of disease. This paper defines stigma types, explains various mechanisms by which stigma can impact health, and explores strategies for changing stigma.

Erving Goffman (1963) first described stigma as a discrediting mark that leads the stigmatized person to feel shame and hide the condition from others. More recently, four necessary components of stigma are described by Link and Phelan (2001). First, a stigmatized group or individual is recognized and labeled as different from others. Next, society perceives these differences as negative. Third, an “us” versus “them” (the stigmatized) mentality is established. Finally, the stigmatized person or group is subject to discrimination and loss of social power. Stigma may include stereotypes (thoughts), prejudice (feelings), and discrimination (unfair behaviors) toward individuals or groups from the stigmatizing condition. For example, a person with HIV may be stereotyped as careless, irresponsible, or morally inferior; may experience prejudice in the form of being feared by others; and may be discriminated against when family members avoid social contact. Stereotypes that comprise stigma are culturally created and disease specific, such that health conditions are connected with unique stereotypes that elicit specific prejudice and discrimination. The stereotype of incompetence in schizophrenia leads to discriminatory behavior such as employers not hiring them. When people with lung cancer are viewed as culpable in causing the illness through smoking, others will be less likely to provide help and support.

Types of Stigma

Several types of stigma have the potential to impact health and are illustrated in Table 1. Thus far, we have described public stigma, the stigma that is perpetrated by members of the public toward the stigmatized individual. This stigma can impact the individual through direct experience (“experienced stigma”) in which the person is treated in unfair ways such as being denied housing (Thornicroft et al., 2009). Public stigma also impacts individuals through mere knowledge or fear of the stigma that could be perpetrated against them (“anticipated stigma”) (Thornicroft et al., 2009). In anticipated stigma, a person who experiences addiction worries how others will treat him or her if they know about the addiction. Stigmatized individuals may also self-stigmatize, whereby they internalize the public perceptions and believe that they really *are* as incompetent as society believes them to be (Corrigan, Watson, & Barr, 2006). The stigmatized individual may cognitively and emotionally give in to the “why try” effect, thinking that efforts toward improving health outcomes are futile (“I’m so stupid, why should I even bother taking my medications”). Not surprisingly, internalized stigma is linked to depression, low self-esteem, and reduced self-efficacy (Corrigan, Larson, & Rüsch, 2009).

Two other types of stigma are important to examine in the context of health psychology: associative stigma and structural stigma. If Fred’s partner has HIV, Fred’s employer might assume he has HIV as well and limit his career advancement opportunities. Sarah’s mother might be judged as incompetent when Sarah is diagnosed with ADHD. In associative stigma, family members, friends, or other acquaintances are tarnished by stigma through their connections to the stigmatized individual. In response to associative stigma, family or friends may distance themselves from their loved one to avoid actual or perceived negative societal reactions. Associative stigma can also apply to health workers who treat stigmatized conditions (Verhaeghe & Bracke, 2012). Mental health workers who provide services to “those crazy people” experience less respect and social status than those working in other helping professions.

Table 1 Stigma types, definitions, and examples.

<i>Type of stigma</i>	<i>Definition</i>	<i>Example</i>
Public stigma	Unfair beliefs, attitudes, or behaviors directed at individuals or groups	Jasper finds out his neighbor Bob has schizophrenia. He feels scared and starts avoiding Bob
Experienced stigma	Stigma experienced by the individual, usually through discrimination	An employer refuses to hire a person with HIV
Anticipated (“felt”) stigma	Stigma felt when stigmatized person fears negative reactions from others	Renee is worried about what others will think if she reveals her addiction
Internalized stigma/ self-stigma	Stigma that occurs when the stigmatized person agrees with and applies the public stigma to themselves	George feels he is just a worthless overweight person who does not deserve a girlfriend
Associative stigma	Stigma experienced by associates of the stigmatized group/individual (family, friends, acquaintances, service providers, etc.)	Ariel’s sister is shunned by neighbors
Structural stigma	Stigma perpetrated through social systems or institutions, either intentionally or unintentionally	Funding for substance abuse treatment is substantially less than for other diseases

In some cases, whole systems fail a stigmatized group. Institutional policies that restrict opportunities of the stigmatized group result in structural stigma (Angermeyer, Matschinger, Link, & Schomerus, 2014). Structural stigma may be intentional, such as laws that limit parental rights of people with mental illness, or unintentional, such as inequalities in research funding for stigmatized conditions (Corrigan et al., 2005). In both instances, stereotypes and prejudices about the group—beliefs that people with mental illness are, for example, dangerous or incompetent—lead to discriminatory policies resulting in exclusion, restriction, or denial of rightful opportunities.

The Impact of Stigma

Disease stigma works through numerous mechanisms and has serious consequences for health. First, stigma has direct effects on psychological health: the experience of stigma is stressful. In a meta-analysis examining the connection between perceptions of discrimination and psychological well-being, average effect sizes in the small to medium range (overall $r = -.23$) were found with self-esteem, depression, anxiety, psychological distress, and life satisfaction (Schmitt, Branscombe, Postmes, & Garcia, 2014). Although chronic health conditions elicit stress and depression on their own, stigma-related distress and depression have been uncovered in people with HIV (Earnshaw, Smith, Cunningham, & Copenhaver, 2015), lung cancer (Chambers et al., 2015), and IBS (Taft et al., 2013).

Second, stigma interferes with care seeking. Affected individuals may engage in a process of label avoidance, whereby they forgo, delay, or minimally engage in treatment in order to elude the label of the condition (“I do not want to be seen walking into an HIV clinic!”) (Corrigan, Druss, & Perlick, 2014). Although there are many reasons that people might not seek treatment, stigma is certainly a factor in preventing 40% of people with serious mental illness from engaging in care (Substance Abuse and Mental Health Services Administration [SAMHSA], 2012) and the 12% of people living with undiagnosed HIV (Valdiserri, 2002). Individuals might avoid entering treatment because of the stigma of diagnosis and in later stages of treatment might avoid visiting healthcare providers or following treatment regimen to prevent being seen by others. Associative stigma experienced by family members or close friends can make social support network unsupportive of treatment efforts and lead to early treatment dropout (Corrigan, Druss, et al., 2014). In addition, health providers can themselves engage in label avoidance. Medical residents, for example, have stigma-related concerns about addictions, mental health diagnoses, and STDs, such that 18% of residents delayed or avoided treatment for themselves (Dunn, Hammond, & Roberts, 2009).

Once sick individuals decide to seek treatment, quality of that treatment is also impacted by stigma (Harangozo et al., 2014). Stigma perpetrated by health providers is especially harmful when it creates a non-nurturing, coercive, or paternalistic environment that drives patients from care or restricts choices for treatment. Structural stigma leads to clinics that fail to train providers in the respectful and culturally competent practices, poor availability of services, lack of access to health insurance, or other hurdles in obtaining care. Taken together, these factors greatly influence decisions of individuals to stay in treatment.

Fourth, stigma influences illness, self-management, and treatment adherence. The distress associated with stigma can make comprehension of medical instructions and follow-up with healthcare recommendations more challenging. People who are preoccupied with stigma stress will have less cognitive resources available for adhering to treatment regimens (Major, Hunger, Bunyan, & Miller, 2014). When exposed to stigma, individuals have more difficulty

self-regulating, including reduced information processing, sustained attention, and persistence on task (Richman & Lattanner, 2014). Also, label avoidance restricts access to self-management resources, and a desire to hide illness status can interfere with treatment. Fearing reactions from supervisors or coworkers, a person with HIV might avoid bringing medications to work. A person with mental illness or addiction will not be comfortable requesting time off work for treatment appointments when such requests would necessitate disclosure. Internalized stigma is another barrier to treatment self-management (Livingston & Boyd, 2010). Depression and lack of self-efficacy occur when a person self-stigmatizes, leading to lack of recovery efforts (“Why even go see a counselor? I’m just a worthless addict who will never recover”). In fact, adults with chronic illness who self-stigmatize or who have experienced stigma in the past from health workers expect future stigma-laden interactions, avoid care, and have a lower quality of life (Earnshaw & Quinn, 2012).

Stigma also harms people through the breakdown of social relationships. Acts of discrimination typically involve social avoidance, exclusions, or microaggressions that serve to damage relationships. Landlords are hesitant to rent to people with schizophrenia, friends avoid inviting the person with IBS out to dinner, or acquaintances use underhanded remarks to people with addiction (“you still cannot kick the habit, huh?”). These interactions chip away at personal relationships and social networks. The absence of social support during an already difficult period of illness causes further stress and limits social resources needed to engage in treatment (e.g., help driving to doctor appointment). Internalized or perceived stigma results in the person withdrawing from others due to feelings of worthlessness (“Now that I have lung cancer, I have only myself to blame, and no one should have to help me”).

Finally, structural stigma has a broad influence on health outcomes. Disparities in research funding, programmatic funding, and insurance coverage for stigmatizing diseases such as mental illness and addiction create fewer quality treatment options that are less accessible for those that would use the services (Angermeyer et al., 2014). People with chronic illness and disability are too often institutionalized despite their capacity for independent living. Given adequate community supports and financial assistance, some individuals in skilled care facilities could live in more independent settings; however, a dearth of supportive services and transition services have continued the segregation of people with disabilities in nursing homes. Legislation and court decisions such as the Americans with Disabilities Act (1990) and *Olmstead v. L.C.* (1999) challenge structural stigma by mandating civil rights for people with disabilities to live and work in the community. Likewise, the Mental Health Parity Act (2008) and the Affordable Care Act have expanded insurance coverage for mental illness. However, insurance coverage concerns continue to plague people with mental illness and especially those seeking addiction treatment (Jones, Campopiano, Baldwin, & McCance-Katz, 2015).

Moderators of Stigma

Several factors moderate disease stigma, including perceived responsibility for illness, fear, and familiarity. Stigma is greater when the person is viewed as having responsibility for the onset of illness (“onset responsibility”) or as having control over improving or ending the illness (“offset responsibility”) (Weiner, 1995). People with HIV, addiction, and lung cancer are viewed as highly responsible for the onset of the illness, whereas those with addiction are seen as responsible for both onset and offset (Corrigan et al., 2002). Obesity, while not a

disease in itself, is a highly stigmatizing condition with high perceived responsibility both for onset and offset. Conditions such as heart disease and diabetes that have been connected with obesity and are seen as preventable also have high perceived responsibility on the part of the affected person.

Greater stigma results when the public views the particular groups as dangerous. For example, people with mental illness are seen as dangerous and unpredictable, resulting in public fears and discrimination (Janulis, Ferrari, & Fowler, 2013). Fear of contagion is also a factor in stigma related to illness such as in H1N1 influenza and SARS (Williams, Gonzalez-Medina, & Le, 2011), potentially leading to ostracism and reduced opportunities for treatment.

Familiarity may be inversely related to stigma (Broussard, Goulding, Talley, & Compton, 2012). Those who have personally experienced the disease, who have a family member with the disease, or who are otherwise familiar with the disease tend to perpetrate less stigma. When community members or family members have a personal connection, they can see the impacted individual as a person, rather than as a member of the stigmatized group. Stigma is especially harmful however when it is perpetrated by family and close friends, as it reduces the person's social and economic resources.

Combating Stigma

Research linking familiarity with stigma is the basis for anti-stigma interventions that use education about disease and contact with impacted individuals to change attitudes and behaviors toward the stigmatized group. Educational interventions provide information that counteracts stigmatizing attitudes (e.g., Power Point presentation outlining myths about mental illness), whereas a contact intervention involves an interaction between a person with the illness and those in the intervention condition (e.g., person with HIV gives presentation about personal experience with HIV stigma). In adults, anti-stigma programs that promote meaningful contact between people impacted by the disease and members of the public seem most powerful. A meta-analysis examining stigma change interventions for people with mental illness found change effect sizes for changes in attitudes and intended behavior ranging from .1 to .3 (Corrigan, Morris, Michaels, Rafacz, & Rüsch, 2012). However, it is important to note that few studies have been able to capture actual changes in behavior that result from anti-stigma interventions.

Not all approaches are effective. In the mental illness literature, biogenetic explanations for illness were introduced to reduce stigma reducing the perceived responsibility of the person ("it's not the person with schizophrenia's fault that he's ill, it's a brain disorder"). However, this approach risks highlighting the differences between the stigmatized individual and society while downplaying possibilities for recovery. A meta-analysis found that biogenetic explanations for mental illness were associated with less blame but that perceptions of dangerousness were greater and that the public desired more social distance from people with mental illness (Kvaale, Haslam, & Gottdiener, 2013).

Anti-stigma approaches that target health providers may improve health outcomes. These programs should ideally target all levels of health provider (physicians, nurses, dieticians, psychologists, case managers, etc.) and aim to reduce negative reactions as well as promote positive or affirming reactions toward people with a health condition (Sheehan, 2015). Whereas more traditional anti-stigma messages have attempted to reduce negative attitudes, advocates have called for more affirming messages focused on recovery and empowerment of

the stigmatized person (Corrigan, Powell, & Michaels, 2014). Research on mental illness stigma interventions aimed at health providers find that those using both education and contact are effective (Stubbs, 2014).

Efforts to reduce internalized stigma (i.e., self-stigma) have focused on helping the stigmatized individual identifying the impact stigma has on his/her life and identifying ways to change personal reactions toward stigma. Interventions to enhance resistance to internalized stigma include peer support groups, peer education, and cognitive behavioral type interventions (Taft et al., 2013). Efforts to explicitly address internalized stigma have only recently begun to be tested, showing some potential for reducing the impact of stigma (Conner, McKinnon, Ward, Reynolds, & Brown, 2015).

In addition to stigma of the primary health condition, service providers must recognize and address multiple levels and types of stigma. Racial, ethnic, or sexual minority stigma can interact with and compound stigma related to the disease condition. Thus, health providers working in an HIV clinic should be sensitive to both stigma related to HIV and stigma related to sexual orientation. Health providers should understand cultural differences in disease-related stigma and the impact these have on minority patients. Latinos being treated for addiction have different cultural stigmas than do Blacks or Asians. An individualized approach that is sensitive to the complex needs of the person in treatment will help to better engage those of minority status.

Also of concern are co-occurring disease-related stigmas such as those of mental illness and obesity. Obesity stigma affects many individuals within the patient population, and stigma perpetrated by health providers can be a barrier to physical healthcare (Teixeira & Budd, 2010). The stress associated with obesity-related stigma is implicated in a cycle of increasing weight gain (Tomiya, 2014), and internalized obesity stigma is recognized as an important concern (Ratcliffe & Ellison, 2015). Recognition of various types of stigma (public, internalized, associative, and structural) points healthcare providers to specific solutions for addressing each.

Conclusion

Primary prevention, early intervention, and prevention of physical deterioration and impact are primary concerns of health psychology (Boyer, 2008). Stigma affects the individuals on all of these levels by increasing vulnerability to psychological problems, creating barriers to care seeking, reducing quality of care, reducing self-management capabilities of the patient, and compromising social support systems. While interventions to combat stigma exist, these can be more thoroughly and systematically implemented and tested throughout the healthcare system.

Author Biographies

Lindsay Sheehan is a senior research associate at the Illinois Institute of Technology where she conducts research on mental illness stigma, suicide stigma, and health disparities for individuals with serious mental illness. She is a managing editor of the American Psychological Association journal, *Stigma and Health*. Dr. Sheehan is also a licensed counselor and has experience working in community mental health.

Patrick Corrigan is a distinguished professor of psychology at the Illinois Institute of Technology. Dr. Corrigan leads the National Consortium on Stigma and Empowerment

(NCSE), which focuses on increasing self-determination and empowerment for people with psychiatric disability. Dr. Corrigan has written more than 300 peer-reviewed articles and is an editor of *Stigma and Health*. Corrigan has authored or edited 15 books, most recently, *The Stigma of Disease and Disability*.

References

- Angermeyer, M. C., Matschinger, H., Link, B. G., & Schomerus, G. (2014). Public attitudes regarding individual and structural discrimination: Two sides of the same coin? *Social Science & Medicine*, 103, 60–66.
- Barry, C. L., McGinty, E. E., Pescosolido, B. A., & Goldman, H. H. (2014). Stigma, discrimination, treatment effectiveness, and policy: Public views about drug addiction and mental illness. *Psychiatric Services*, 65(10), 1269–1272.
- Boyer, B. A. (2008). Theoretical models of health psychology and the model for integrating medicine and psychology. In M. I. Paharia & B. A. Boyer (Eds.), *Comprehensive handbook of clinical health psychology* (pp. 3–30). Hoboken, NJ: Wiley.
- Broussard, B., Goulding, S. M., Talley, C. L., & Compton, M. T. (2012). Social distance and stigma toward individuals with schizophrenia: Findings in an urban, African-American community sample. *Journal of Nervous and Mental Disease*, 200(11), 935–940.
- Chambers, S. K., Baade, P., Youl, P., Aitken, J., Occhipinti, S., Vinod, S., ... Zorbas, H. (2015). Psychological distress and quality of life in lung cancer: The role of health-related stigma, illness appraisals and social constraints. *Psycho-Oncology*, 24(11), 1569–1577.
- Conner, K. O., McKinnon, S. A., Ward, C. J., Reynolds, C. F., III, & Brown, C. (2015). Peer education as a strategy for reducing internalized stigma among depressed older adults. *Psychiatric Rehabilitation Journal*, 38(2), 186–193.
- Corrigan, P. W., Druss, B. G., & Perlick, D. A. (2014). The impact of mental illness stigma on seeking and participating in mental health care. *Psychological Science in the Public Interest*, 15(2), 37–70.
- Corrigan, P. W., Larson, J. E., & Rüsch, N. (2009). Self-stigma and the “why try” effect: Impact on life goals and evidence-based practices. *World Psychiatry*, 8(2), 75–81.
- Corrigan, P. W., Morris, S. B., Michaels, P. J., Rafacz, J. D., & Rüsch, N. (2012). Challenging the public stigma of mental illness: A meta-analysis of outcome studies. *Psychiatric Services*, 63(10), 963–973.
- Corrigan, P. W., Powell, K. J., & Michaels, P. J. (2014). Brief battery for measurement of stigmatizing versus affirming attitudes about mental illness. *Psychiatry Research*, 215(2), 466–470.
- Corrigan, P. W., Rowan, D., Green, A., Lundin, R., River, P., Uphoff-Wasowski, K., ... Kubiak, M. A. (2002). Challenging two mental illness stigmas: Personal responsibility and dangerousness. *Schizophrenia Bulletin*, 28(2), 293.
- Corrigan, P. W., Watson, A. C., & Barr, L. (2006). Understanding the self-stigma of mental illness. *Journal of Social and Clinical Psychology*, 25(8), 875–884.
- Corrigan, P. W., Watson, A. C., Heyrman, M. L., Warpinski, A., Gracia, G., Slopen, N., & Hall, L. L. (2005). Structural stigma in state legislation. *Psychiatric Services*, 56(5), 557–563.
- Dunn, L. B., Hammond, K. A. G., & Roberts, L. W. (2009). Delaying care, avoiding stigma: Residents’ attitudes toward obtaining personal health care. *Academic Medicine*, 84(2), 242–250.
- Earnshaw, V. A., & Quinn, D. M. (2012). The impact of stigma in healthcare on people living with chronic illnesses. *Journal of Health Psychology*, 17(2), 157–168.
- Earnshaw, V. A., Smith, L. R., Cunningham, C. O., & Copenhaver, M. M. (2015). Intersectionality of internalized HIV stigma and internalized substance use stigma: Implications for depressive symptoms. *Journal of Health Psychology*, 20(8), 1083–1089.
- Farber, E. W., Shahane, A. A., Brown, J. L., & Campos, P. E. (2014). Perceived stigma reductions following participation in mental health services integrated within community-based HIV primary care. *AIDS Care*, 26(6), 750–753.

- Goffman, E. (1963). *Stigma: Notes on the management of spoiled identity*. Englewood Cliffs, NJ: Prentice-Hall.
- Harangozo, J., Reneses, B., Brohan, E., Sebes, J., Csukly, G., López-Ibor, J. J., ... Thornicroft, G. (2014). Stigma and discrimination against people with schizophrenia related to medical services. *International Journal of Social Psychiatry*, 60(4), 359–366.
- Janulis, P., Ferrari, J. R., & Fowler, P. (2013). Understanding public stigma toward substance dependence. *Journal of Applied Social Psychology*, 43(5), 1065–1072.
- Jones, C. M., Campopiano, M., Baldwin, G., & McCance-Katz, E. (2015). National and state treatment need and capacity for opioid agonist medication-assisted treatment. *American Journal of Public Health*, 105(8), e55–e63.
- Kvaale, E. P., Haslam, N., & Gottdiener, W. H. (2013). The ‘side effects’ of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clinical Psychology Review*, 33(6), 782–794.
- Link, B. G., & Phelan, J. C. (2001). Conceptualizing stigma. *Annual Review of Sociology*, 27, 363–385.
- Livingston, J. D., & Boyd, J. E. (2010). Correlates and consequences of internalized stigma for people living with mental illness: A systematic review and meta-analysis. *Social Science & Medicine*, 71(12), 2150–2161.
- Major, B., Hunger, J. M., Bunyan, D. P., & Miller, C. T. (2014). The ironic effects of weight stigma. *Journal of Experimental Social Psychology*, 51, 74–80.
- Parcesepe, A. M., & Cabassa, L. J. (2013). Public stigma of mental illness in the United States: A systematic literature review. *Administration and Policy in Mental Health and Mental Health Services Research*, 40(5), 384–399.
- Ratcliffe, D., & Ellison, N. (2015). Obesity and internalized weight stigma: A formulation model for an emerging psychological problem. *Behavioural and Cognitive Psychotherapy*, 43(2), 239–252.
- Richman, L. S., & Lattanner, M. R. (2014). Self-regulatory processes underlying structural stigma and health. *Social Science & Medicine*, 103, 94–100.
- Schmitt, M. T., Branscombe, N. R., Postmes, T., & Garcia, A. (2014). The consequences of perceived discrimination for psychological well-being: A meta-analytic review. *Psychological Bulletin*, 140(4), 921.
- Sheehan, L. (2015). *Strategies for changing the stigma of behavioral healthcare*. Commissioned paper for the National Academy of Sciences: Committee on the science of changing behavioral health norms. Retrieved from http://sites.nationalacademies.org/DBASSE/BBCSS/DBASSE_170049
- Stubbs, A. (2014). Reducing mental illness stigma in health care students and professionals: A review of the literature. *Australasian Psychiatry*, 22(6), 579–584.
- Substance Abuse and Mental Health Services Administration. (2012). *Results from the 2011 National Survey on Drug Use and Health: Mental health findings* (NSDUH Series H-45, HHS Publication No. [SMA] 12-4725). Rockville, MD: Author.
- Taft, T., Ballou, S., & Keefer, L. (2013). A preliminary evaluation of internalized stigma and stigma resistance in inflammatory bowel disease. *Journal of Health Psychology*, 18(4), 451–460.
- Teixeira, M. E., & Budd, G. M. (2010). Obesity stigma: A newly recognized barrier to comprehensive and effective type 2 diabetes management. *Journal of the American Academy of Nurse Practitioners*, 22(10), 527–533.
- Thornicroft, G., Brohan, E., Rose, D., Sartorius, N., Leese, M., & INDIGO Study Group. (2009). Global pattern of experienced and anticipated discrimination against people with schizophrenia: A cross-sectional survey. *The Lancet*, 373(9661), 408–415.
- Tomiya, A. J. (2014). Weight stigma is stressful. A review of evidence for the Cyclic Obesity/Weight-Based Stigma model. *Appetite*, 82, 8–15.
- Valdiserri, R. O. (2002). HIV/AIDS stigma: An impediment to public health. *American Journal of Public Health*, 92(3), 341.
- Verhaeghe, M., & Bracke, P. (2012). Associative stigma among mental health professionals: Implications for professional and service user well-being. *Journal of Health and Social Behavior*, 53(1), 17–32.

- Weiner, B. (1995). *Judgment of responsibility: A foundation for a theory of social conduct*. New York, NY: Guilford Press.
- Williams, J., Gonzalez-Medina, D., & Le, Q. (2011). Infectious diseases and social stigma. *Applied Technologies & Innovations*, 4(1), 58–70.

Suggested Reading

- Corrigan, P. W., Druss, B. G., & Perlick, D. A. (2014). The impact of mental illness stigma on seeking and participating in mental health care. *Psychological Science in the Public Interest*, 15(2), 37–70.
- Earnshaw, V. A., & Quinn, D. M. (2012). The impact of stigma in healthcare on people living with chronic illnesses. *Journal of Health Psychology*, 17(2), 157–168.
- Valdiserri, R. O. (2002). HIV/AIDS stigma: An impediment to public health. *American Journal of Public Health*, 92(3), 341.

Alcohol Use Disorder: Overview

Brittany E. Blanchard, Angela K. Stevens, Molin Shi,
and Andrew K. Littlefield

Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Definitions of Alcohol Use Disorder

Initially, alcohol use disorder (AUD), labeled *alcoholism*, was classified under the personality and other nonpsychotic disorders sections in the first two editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), and this personality-based conceptualization of AUDs remained prominent in mental health until 1980 (American Psychiatric Association [APA], 1980). Although the disease concept of alcoholism, which led to the medicalization of this disorder, appeared in 1956 (see Jellinek, 1960), it was not until the third edition of the DSM that “alcoholism” was replaced with alcohol abuse and dependence and was moved to the substance use disorder section (APA, 1980). These changes, in part, reflected the conceptualization of alcohol dependence syndrome (see Edwards & Gross, 1976).

Recently, the fifth edition of the DSM drastically changed the classification of alcohol-related diagnoses (and other substance use disorders). In addition to abolishing the biaxial system in favor of single AUD, which combines abuse and dependence criteria, critical changes to criteria were made based on research conducted since the publication of DSM-IV (e.g., removal of legal issues and addition of craving; APA, 2013; see Hasin et al., 2013). Under DSM-5 criteria, an individual must exhibit at least 2 of the 11 criteria in the past year (i.e., briefly, hazardous use, difficulty quitting, social/occupational problems, tolerance, withdrawal, drinking for larger/longer than intended, great deal of time spent obtaining/using/recovering from alcohol, craving, failure to fulfill role obligations, continued drinking despite interpersonal/social problems, continued drinking despite psychological/physical problems; see APA, 2013), and these symptoms must yield significant distress and/or impairment for diagnosis (APA, 2013, p. 491). A severity specifier allows clinicians to rate severity of the AUD based on a symptom count (APA, 2013).

Because only one symptom was necessary to establish an alcohol abuse diagnosis in DSM-IV (APA, 2000), the latest criteria change (i.e., 2+ symptoms required) may improve diagnostic

reliability and validity by reducing the number of diagnostic imposters (Lane & Sher, 2015). Although the newest edition of the *DSM* has made great improvements in classification of individuals with AUD for clinical and research purposes, several limitations remain (e.g., varying severity among symptoms, disregard for symptom constellations; see Lane & Sher, 2015). One proposal to rectify this issue is to require both harm (e.g., presence of alcohol-related problems) and dysfunction (e.g., presence of either compulsive use or withdrawal) to make an AUD diagnosis (Wakefield & Schmitz, 2015).

Prevalence of AUDs

Based on data from the 2012 to 2013 National Epidemiologic Survey on Alcohol and Related Conditions III, a representative US noninstitutionalized civilian sample of adults 18 years or older, the lifetime and 12-month prevalence of *DSM-5* AUD were 29.1 and 13.9%, respectively (Grant et al., 2015). Both lifetime and past-year rates of AUD were highest among males, White and Native American individuals, and younger, never married, or previously married adults (Grant et al., 2015). Normatively, the prevalence of AUD and levels of heavy, problematic alcohol use peak in the early 20s and decline thereafter into later adulthood, although heterogeneity exists in trajectories of alcohol outcomes (see Chassin, Sher, Hussong, & Curran, 2013).

Etiological Influences

A myriad of theoretical conceptualizations that attempt to explain the mechanisms responsible for the onset, maintenance, and relapse processes of AUD have been proffered (e.g., Cox & Klinger, 1988; Sher, Grekin, & Williams, 2005). Extant literature highlights multiple etiological pathways for problematic alcohol involvement. Importantly, different pathways are not mutually exclusive, and there is potential for both additive and superadditive effects (see Sher, Martinez, & Littlefield, 2011, for a detailed review). Most widely accepted models in research and clinical work utilize the biopsychosocial framework, which posits that biological (e.g., genetics, neuroadaptation, pharmacological vulnerability; see Kendler, Myers, & Prescott, 2007; Sher, Trull, Bartholow, & Vieth, 1999; Volkow, Koob, & McLellan, 2016), psychological (e.g., expectancies, motives, personality; see Jones, Corbin, & Fromme, 2001; Kuntsche, Knibbe, Gmel, & Engels, 2005; Littlefield & Sher, 2014), and social factors (e.g., parental and peer influences; Nash, McQueen, & Bray, 2005) contribute to the onset and maintenance of AUD (Marlatt, Baer, Donovan, & Kivlahan, 1988). For example, the motivational model of alcohol use postulates that historical factors (biochemical reactivity, personality, and sociocultural factors), current factors (current affective state and alcohol availability), and alcohol-related expectancies interact to affect decision-making processes regarding alcohol consumption (Cox & Klinger, 1988). Diathesis–stress models (including genetic diathesis; see Dick et al., 2008) propose that individuals have biological and/or psychological vulnerabilities, which trigger the onset of AUD in the face of stressful life events (Goldstein, Abela, Buchanan, & Seligman, 2000). Supporting this model are findings from gene–environment interaction research, which suggests that individuals with specific genetic variants develop AUD only under certain environmental conditions (Caspi & Moffitt, 2006).

Social cognitive theories of alcohol use integrate psychological factors (e.g., cognitive expectancies, drinking refusal self-efficacy, drinking motives) and social environmental influences (e.g., parental and peer alcohol use) to predict alcohol consumption and AUD development

(Cooper, Russell, & George, 1988; Giovazolias & Themeli, 2014). The deviance proneness model (Sher et al., 1999) suggests that substance use behavior is a facet of a general pattern of deviant behaviors (i.e., heterotypic continuity; Moffitt, 1993). In addition to the genetic vulnerability of externalizing behaviors in childhood, these behaviors are also thought to be a result of environmental factors such as inadequate socialization and/or associating with a deviant peer group. In sum, multiple integrative models exist within the literature, the most comprehensive of which include examination biological, psychological, and sociocultural/environmental factors in considering etiological pathways to AUD, as well as alcohol consumption, more broadly.

Definitions of Alcohol Consumption

Alcohol consumption is necessary to experience alcohol-related problems and receives a diagnosis of AUD, yet this is not assessed in the *DSM-5* or *the International Classification of Diseases (ICD-10)* (World Health Organization, 1992). Despite exclusion from formal criteria for AUDs, alcohol quantity, frequency, and “binge drinking” are typically assessed. Although there are discrepancies in definitions of binge drinking (also called heavy episodic drinking), the current standard is the National Institute on Alcohol Abuse and Alcoholism (NIAAA) definition, which is five or more standard drinks for males and four or more standard drinks for females, over approximately 2 hr. This pattern of consumption increases blood alcohol concentration (BAC) to roughly 0.08 g/dL, the legal limit in the United States (NIAAA, 2004). Other definitions include that of the Substance Abuse and Mental Health Services Administration (SAMHSA), which defines binge drinking as more than five drinks per occasion, with heavy drinking defined as binge drinking on 5 or more days within the past month (Center for Behavioral Health Statistics and Quality, 2015).

Acute Effects of Alcohol Consumption

Effects of acute alcohol consumption can range from increased talkativeness and relaxation to clumsiness, to, at higher levels of intoxication, respiratory depression, and even death (see Vonghia et al., 2008). Broadly, effects tend to vary as a function of BAC with more severe effects occurring at higher levels of BAC. For example, relaxation is typically experienced by individuals with a BAC of less than 0.05 g/dL, whereas symptoms like respiratory depression and coma occur at BACs of 0.40 g/dL and higher (Vonghia et al., 2008). Although BAC typically increases with consumption, an individual’s BAC depends on a number of factors affecting bioavailability, such as genetics, biological sex, eating prior to the drinking episode, and alcohol tolerance (e.g., Ramchandani, Bosron, & Li, 2001). Regardless, within a given drinking episode, an individual will typically experience both stimulant-like and depressant-like effects, due to alcohol’s biphasic effects (i.e., stimulant-like effects on the ascending limb of the blood alcohol curve and depressant-like effects on the descending limb). However, the experience of these effects also varies as a function of individual differences (e.g., alcohol use status; King, De Wit, McNamara, & Cao, 2011).

Acute alcohol consumption can impact a number of physiological functions, which can influence health outcomes. For example, evidence suggests that alcohol consumption often yields increases in appetite and food consumption (Yeomans, 2010). Although evidence is mixed regarding the effects of alcohol intake on sexual arousal, research indicates a decrease in genital arousal, including decreased penile tumescence (see George & Stoner, 2000). Contrary to popular belief, consuming alcohol before bed may negatively impact sleep; more specifically, alcohol appears to delay the onset of rapid eye movement (REM) sleep and increase disruption

in the second half of sleep (see Ebrahim, Shapiro, Williams, & Fenwick, 2013). Many individuals also experience hangovers after consuming alcohol, which may include symptoms such as headache, nausea, vomiting, diarrhea, fatigue, and decreased motor and cognitive functioning (Wiese, Shlipak, & Browner, 2000). In some cases hangover may negatively impact or prevent performance of work-related responsibilities.

Higher doses of alcohol (i.e., BAC of approximately 0.10 g/dL or higher) can impair reaction times and decision making (Vonghia et al., 2008), resulting in an increased chance of risky sexual behaviors, sexual assault, physical injury, homicidal and suicidal behaviors, and motor vehicle accidents (Cooper, 2002; see George & Stoner, 2000). Indeed, the National Highway Traffic Safety Administration (U.S. Department of Transportation, National Highway Traffic Safety Administration, 2015) estimates that approximately 28 people die each day in the United States in alcohol-related motor vehicle accidents. In sum, at lower doses, alcohol typically produces rewarding effects, such as relaxation and reductions in anxiety, whereas higher doses yield depressant-like effects and increase the risk of alcohol-related consequences. In light of this evidence, to maximize the short-term effects of alcohol and minimize consequences, it is recommended that individuals consume alcohol in a manner consistent with the 2015–2020 Dietary Guidelines for Americans, which defines moderate consumption as no more than two drinks per day for men and one drink a day for women of the legal drinking age, with some exceptions (e.g., those taking certain medications or with certain conditions, pregnant or breastfeeding women; US Department of Health and Human Services and US Department of Agriculture, 2015).

Author Biographies

Brittany E. Blanchard is currently a PhD student in clinical psychology at Texas Tech University in the Department of Psychological Sciences. Her research examines individual differences in alcohol and substance use, including use of protective behavioral strategies.

Angela K. Stevens is currently a PhD student in clinical psychology at Texas Tech University in the Department of Psychological Sciences. Broadly, her research interests include individual differences in adult problematic substance use.

Molin Shi is currently a PhD student in clinical psychology at Texas Tech University in the Department of Psychological Sciences. Her research focuses on the influences and consequences of substance-related behaviors, with an emphasis on the familial, social, and acculturative impact of substance use in minority subgroups.

Andrew K. Littlefield is currently an assistant professor at Texas Tech University in the Department of Psychological Sciences. He obtained his predoctoral training in clinical psychology at the University of Missouri and completed his clinical internship at the University of Mississippi Medical Center. Dr. Littlefield's research focuses on addictive behaviors.

References

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

- Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience*, 7(7), 583–590.
- Chassin, L., Sher, K. J., Hussong, A., & Curran, P. (2013). The developmental psychopathology of alcohol use and alcohol disorders: Research achievements and future directions. *Development and Psychopathology*, 25, 1567–1584.
- Cooper, M. L. (2002). Alcohol use and risky sexual behavior among college students and youth: Evaluating the evidence. *Journal of Studies on Alcohol*, 63, 101–117.
- Cooper, M. L., Russell, M., & George, W. H. (1988). Coping, expectancies, and alcohol abuse: A test of social learning formulations. *Journal of Abnormal Psychology*, 92, 218–230.
- Cox, W. M., & Klinger, E. (1988). A motivational model of alcohol use. *Journal of Abnormal Psychology*, 97, 168–180.
- Dick, D. M., Aliev, F., Wang, J. C., Grucza, R. A., Schuckit, M., Kuperman, S., ... Hesselbrock, V. (2008). Using dimensional models of externalizing psychopathology to aid in gene identification. *Archives of General Psychiatry*, 65, 310–318.
- Ebrahim, I. O., Shapiro, C. M., Williams, A. J., & Fenwick, P. B. (2013). Alcohol and sleep I: Effects on normal sleep. *Alcoholism: Clinical and Experimental Research*, 37(4), 539–549.
- Edwards, G., & Gross, M. M. (1976). Alcohol dependence: Provisional description of a clinical syndrome. *British Medical Journal*, 1(6017), 1058–1061.
- George, W. H., & Stoner, S. A. (2000). Understanding acute alcohol effects on sexual behavior. *Annual Review of Sex Research*, 11(1), 92–124.
- Giovazolias, T., & Themeli, O. (2014). Social learning conceptualization for substance abuse: Implications for therapeutic interventions. *The European Journal of Counseling Psychology*, 3(1), 69–88.
- Goldstein, B. I., Abela, J. R., Buchanan, G. M., & Seligman, M. E. (2000). Attributional style and life events: A diathesis-stress theory of alcohol consumption. *Psychological Reports*, 87(3), 949–955.
- Grant, B. F., Goldstein, R. B., Saha, T. D., Chou, S. P., Jung, J., Zhang, H., ... Hasin, D. S. (2015). Epidemiology of DSM-5 alcohol use disorder: Results from the National Epidemiologic Survey on Alcohol and Related Conditions III. *JAMA Psychiatry*, 72(8), 757–766.
- Hasin, D. S., O'Brien, C. P., Auriacombe, M., Borges, G., Bucholz, K., Budney, A., ... Schuckit, M. (2013). DSM-5 criteria for substance use disorders: Recommendations and rationale. *American Journal of Psychiatry*, 170(8), 834–851.
- Jellinek, E. M. (1960). *The disease concept of alcoholism*. Oxford, England: Hillhouse.
- Jones, B. T., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction*, 96(1), 57–72.
- Kendler, K. S., Myers, J., & Prescott, C. A. (2007). Specificity of genetic and environmental risk factors for symptoms of cannabis, cocaine, alcohol, caffeine, and nicotine dependence. *Archives of General Psychiatry*, 64(11), 1313–1320.
- King, A. C., De Wit, H., McNamara, P. J., & Cao, D. (2011). Rewarding, stimulant, and sedative alcohol responses and relationship to future binge drinking. *Archives of General Psychiatry*, 68(4), 389–399.
- Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2005). Why do young people drink? A review of drinking motives. *Clinical Psychology Review*, 25, 841–861.
- Lane, S. P., & Sher, K. J. (2015). Limits of current approaches to diagnosis severity based on criterion counts: An example with DSM-5 alcohol use disorder. *Clinical Psychological Science*, 3(6), 819–835.
- Littlefield, A. K., & Sher, K. J. (2014). Personality and substance use disorders. In K. J. Sher (Ed.), *Handbook of substance use and substance use disorders*. New York, NY: Oxford University Press.
- Marlatt, G. A., Baer, J. S., Donovan, D. M., & Kivlahan, D. R. (1988). Addictive behaviors: Etiology and treatment. *Annual Review of Psychology*, 39(1), 223–252.
- Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, 100(4), 674–701.
- Nash, S. G., McQueen, A., & Bray, J. H. (2005). Pathways to adolescent alcohol use: Family environment, peer influence, and parental expectations. *Journal of Adolescent Health*, 37(1), 19–28.

- National Institute on Alcohol Abuse and Alcoholism. (2004, Winter). Council approve definition of binge drinking. *National Institute of Alcohol Abuse and Alcoholism Newsletter*, (3), 3.
- Ramchandani, V. A., Bosron, W. F., & Li, T. K. (2001). Research advances in ethanol metabolism. *Pathologie Biologie*, 49(9), 676–682.
- Sher, K. J., Grekin, E. R., & Williams, N. A. (2005). The development of alcohol use disorders. *Annual Review of Clinical Psychology*, 1, 493–523.
- Sher, K. J., Martinez, J. A., & Littlefield, A. K. (2011). Alcohol use disorders. In D. H. Barlow (Ed.), *Handbook of clinical psychology* (pp. 405–445). New York, NY: Oxford University Press.
- Sher, K. J., Trull, T. J., Bartholow, B., & Vieth, A. (1999). Personality and alcoholism: Issues, methods, and etiological processes. In H. Blane & K. Leonard (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 55–105). New York, NY: Plenum.
- U.S. Department of Health and Human Services and U.S. Department of Agriculture. (2015). *2015–2020 Dietary Guidelines for Americans* (8th ed.). Retrieved from <http://health.gov/dietaryguidelines/2015/guidelines/>
- U.S. Department of Transportation, National Highway Traffic Safety Administration. (2015). *Drunk driving: Overview*. Retrieved from <https://www.nhtsa.gov/risky-driving/drunk-driving>
- Volkow, N. D., Koob, G. F., & McLellan, A. T. (2016). Neurobiologic advances from the brain disease model of addiction. *New England Journal of Medicine*, 374(4), 363–371.
- Vonghia, L., Leggio, L., Ferrulli, A., Bertini, M., Gasbarrini, G., Addolorato, G., & Alcoholism Treatment Study Group. (2008). Acute alcohol intoxication. *European Journal of Internal Medicine*, 19(8), 561–567.
- Wakefield, J. C., & Schmitz, M. F. (2015). The harmful dysfunction model of alcohol use disorder: Revised criteria to improve the validity of diagnosis and prevalence estimates. *Addiction*, 110(6), 931–942.
- Wiese, J. G., Shlipak, M. G., & Browner, W. S. (2000). The alcohol hangover. *Annals of Internal Medicine*, 132(11), 897–902.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.
- Yeomans, M. R. (2010). Alcohol, appetite and energy balance: Is alcohol intake a risk factor for obesity? *Physiology & Behavior*, 100(1), 82–89.

Suggested Reading

- Ramchandani, V. A., Bosron, W. F., & Li, T. K. (2001). Research advances in ethanol metabolism. *Pathologie Biologie*, 49(9), 676–682.
- Sher, K. J., Grekin, E. R., & Williams, N. A. (2005). The development of alcohol use disorders. *Annual Review of Clinical Psychology*, 1, 493–523.
- Volkow, N. D., Koob, G. F., & McLellan, A. T. (2016). Neurobiologic advances from the brain disease model of addiction. *New England Journal of Medicine*, 374(4), 363–371.

Cancer and Psycho-Oncology

Lauren N. Harris¹ and Annette L. Stanton²

¹*VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA*

²*Department of Psychology, University of California, Los Angeles, CA, USA*

Introduction

Cancer is a collection of diseases characterized by uncontrolled division of abnormal cells. In the United States, cancers of the prostate and breast are the most common cancer sites in men and women, respectively. Lung and colon/rectum cancers are the second and third most common cancers in both sexes. More than 15.5 million cancer survivors are living in the United States today; this figure is projected to rise to more than 20 million by 2026 (American Cancer Society, 2017). Treatment for cancer varies but often includes surgery to remove the tumor, chemotherapy, radiation, targeted biologic therapy, and/or hormonal therapy. Treatments can cause immediate and long-term side effects including pain, nausea, fatigue, sleep disturbance, and infertility.

Cancer mortality has declined 25% over the past two decades, primarily as a result of decreased tobacco use and improvements in cancer prevention, early detection, and treatment (American Cancer Society, 2017). Despite this trend, cancer remains the second leading cause of death in the United States, accounting for approximately 25% of all deaths, and lung cancer is the leading cause of cancer mortality in women and men. Furthermore, although cancer survival has improved overall, racial/ethnic and socioeconomic disparities in cancer deaths remain. US cancer death rates are higher in African Americans than any other racial or ethnic group. Among African Americans, rates of death from cancer are 24% higher in men and 14% higher in women compared with non-Hispanic White men and women (American Cancer Society, 2017). Causes of disparities include a complex combination of economic, sociocultural, biological, environmental, and system-level factors. Contributors to higher cancer death rates among racial/ethnic minorities and economically disadvantaged individuals include inequalities in education, income, wealth, work, housing, and barriers to receiving high-quality cancer-related healthcare services (American Cancer Society, 2017).

As people live longer after cancer diagnosis, psychosocial concerns and quality of life among cancer survivors become an increasingly important focus of research and clinical attention. Research in the field of psycho-oncology examines (a) behavioral and psychosocial factors in the initiation and progression of cancer, (b) factors influencing adherence to cancer screening recommendations for early detection, (c) the biopsychosocial experience and trajectory of psychosocial adjustment to cancer, and (d) predictors of psychosocial adjustment to cancer (Holland et al., 2015). Research in psycho-oncology is also dedicated to developing and implementing psychosocial and behavioral interventions to promote health and well-being following cancer diagnosis (see *Cancer and Psychosocial Interventions* entry). Reviews of the literature suggest that these interventions, which include approaches such as cognitive behavioral therapy, relaxation, coping skills training, and supportive-expressive therapy, are effective in improving psychological functioning, fatigue, pain, and other psychological and physical health outcomes (e.g., Faller et al., 2013). This article focuses on adults with cancer; for information regarding the unique needs and concerns of children with cancer, refer to the *Childhood Cancer* entry.

Behavioral and Psychosocial Factors in the Initiation and Progression of Cancer

Behavioral factors are implicated in both cancer initiation (i.e., development of cancer in individuals with no previous tumor) and progression (i.e., cancer growth and/or spread in patients with existing disease). Indeed, more than half of cancer cases worldwide could be prevented by reducing known risk factors, most of which are behavioral (American Association for Cancer Research, 2017). Behavioral risk factors for the development of cancer include tobacco use, obesity, poor-quality diet, physical inactivity, excessive alcohol intake, risky sexual behavior, and insufficient sun protection (Spring, King, Pagoto, Van Horn, & Fisher, 2015). Although risk factors vary by cancer type, known behavioral factors associated with cancer progression, recurrence, and/or mortality include obesity, physical inactivity, poor-quality diet, tobacco use, and nonadherence to recommended cancer treatments (Gillison et al., 2012; Hershman et al., 2011; Rock et al., 2012). Cancer survivors are advised to achieve or maintain a normal weight, exercise regularly, stop smoking, and eat a healthy diet, which have the potential to improve prognosis (Brandon, Unrod, & Simmons, 2015; Rock et al., 2012).

Researchers in psycho-oncology seek to develop effective interventions to reduce the risk of cancer initiation and improve outcomes following cancer diagnosis. Interventions can target multiple levels of influences on behavior, from public policy initiatives to changing sociocultural contexts and intervening on an individual level (Spring et al., 2015). The reduction in tobacco use in the United States over the past several decades, for instance, is largely attributable to public policy and environmental interventions such as anti-smoking campaigns, clean indoor air laws, and price deterrents. Effective individual-level interventions for quitting smoking also exist, including behavioral counseling, social support, and pharmacotherapy. Despite effective interventions to reduce smoking prevalence, tobacco use remains responsible for nearly one-third of cancer deaths in the United States each year, and cancer deaths related to tobacco use continue to climb in low-income countries (American Cancer Society, 2017).

Historically, it was believed that individuals with particular personality types were more likely to develop cancer. However, no psychological factor has been demonstrated to promote

the initiation of cancer (Lutgendorf & Andersen, 2015). The majority of research examines the role of psychosocial factors in the progression, rather than initiation, of cancer. Factors implicated in cancer progression include depression, distress, trauma history, and social isolation (Lutgendorf & Andersen, 2015). Psychological research has identified plausible mechanisms through which these factors may promote disease progression. For instance, psychosocial stressors can trigger activation of the autonomic nervous system (ANS) and the hypothalamic–pituitary–adrenal (HPA) axis, causing release of stress hormones such as catecholamines and glucocorticoids. Over time, chronic activation of these physiological systems can impair immune functioning and promote inflammation, which has been shown to promote tumor growth, particularly in nonhuman animal models (Lutgendorf & Andersen, 2015). Research is needed to better understand the pathways through which biopsychosocial factors might contribute to tumor initiation, growth, and spread.

Screening for Early Detection of Cancer

Early detection of cancer can improve treatment outcomes and survival. Indeed, cancer screening has contributed to a major public health advance against cancer over the past three decades. The US Preventive Services Task Force (USPSTF) issues recommendations for cervical, breast, and colorectal cancer screening based on age and gender. In issuing screening recommendations, experts must weigh the clear benefits of cancer screening (i.e., early cancer detection and intervention) against potential risks of screening.

Most individuals who are screened for cancer do not have, and will never develop, the disease. Risks of screening large swaths of the population include psychological distress and physical health consequences from follow-up investigations associated with false positives, radiation exposure from screening, and overdiagnosis (i.e., identification of pathology that is nonprogressive but is indistinguishable from progressive disease and therefore treated as progressive; Wardle, Robb, Vernon, & Waller, 2015). For instance, many prostate cancers progress so slowly that they are unlikely to cause harm within a man's lifetime. Early identification of prostate cancer typically prompts treatment, which can cause important untoward side effects. Based on findings from randomized controlled trials, the USPSTF recently recommended against prostate cancer screening through prostate-specific antigen (PSA) testing after concluding that the risks of screening for prostate cancer through PSA measurement outweigh the benefits.

Adherence to cancer screening recommendations is suboptimal, although rates have improved over the past several decades. In 2013, 73% of American women reported mammography, and 81% reported undergoing cervical screening within the recommended period. Only 58% of women and men were up to date with colorectal cancer screening (Sabatino, White, Thompson, & Klabunde, 2015). Rates of cancer screening in the United Kingdom, which are based on national databases and therefore not subject to biased reporting, are similar (Health and Social Care Information Centre, 2013a, 2013b).

Documented barriers to cancer screening include both contextual and individual factors. Contextual factors include absence of a regular physician and/or health insurance, failure of physicians to recommend screening, language barriers, low education, and low social support for screening (Wardle et al., 2015). For these and other reasons, disparities in rates of cancer screening exist: in the United States, individuals of low socioeconomic status and Hispanic Americans are less likely to be up to date with screening than are more advantaged and non-Hispanic White Americans.

Individual-level factors that predict low rates of cancer screening include lack of knowledge about cancer and cancer screening, low perceived risk of cancer, fatalistic beliefs that cancer is incurable, and perception that screening behavior is outside the social norm (Wardle et al., 2015). Research to examine the relationship between worry about cancer and likelihood of undergoing cancer screening is mixed (Hay, Buckley, & Ostroff, 2005). Some studies demonstrate a link between cancer-related worry and higher screening behavior, whereas others find that cancer-related worry predicts lower uptake of screening. The relation between cancer worry and participation in screening may be nonlinear, such that moderate worry facilitates screening, whereas high and low levels of worry inhibit screening (Wardle et al., 2015).

To improve adherence to screening recommendations, many interventions have targeted structural and organizational, rather than individual or psychological, barriers. These strategies include improving access to healthcare, facilitating the establishment of regular relationships with physicians, and implementing public health campaigns to promote awareness about the benefits of early detection through screening. Other effective approaches for improving participation in screening for breast, cervical, and colorectal cancer include reminders for screening and one-on-one education.

Psychosocial Adjustment to Cancer

Cancer is a profound stressor that can cause significant declines in physical, emotional, and social functioning. Most people diagnosed with cancer adjust well, however. Although prevalence estimates of depression and anxiety following cancer diagnosis vary across studies, meta-analyses (e.g., Mitchell et al., 2011) suggest that more than one-third of cancer patients meet diagnostic criteria for depression, anxiety, or an adjustment disorder.

Adjustment Across the Cancer Trajectory

Longitudinal research reveals commonalities in experiences and adjustment along the continuum of the cancer trajectory, from diagnosis to long-term survivorship and/or end of life. Shortly after cancer diagnosis, anxiety is common as patients are faced with uncertainty regarding treatment options and must make difficult decisions related to treatment (Stanton & Bower, 2015). Individuals may also face challenges as they arrange for changes in employment and other life roles.

During active oncologic treatment, many patients experience significant negative side effects such as pain and fatigue (Jacobsen & Andrykowski, 2015). Other common side effects of cancer treatment include sleep disturbance, nausea, and cognitive impairment. Patients also can face challenges in communicating effectively with their medical team and their interpersonal network. During treatment, patients may feel too tired to keep family and friends updated on their health status or feel hesitant to ask for help. Many patients also must navigate changes in work and other responsibilities as they undergo treatment.

During the reentry phase (i.e., the months after primary medical treatment is completed), challenges often include resuming work, familial, and social roles despite persistent or late-emerging side effects of treatment (Stanton, Rowland, & Ganz, 2015). Fatigue, for instance, is a long-term consequence of cancer and its treatment for some. Patients can experience a sense of abandonment from the medical team and other sources of support. Fear of cancer recurrence is also common during the reentry phase. Long-term survivors may have problems

with employment, enduring and late-emerging side effects of treatment, and lasting concerns about future health (Institute of Medicine [IOM], 2005).

Cancer recurrence and diagnosis of metastatic disease (i.e., cancer that has spread to distant organs) present a host of challenges for patients and their loved ones (Rainbird, Perkins, Sanson-Fisher, Rolfe, & Anseline, 2009). Metastatic cancer is usually life limiting. Treatment is often intensive and variable over time. Patients with advanced disease may face compromised role functioning and concerns about losing independence and burdening their loved ones. Patients with metastatic disease must also make difficult treatment-related decisions, which can include determining when to end treatment and prepare for the end of life.

Characterizing the Experience of Cancer

Despite the challenges associated with a diagnosis of cancer, most survivors report finding benefit in the cancer experience (Stanton et al., 2015). Facets of benefit finding, also known as posttraumatic growth, include strengthened interpersonal relationships, enhanced life appreciation, greater focus on life priorities, and a deepened sense of spirituality. Although findings are inconsistent, research suggests that finding benefit can promote improved psychological adjustment among cancer patients, with enduring effects in long-term survivorship (e.g., Bower et al., 2005).

Research also documents the challenges and distress that partners, family members, and friends of cancer patients experience. During the continuum of the cancer experience, caregivers assume varied responsibilities such as assisting patients with activities of daily living, coordinating medical care, providing emotional support, and gathering information. Rates of depression and anxiety among caregivers of patients with cancer are comparable with, or higher than, those of cancer patients themselves (e.g., Hagedoorn, Sanderman, Bolks, Tuinstra, & Coyne, 2008; Sklenarova et al., 2015). A majority of caregivers have unmet needs for support, particularly with regard to addressing fears about the patient's condition, obtaining adequate disease- and treatment-related information, and receiving emotional support for themselves (Sklenarova et al., 2015; see *Caregivers* entry).

Although many cancer patients adjust well and do not demonstrate significant deterioration in mental health, wide individual variability exists. Longitudinal studies have identified four distinct trajectories of psychological adjustment to breast cancer diagnosis (e.g., Henselmans et al., 2010). In these studies, about one-third of individuals experienced no distress throughout diagnosis and treatment. Another third showed a pattern of recovery, wherein patients experienced distress during diagnosis and active treatment, followed by a significant decrease in distress after completing treatment. A smaller group (15%) evidenced an increase in distress shortly after treatment completion (during the reentry phase). Finally, a small but notable group of individuals experienced unremittingly high distress throughout diagnosis, treatment, and beyond.

Predictors of Psychosocial Adjustment to Cancer

Specifying risk and protective factors for psychosocial adjustment to cancer builds on our understanding of trajectories of adjustment and can help identify individuals who could benefit from intervention. To this end, longitudinal research has identified demographic, cancer-related, psychosocial, and interpersonal factors associated with adjustment to cancer.

Demographic risk factors for poor psychosocial adjustment to cancer include younger age, Latino ethnicity, and low socioeconomic status (Stanton & Yanez, 2013). Chemotherapy receipt is associated with less favorable physical adjustment (Ganz, Kwan, Stanton, Bower, & Belin, 2011). Research examining the association between advanced cancer stage and psychological adjustment has yielded mixed results; research is needed to characterize the relationship between cancer stage and emotional functioning and identify potential moderators of the relationship. Personality attributes such as optimism and mastery (i.e., perceived control) are associated with more favorable adjustment, whereas neuroticism is related to poor adjustment (Henselmans et al., 2010). Negative expectancies about cancer-related outcomes contribute to increased distress.

Cancer-specific coping processes are important predictors of psychological adjustment to cancer (Stanton & Bower, 2015). Coping through approach-oriented strategies, which include emotional expression, problem solving, seeking social support, positive reappraisal, and active acceptance, predicts better psychological adjustment. Conversely, coping through avoidance of thoughts and feelings regarding cancer predicts increases in distress. Social factors also influence psychological adjustment to cancer (Michael, Berkman, Colditz, Holmes, & Kawachi, 2002). Social isolation, perceived loneliness, and lack of interpersonal support are associated with compromised adjustment.

These risk and protective factors may also influence physical health and functioning (e.g., fatigue, pain, sleep disturbance) during and after cancer. Continued research is needed to examine factors that predict both psychological and physical functioning. Research in this area will inform targeted interventions to prevent depression, anxiety, and other negative sequelae by addressing individual risk factors.

Directions for Future Research

Much of the research in psycho-oncology is composed of studies of well-educated, non-Hispanic White women with early-stage breast cancer. Research is needed using diverse samples with various cancer diagnoses and underserved groups including those with advanced cancer, socioeconomically disadvantaged individuals, and racial/ethnic minorities. Furthermore, although the majority of people with cancer are older adults, research in psycho-oncology largely focuses on middle-aged and younger adults. Research findings may not apply to older individuals with unique needs and concerns including multiple comorbid conditions, loss of independence, and pronounced side effects of treatment.

Despite the documented psychosocial needs and concerns of cancer patients, psychosocial support is not routinely provided to patients as a component of their oncology care (IOM, 2013). Recognizing the importance of psychosocial care for individuals with cancer and implemented in 2015, the American College of Surgeons Commission on Cancer (CoC) mandated universal psychosocial distress screening for cancer patients as a new patient care standard for CoC-accredited cancer programs (American College of Surgeons Commission on Cancer, 2012). Psychologists are well positioned to conduct research and make recommendations regarding appropriate screening measures, as well as to develop and disseminate evidence-based interventions for patients who screen positive for distress (see *Assessment in Health Care Settings* entry). It will also be critical for psychological researchers to investigate the potential benefits of integrated oncologic care such that patients' psychosocial needs are addressed within their existing network of cancer healthcare professionals.

Researchers in psychological science have increasing opportunities to form multidisciplinary collaborations with investigators in medicine, public health, and public policy to inform

cutting-edge research and advance knowledge in the field of psycho-oncology. For instance, psychologists could assess patient-reported outcomes, including psychosocial and physical functioning, in clinical trials of new medical treatments for cancer. Psycho-oncology will benefit from adopting a biopsychosocial approach to investigate and address proactively the complex interplay of biological, intrapersonal, and interpersonal factors that influence cancer-related decision making and adjustment. A recent review regarding fatigue in cancer survivors, for example, exemplifies a biopsychosocial approach as it examines the demographic, medical, psychosocial, behavioral, and biological factors that influence cancer-related fatigue (Bower, 2014).

A recent Institute of Medicine report concluded that the cancer care system is unprepared to care for the growing—and aging—population of cancer survivors in the United States (IOM, 2013). Psychologists will have an important role—as both researchers and clinicians—in reducing the burden of cancer through prevention, early detection, and effective management of the challenges of cancer diagnosis, treatment, and posttreatment survivorship.

Author Biographies

Lauren N. Harris received her PhD in clinical psychology from the University of California, Los Angeles. Her research focuses on identifying risk and protective factors in adjustment to cancer and developing targeted interventions to improve psychosocial adjustment for cancer survivors. She is currently a postdoctoral fellow specializing in clinical health psychology at the West Los Angeles VA Medical Center.

Annette L. Stanton, PhD, is professor of psychology and psychiatry/biobehavioral sciences at the University of California, Los Angeles, and a member of the Jonsson Comprehensive Cancer Center. Her research centers on identifying contributors to biopsychosocial health in adults undergoing chronic stress, with a focus on the experience of cancer, and to translate findings into evidence-based interventions.

References

- American Association for Cancer Research. (2017). *AACR cancer progress report 2017*. Philadelphia, PA: Author.
- American Cancer Society. (2017). *Cancer facts & figures, 2017*. Atlanta, GA: Author.
- American College of Surgeons Commission on Cancer. (2012). *Cancer program standards 2012: Ensuring patient-centered care*. Chicago, IL: American College of Surgeons.
- Bower, J. E. (2014). Cancer-related fatigue—Mechanisms, risk factors, and treatments. *Nature Reviews. Clinical Oncology*, 11(10), 597–609.
- Bower, J. E., Meyerowitz, B. E., Bernaards, C. A., Rowland, J. H., Ganz, P. A., & Desmond, K. A. (2005). Perceptions of positive meaning and vulnerability following breast cancer: Predictors and outcomes among long-term breast cancer survivors. *Annals of Behavioral Medicine*, 29(3), 236–245.
- Brandon, T. H., Unrod, M., & Simmons, V. N. (2015). Tobacco use and cessation. In J. C. Holland, W. S. Breitbart, P. N. Butow, P. B. Jacobsen, M. J. Loscalzo, & R. McCorkle (Eds.), *Psycho-oncology* (3rd ed., pp. 3–7). New York, NY: Oxford University Press.
- Faller, H., Schuler, M., Richard, M., Heckl, U., Weis, J., & Küffner, R. (2013). Effects of psycho-oncologic interventions on emotional distress and quality of life in adult patients with cancer: Systematic review and meta-analysis. *Journal of Clinical Oncology*, 31, 782–793.
- Ganz, P. A., Kwan, L., Stanton, A. L., Bower, J. E., & Belin, T. R. (2011). Physical and psychosocial recovery in the year after primary treatment of breast cancer. *Journal of Clinical Oncology*, 29(9), 1101–1109.

- Gillison, M. L., Zhang, Q., Jordan, R., Xiao, W., Westra, W. H., Trotti, A., ... Ang, K. K. (2012). Tobacco smoking and increased risk of death and progression for patients with p16-positive and p16-negative oropharyngeal cancer. *Journal of Clinical Oncology*, 30, 1–12.
- Hagedoorn, M., Sanderman, R., Bolks, H. N., Tuinstra, J., & Coyne, J. C. (2008). Distress in couples coping with cancer: A meta-analysis and critical review of role and gender effects. *Psychological Bulletin*, 134(1), 1–30.
- Hay, J. L., Buckley, T. R., & Ostroff, J. S. (2005). The role of cancer worry in cancer screening: A theoretical and empirical review of the literature. *Psycho-Oncology*, 14(7), 517–534.
- Health and Social Care Information Centre. (2013a). *Breast Screening Programme, England 2011–12*.
- Health and Social Care Information Centre. (2013b). *Cervical Screening Programme, England 2012–2013*.
- Henselmans, I., Helgeson, V. S., Seltman, H., de Vries, J., Sanderman, R., & Ranchor, A. V. (2010). Identification and prediction of distress trajectories in the first year after a breast cancer diagnosis. *Health Psychology*, 29(2), 160–168.
- Hershman, D. L., Shao, T., Kushi, L. H., Buono, D., Tsai, W. Y., Fehrenbacher, L., ... Neugut, A. I. (2011). Early discontinuation and non-adherence to adjuvant hormonal therapy are associated with increased mortality in women with breast cancer. *Breast Cancer Research and Treatment*, 126(2), 529–537.
- Holland, J. C., Breitbart, W. S., Butow, P. N., Jacobsen, P. B., Loscalzo, M. J., & McCorkle, R. (2015). *Psycho-oncology* (3rd ed.). New York, NY: Oxford University Press.
- IOM (Institute of Medicine). (2005). *From cancer patient to cancer survivor: Lost in transition*. Washington, DC: The National Academies Press.
- IOM (Institute of Medicine). (2013). *Delivering high-quality cancer care: Charting a new course for a system in crisis*. Washington, DC: The National Academies Press.
- Jacobsen, P. B., & Andrykowski, M. A. (2015). Tertiary prevention in cancer care: Understanding and addressing the psychological dimensions of cancer during the active treatment period. *American Psychologist*, 70(2), 134–145.
- Lutgendorf, S. K., & Andersen, B. L. (2015). Biobehavioral approaches to cancer progression and survival: Mechanisms and interventions. *American Psychologist*, 70(2), 186–197.
- Michael, Y. L., Berkman, L. F., Colditz, G. A., Holmes, M. D., & Kawachi, I. (2002). Social networks and health-related quality of life in breast cancer survivors: A prospective study. *Journal of Psychosomatic Research*, 52(5), 285–293.
- Mitchell, A. J., Chan, M., Bhatti, H., Halton, M., Grassi, L., Johansen, C., & Meader, N. (2011). Prevalence of depression, anxiety, and adjustment disorder in oncological, haematological, and palliative-care settings: A meta-analysis of 94 interview-based studies. *The Lancet Oncology*, 12(2), 160–174.
- Rainbird, K., Perkins, J., Sanson-Fisher, R., Rolfe, I., & Anseline, P. (2009). The needs of patients with advanced, incurable cancer. *British Journal of Cancer*, 101(5), 759–764.
- Rock, C. L., Doyle, C., Demark-Wahnefried, W., Meyerhardt, J., Courneya, K. S., Schwartz, A. L., ... Byers, T. (2012). Nutrition and physical activity guidelines for cancer survivors. *CA: A Cancer Journal for Clinicians*, 62(4), 242–274.
- Sabatino, S. A., White, M. C., Thompson, T. D., & Klabunde, C. N. (2015). Cancer screening test use—United States, 2013. *Morbidity and Mortality Weekly Report*, 64(17), 464–468.
- Sklenarova, H., Krümpelmann, A., Haun, M. W., Friederich, H. C., Huber, J., Thomas, M., ... Hartmann, M. (2015). When do we need to care about the caregiver? Supportive care needs, anxiety, and depression among informal caregivers of patients with cancer and cancer survivors. *Cancer*, 121(9), 1513–1519.
- Spring, B., King, A. C., Pagoto, S. L., Van Horn, L., & Fisher, J. D. (2015). Fostering multiple healthy lifestyle behaviors for primary prevention of cancer. *American Psychologist*, 70, 75–90.
- Stanton, A. L., & Bower, J. E. (2015). Psychological adjustment in breast cancer survivors. In *Improving outcomes for breast cancer survivors* (pp. 231–242). NY, USA: Springer International Publishing.

- Stanton, A. L., Rowland, J. H., & Ganz, P. A. (2015). Life after diagnosis and treatment of cancer in adulthood: Contributions from psychosocial oncology research. *American Psychologist*, 70(2), 159–174.
- Stanton, A. L., & Yanez, B. (2013). The experience of cancer in women. In M. V. Spiers, P. A. Geller, & J. D. Kloss (Eds.), *Women's health psychology* (pp. 491–513). New York, NY: Wiley.
- Wardle, J., Robb, K., Vernon, S., & Waller, J. (2015). Screening for prevention and early diagnosis of cancer. *American Psychologist*, 70(2), 119–133.

Suggested Reading

- Green McDonald, P., Suls, J., & Glasgow, R. (Eds.) (2015). Special issue on cancer and psychology. *American Psychologist*, 70(2), 61–224.
- Holland, J. C., Breitbart, W. S., Butow, P. N., Jacobsen, P. B., Loscalzo, M. J., & McCorkle, R. (2015). *Psycho-Oncology* (3rd ed.). New York, NY: Oxford University Press.
- IOM (Institute of Medicine). (2013). *Delivering high-quality cancer care: Charting a new course for a system in crisis*. Washington, DC: The National Academies Press.

Ischemic Heart Disease, Depression, and Tobacco Smoking: Implications for Health Psychology

Allison J. Carroll

*Department of Preventive Medicine, Northwestern University Feinberg School of Medicine,
Chicago, IL, USA*

Between the years 2009 and 2012, the prevalence of ischemic heart disease among US adults was estimated to be roughly 6% (Benjamin et al., 2017). High-quality evidence supports an association between ischemic heart disease and a number of behavioral risk factors, including tobacco smoking, physical inactivity, sedentary behavior, and poor diet. More recently, there has been a focus on how the development and progression of ischemic heart disease may be affected by psychological factors, including depression, anxiety, and hostility (Davidson, 2012; Kubzansky, Davidson, & Rozanski, 2005). Significant debate remains, however, regarding the nature of the association between these psychological factors and primary onset of ischemic heart disease, and many argue that the observed associations between psychological factors or personality traits and ischemic heart disease are purely moderated by one or more of the established risk behaviors for ischemic heart disease. This line of discovery provides an opportunity for health psychologists to assist in advancing the understanding and treatment of comorbid risk factors for both primary and secondary prevention of ischemic heart disease.

To illustrate the complex relationships between psychological conditions, behavioral risk factors, and ischemic heart disease, this chapter will provide an overview of the literature examining the interplay between depression, tobacco smoking, and ischemic heart disease. First, a brief discussion is provided regarding the measurement of ischemic heart disease. Second, the evidence supporting the association between depression and ischemic heart disease is outlined. Third, a description of the basis for evaluating the role of smoking in the association between depression and ischemic heart disease, and why depression and smoking are hypothesized to be synergistically associated with ischemic heart disease, is provided. The chapter concludes with an overview of the societal impact of this research and recommendations for health psychologists.

Measuring Ischemic Heart Disease

There are many definitions of heart disease, but the focus of this chapter will be on ischemic heart disease (i.e., coronary artery disease or coronary heart disease). Ischemic heart disease most typically develops as a result of atherosclerosis, a buildup or blockage, in the coronary arteries that reduces blood flow to the heart. The reduction in blood flow to the heart may have consequences that range from minor physical discomfort, such as angina (chest pain), to death from myocardial infarction (heart attack). The primary reason for focusing specifically on ischemic heart disease (as opposed to cardiovascular diseases more broadly) is because coronary artery calcification (CAC), a subclinical measure that is largely specific to ischemic heart disease, has been the focus of available research studies assessing risk for primary ischemic heart disease.

CAC is the best available predictor of future cardiac events (Greenland et al., 2007). CAC is a measure of atherosclerosis, assessed by computed tomography to assess areas of calcium deposits in the coronary arteries. It is most often reported using Agatston scores, and many research studies simply report the presence of CAC (i.e., “detectable” CAC; Agatston score >0). In a 2007 report from the American College of Cardiology Foundation and the American Heart Association Task Force on Practice Guidelines, data compiled from 6 published studies with a total of 27,622 patients demonstrated that detectable CAC was associated with a 4.3-fold (95% confidence interval [CI]: 3.5–5.2) increase in the relative risk of a cardiac event over 3–5 years (Greenland et al., 2007). CAC scores are highly correlated with age, sex, and race/ethnicity, where older age, male sex, and White race are at highest risk for detectable CAC. For further information about the probability of detectable CAC by age, sex, and race/ethnicity, readers are referred to the Multi-Ethnic Study of Atherosclerosis (MESA) calcium calculator (<http://www.mesa-nhlbi.org/calcium/input.aspx>).

The Negative Effects of Depression on Ischemic Heart Disease

Depression Predicts Ischemic Heart Disease Incidence and Mortality

Though significant available evidence supports depression as a risk factor for poor prognosis among patients with ischemic heart disease (Lichtman et al., 2014), research supporting depression as a risk factor for incident ischemic heart disease is still developing. Emerging evidence indicates that the association between depression and the heart is quite broad. In a study of 1.9 million adults in the UK health system, a documented diagnosis of depression or a prescription for antidepressant medication was predictive of 12 different cardiovascular disease diagnoses, with hazard ratios (HRs) ranging from 1.12 (95% CI: 1.01–1.24) for abdominal aortic aneurysm to 1.70 (95% CI: 1.60–1.82) for unstable angina, even after adjusting for traditional risk factors for cardiovascular diseases (Daskalopoulou et al., 2016). In 2006, Nicholson, Kuper, and Hemingway (2006) conducted a systematic review and meta-analysis of 11 etiological cohort studies published between 1966 and 2003 evaluating the association between depression and ischemic heart disease. Nicholson et al. observed that even after studies adjusted for other risk factors, the risk of developing ischemic heart disease among individuals with depression is nearly double (95% CI: 1.5–2.4) the risk compared with those without depression (Nicholson et al., 2006).

Prospective cohort studies have provided much of the evidence for an association between a clinical diagnosis of depression and ischemic heart disease. Brown, Stewart, Stump, and

Callahan (2011) analyzed data from a prospective cohort study of older adults (≥ 60 years of age) followed for 13–16 years in primary care. The authors observed that compared with older adults without depression, those individuals with a history of depression were 46% more likely to experience either an acute myocardial infarction or death due to ischemic heart disease (31.9 vs. 25.7%); importantly, the two groups did not differ in rates of all-cause mortality, demonstrating specificity of the association between depression and ischemic heart disease. Similarly, in an 18-year longitudinal study of randomly selected women in Australia, O’Neil et al. (2016b) observed that a baseline diagnosis of clinical depression was associated with three times greater odds of a coronary event (cardiac death, myocardial infarction or intervention) over the next 18 years. Furthermore, adding the baseline clinical depression diagnosis to the Framingham Risk Equation, a “risk calculator” for cardiac disease and events commonly used in medical practice (<https://www.framinghamheartstudy.org/risk-functions/index.php>), modestly but significantly improved risk prediction of 10-year coronary events (O’Neil et al., 2016a).

Unfortunately, not everyone who suffers from depression will receive a formal diagnosis, and others may have persistent but subthreshold symptomatology. In the Whitehall II study, rather than evaluating a clinical diagnosis, Brunner et al. (2014) evaluated whether depressive symptomatology at up to six exams would predict ischemic heart disease outcomes over 24 years of follow-up. They found a dose–response relationship between number of exams with elevated depressive symptoms and ischemic heart disease, where participants with elevated depressive symptoms at two or more exams had nearly 50% increased risk of developing ischemic heart disease compared with participants without elevated depressive symptoms at any exams (HR: 1.47, 95% CI: 1.13–1.91) (Brunner et al., 2014). Taken together, the available research suggests that researchers and clinicians should consider “nontraditional” factors, particularly affective symptomatology, when evaluating risk for ischemic heart disease.

Mechanisms by Which Depression Is Hypothesized to Increase Risk for Ischemic Heart Disease

The pathophysiological mechanisms underlying the association between depression and ischemic heart disease are poorly understood at this time, though several physiological and behavioral mechanisms have been theorized. Physiologically based processes by which depression is hypothesized to promote ischemic heart disease include platelet reactivity, blood coagulation, inflammation, endothelial dysfunction, neuroendocrine dysregulation (i.e., increased sympathetic nervous system activity and decreased parasympathetic nervous system activity), cardiac rhythm disturbances (e.g., heart rate variability), and metabolic disorders (e.g., diabetes) (Davidson, 2012). Data from twin studies support this hypothesis, suggesting that certain genetic pathways that underlie both depression and ischemic heart disease may simultaneously increase risk for both conditions (Mulle & Vaccarino, 2013).

Others hypothesize that certain behaviors that are highly comorbid with depression, such as smoking and alcohol use, and those that are symptoms of depression, such as fatigue/lethargy and sleep disturbance, may account for the observed relationships between depression and ischemic heart disease. For example, Appleton et al. (2016) found that ischemic heart disease mortality associated with depression (unadjusted HR: 1.28, 95% CI: 1.02–1.62) was no longer significant after adjusting for behavioral factors (adjusted HR: 1.20, 95% CI: 0.94–1.52), including fruit and vegetable intake, physical activity, current smoking, and current alcohol consumption.

Depression and Coronary Artery Calcification

Due to the amount of time required for adults to develop ischemic heart disease or experience a cardiac event, until these mechanisms are better delineated, research progress will benefit from focusing on subclinical measures of ischemic heart disease for the purposes of evaluating the role of depression and depression treatment in the primary prevention of ischemic heart disease. The relationship between depression and CAC has been evaluated in several forms.

Persistent or recurrent depression (e.g., diagnosis of major depressive disorder or multiple episodes of significantly elevated depressive symptoms) has consistently been shown to be predictive of CAC. Women with a history of recurrent major depression, compared with those who reported only a single episode or no depression, are twice as likely to have CAC (Agatista et al., 2005; Jones, Bromberger, Sutton-Tyrrell, & Matthews, 2003). Furthermore, the progression of CAC (i.e., the magnitude of change in Agatston scores) is directly related to level of depressive symptoms: Janssen et al. (2011) observed that the risk of CAC progression in women increases by approximately 25% for each incremental increase in depressive symptoms.

In two different studies, researchers attempted to model the longitudinal effect of the chronicity of depression on the development of subclinical ischemic heart disease by categorizing participants by the number of depressive episodes according to self-reported current depressive symptomatology assessed at multiple exams. In the Whitehall II study, Hamer, Kivimaki, Lahiri, Marmot, and Steptoe (2010) observed that individuals with two or three depressive episodes (focused only on the cognitive symptoms of depression) had approximately 2.5 times the odds of detectable CAC compared with individuals without any depressive episodes (OR: 2.56, 95% CI: 1.14–5.78). Janssen et al. (2016) similarly observed that women in the Study of Women's Health Across the Nation Heart Study with three or more depressive episodes (using a global depression score) had over two times greater odds of CAC compared with women without any depressive episodes (OR: 2.20, 95% CI: 1.13–4.28). Interestingly, in both studies, participants with only one depressive episode did not have significantly higher odds of CAC compared with individuals without any depressive episodes (Hamer et al., 2010; Janssen et al., 2016).

On the other hand, the majority of available evidence indicates that current depressive symptomatology alone does not predict CAC. Cross-sectional studies evaluating associations between depression and depressive scores and CAC have not found significant associations (e.g., Devantier et al., 2013). Likewise, when participants were only asked about past-week depressive symptoms, neither continuous depression scores in an older, multiethnic population (Diez Roux et al., 2006) nor a categorical diagnosis of depression among women (Matthews, Owens, Edmundowicz, Lee, & Kuller, 2006) was associated with greater likelihood of CAC.

Overall, it appears as though chronic exposure to and accumulation of depressive symptoms is required to increase risk for ischemic heart disease, similar to other traditional risk factors, bearing in mind that the majority of studies to date have been conducted in women. While these studies provide evidence for an association between depression and subclinical ischemic heart disease, researchers argue that the presence of depression alone does not fully explain the negative association we see with ischemic heart disease. Therefore, it becomes important to evaluate the potential role of comorbid risk factors in the relationship between depression and ischemic heart disease. To illustrate this concept, we chose to focus on the role of tobacco smoking, because it is highly comorbid with depression and because it is a strong and modifiable risk factor for ischemic heart disease, which has implications for prevention and treatment of ischemic heart disease.

The Role of Smoking in the Association Between Depression and Ischemic Heart Disease

A Brief Overview of the Comorbidity Between Depression and Smoking

A strong, bidirectional, and causal association exists between depression and smoking, where these two factors are both predisposing to and maintaining of each other. In the United States, adults with elevated depressive symptoms smoke at over twice the rate of the general population (43 vs. 22% in 2005–2008) (Pratt & Brody, 2010). Chaiton, Cohen, O’Loughlin, and Rehm (2009) conducted a meta-analysis of prospective studies in adolescents, where they found that smoking increased the odds of depression by 73% (95% CI: 1.3–2.4) and also that depression increased the odds of smoking by 41% (95% CI: 1.2–1.6). Genetic studies in identical twins also lend strong support for a biological basis of this comorbidity (Lyons et al., 2008).

Novel theories have been invoked to explain this association. One example is a transdiagnostic vulnerability framework, whereby anhedonia, anxiety sensitivity, and distress tolerance are key traits underlying the association between smoking and emotion (Leventhal & Zvolensky, 2015). A second example is an incentive learning theory, which posits that low mood (e.g., depression) increases the expected value of and therefore desire for a cigarette, which ultimately serves to maintain smoking behavior (Hogarth et al., 2015). Taken together, these theories and studies present not only an underlying genetic basis but also a behavioral or trait vulnerability to and maintenance of comorbid depression and smoking.

Smoking Predicts Ischemic Heart Disease

Research unequivocally indicates that cigarette smoking increases risk for premature ischemic heart disease morbidity and mortality in a dose-dependent manner (USDHHS, 2014). With regard to subclinical ischemic heart disease, smoking is a consistent and dose-dependent predictor of prevalence and progression of CAC (McEvoy et al., 2012). Studies have demonstrated that, among individuals with CAC, those who smoke are between four and nine times more likely to suffer cardiac death compared with their nonsmoking counterparts, which equates to an average loss of nearly 5 life years (Shaw, Raggi, Callister, & Berman, 2006).

The large list of ingredients and chemicals that compose cigarette smoke complicates the determination of any one specific mechanism by which cigarette smoking causes heart disease; discussion of all of the possibilities is beyond the scope of this chapter. Briefly, cigarette smoking stimulates the sympathetic nervous system and the heart. This stimulus induces several downstream physiological effects, including increased demand for oxygen, faster heart rate, elevated blood pressure, and greater myocardial contractility. Other suggested mechanisms include inflammation, endothelial injury, insulin sensitivity, and lipid abnormalities. Several of these mechanisms overlap with those by which depression is hypothesized to affect ischemic heart disease, providing further basis for assessing the potential synergistic association between these two risk conditions in relation to ischemic heart disease.

How Comorbid Depression and Smoking Have Been Observed to Affect the Heart

A representation of the hypothesized association between depression and ischemic heart disease is presented in Figure 1. Research evaluating the potential interactive effect of depression and

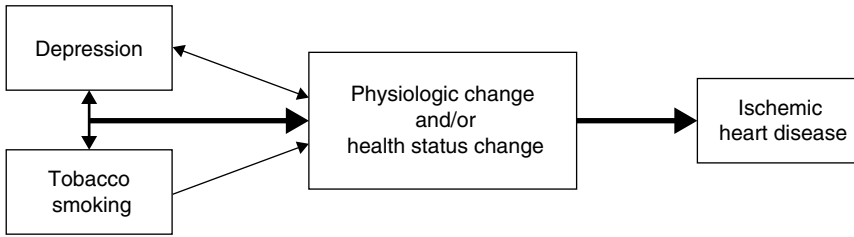


Figure 1 Hypothesized association between depression, tobacco smoking, and ischemic heart disease based on available research, where the thickness and direction of the arrows are indicative of the proposed strength and directionality of the associations. *Source:* This diagram was adapted for this chapter from Joynt, Whellan, and O'Connor (2003), figure 1 p. 249.

smoking on incident ischemic heart disease or cardiac events is limited. However, a synergistic association between depression and smoking has been observed with physical cardiac symptoms that may be indicators of future or subthreshold ischemic heart disease. For example, high heart rate variability (i.e., variability in the time interval between heart beats) is indicative of healthy cardiac function and may serve as a buffer for cardiac stress. Harte et al. (2013) observed that among adults with depression, those who smoked had lower heart rate variability than individuals who did not smoke. This finding suggests that cardiac function may be worst among adults who are both depressed and smoking compared with adults with only one or neither risk factor.

As noted above, several of the proposed mechanisms for the association between smoking and ischemic heart disease overlap with those relating depression and ischemic heart disease. Some researchers found evidence for a synergistic association between smoking, depression, and measures of these hypothesized mechanisms. Using inflammation, or the inflammatory response, as an example, Vulser et al. (2015) showed that the observed association between depressive symptoms with absolute neutrophil count was fully mediated by smoking. In another study, Stewart, Rand, Muldoon, and Kamarck (2009) argued that depressed individuals experience a dysfunction in the bodily systems that typically facilitate termination of an acute inflammatory response. They suggest that when a proinflammatory stimulus, such as smoking, is introduced to a person suffering from depression, he or she may exhibit a larger and/or more prolonged response to each exposure (e.g., each time that person smokes). The result is that, over time, these exaggerated inflammatory responses may accelerate the progression of atherosclerosis. Though preliminary, this evidence provides a basis and theory for a synergistic association between depression and smoking in relation to ischemic heart disease.

Depression, Smoking, and Coronary Artery Calcification

Only one study to date has looked for a potential interactive association between depression and smoking in relation to ischemic heart disease. Using data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, Carroll et al. (2017) evaluated the association between cumulative, 25-year smoking exposure and depressive symptoms with CAC at year 25 among the 3,188 US adults. After adjusting for relevant sociodemographic, clinical, and behavioral covariates, participants with both high smoking exposure (30 pack-years) and clinically elevated depressive symptoms (a score of 16 on the Centers for Epidemiologic Studies—Depression [CES-D] scale) had nearly four times greater odds of moderate-risk CAC (scores 1–99) and over six times greater odds of higher-risk CAC (scores ≥ 100) compared

with individuals without any depressive symptoms or smoking history (Figure 2) (Carroll et al., 2017). These findings provide preliminary evidence for a synergistic effect of depression and smoking with the onset of ischemic heart disease.

Other factors must be considered when evaluating the association between depression, smoking, and subclinical ischemic heart disease. First, subtypes of depression and clusters of depressive symptomatology (e.g., cognitive disturbance, somatic symptoms, anhedonia) may have differential associations in the relationship between depression, smoking, and ischemic

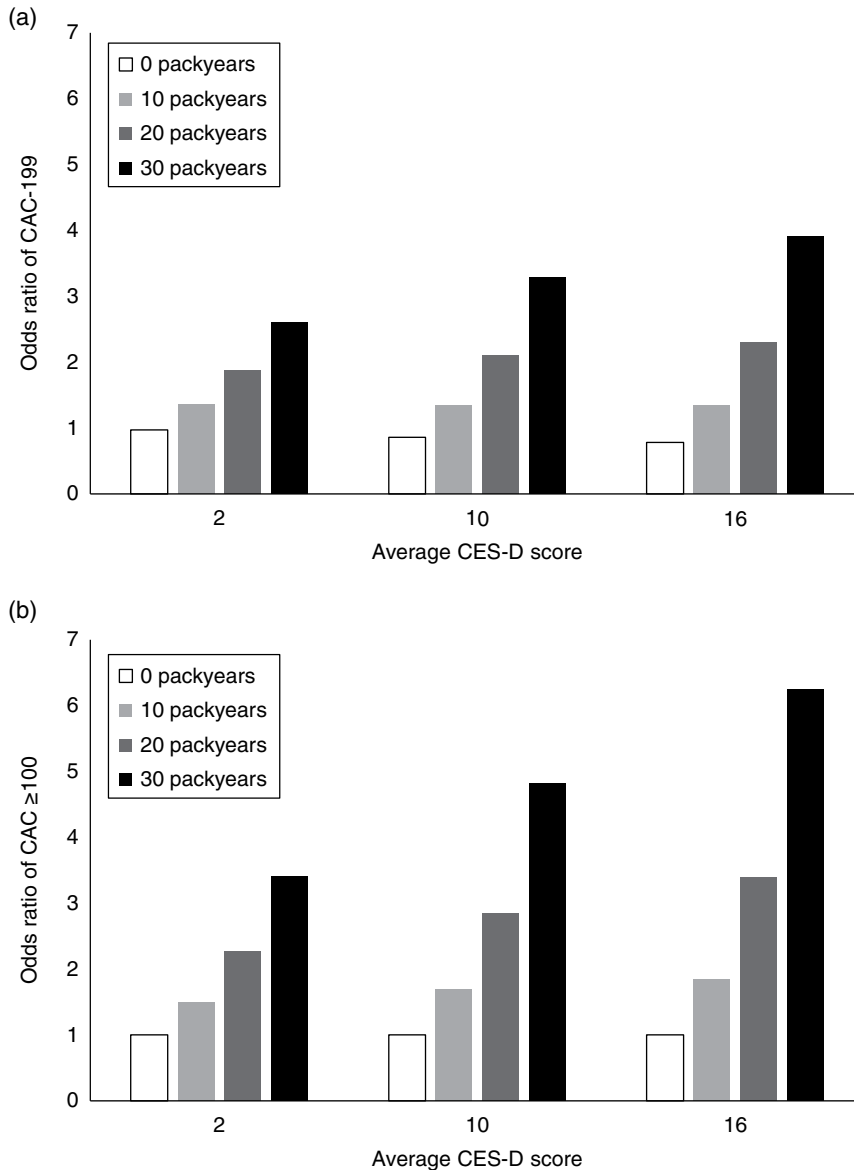


Figure 2 Odds ratios of lower-risk (Agatston scores 1–99; Panel A) and higher-risk (Agatston scores ≥ 100 ; Panel B) coronary artery calcification by depressive symptoms (CES-D score) $\times$ smoking (pack-years) relative to CES-D score = 0 and pack-years = 0, $N = 3,188$. *Source:* Adapted from Carroll et al. (2017).

heart disease. Second, the influence of age on ischemic heart disease cannot be ignored, given that the association between measures of subclinical ischemic heart disease and cardiac events is not strictly linear. In other words, younger individuals with detectable subclinical ischemic heart disease have lower relative risk, but higher absolute risk, of suffering a cardiac event. Finally, further study of the complex interplays between additional behavioral (e.g., alcohol use, physical activity) and/or psychological (e.g., anxiety, anger) conditions that contribute to the “Perfect Storm”—that is, the “joint presence of many co-occurring pathophysiologic processes that lead to a clinical event” (Burg et al., 2013)—remains crucial.

Societal Implications and the Role of the Health Psychologist

Ischemic heart disease accounts for approximately one in seven deaths among US adults, and the estimated direct and indirect costs of ischemic heart disease exceed \$200 billion per year (Benjamin et al., 2017). Understanding the influence of psychological and behavioral factors on CAC is essential because, as of yet, there is no evidence to indicate that development of subclinical ischemic heart disease can be reversed. Therefore, preventing the development or limiting the progression of CAC is of the utmost importance. Much of the preservation of cardiovascular health could be achieved through early intervention on modifiable risk factors.

Smoking cessation swiftly and effectively reduces risk for ischemic heart disease and overall mortality (USDHHS, 2014); little is known about treating depression for the prevention of ischemic heart disease. However, one intervention study among older adults (>60 years of age) in primary care evaluated the effect of a collaborative care treatment for depression using antidepressants and pharmacotherapy on ischemic heart disease. Stewart, Perkins, and Callahan (2014) found that participants without an ischemic heart disease diagnosis who underwent treatment for their depression ($n = 85$), compared with those in usual care (i.e., encouragement to follow up with provider; $n = 83$), experienced fewer cardiac events (28 vs. 47%) over 8 years (HR: 0.52, 95% CI: 0.31–0.86). Consistent with previous research (Baumeister, Hutter, & Bengel, 2011), the authors did not observe improved mortality outcomes associated with depression treatment among individuals with preexisting ischemic heart disease. As such, an emphasis on ischemic heart disease prevention in those suffering from depression should be included in future clinical and public health efforts.

Recognizing and treating the depression–smoking comorbidity has the potential to lead to even greater advances in ischemic heart disease prevention. In an updated systematic review and meta-analysis comprising 42 smoking cessation studies, Hitsman et al. (2013) found that smokers with a history of depression had 17% (95% CI: 0.72–0.95) and 19% (95% CI: 0.67–0.97) lower odds of short-term (≤ 3 months) and long-term (≥ 6 months) abstinence, respectively. Furthermore, smokers who quit smoking experience significant improvements in negative affect (Taylor et al., 2014), contrary to popularly held beliefs. Given the recurrent, reciprocal, and relapsing nature of depression and smoking, leading experts in the field recommend a long-term and/or continuous care model using integrated evidence-based treatments for depressed smokers in order to address the high rates of relapse for both depression and smoking (Richards, Cohen, Morrell, Watson, & Low, 2013).

A number of national and global initiatives have been set forth to reduce the number of deaths due to noncommunicable diseases, including ischemic heart disease. These initiatives include the American Heart Association’s Strategic Impact Goal of reducing cardiovascular disease mortality by 20% and improving cardiovascular health by 20% by the year 2020 (Lloyd-Jones et al., 2010), the United Nations Sustainable Development Goals to reduce mortality

due to noncommunicable diseases by one-third by 2030 (<http://www.un.org/sustainabledevelopment/health/>), and the World Health Organization target to reduce mortality due to noncommunicable diseases by 25% by the year 2025 (World Health Organization, 2013). These initiatives provide an impetus for health psychologists to get involved in the prevention of ischemic heart disease, given their growing presence in primary care and other medical settings. Health psychologists may be in the best position to identify and treat these at-risk individuals to prevent development and progression of ischemic heart disease. Furthermore, health psychologists, both clinicians and researchers, can contribute to prevention efforts by developing and implementing novel targeted treatments for ischemic heart disease risk factors among at-risk patients.

Conclusion

Recognition of the role of nontraditional risk factors for ischemic heart disease is growing, especially for psychological conditions such as depression. The evidence supporting an association between depression and ischemic heart disease is tantalizing but mixed, likely related to the confounding effects of risk behaviors, and tobacco smoking in particular. As such, further research in this area is needed to fully elucidate the association between depression and risk for ischemic heart disease. Nonetheless, the available evidence indicates that health psychologists can and should play an important role in ischemic heart disease prevention through interventions designed to target and treat comorbid depression and tobacco smoking.

Acknowledgments

I would like to thank Dr. Mark Huffman for his conceptual and editorial suggestions in the development of this chapter.

Author Biography

Allison J. Carroll is a doctoral candidate in clinical psychology (behavioral medicine) at Northwestern University Feinberg School of Medicine. Her research interests include understanding the associations between multiple risk factors with chronic disease risk and evaluating clinical interventions for disease prevention and health promotion.

References

- Agatsuma, P. K., Matthews, K. A., Bromberger, J. T., Edmundowicz, D., Chang, Y. F., & Sutton-Tyrrell, K. (2005). Coronary and aortic calcification in women with a history of major depression. *Archives of Internal Medicine*, 165(11), 1229–1236.
- Appleton, K. M., Woodside, J. V., Arveiler, D., Haas, B., Amouyel, P., Montaye, M., ... The PRIME Study Group. (2016). A role for behavior in the relationships between depression and hostility and cardiovascular disease incidence, mortality, and all-cause mortality: The prime study. *Annals of Behavioral Medicine*, 50(4), 582–591.
- Baumeister, H., Hutter, N., & Bengel, J. (2011). Psychological and pharmacological interventions for depression in patients with coronary artery disease. *Cochrane Database of Systematic Reviews*, 7(9).

- Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., ... American Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2017). Heart disease and stroke statistics-2017 update: A report from the American Heart Association. *Circulation*, 135(10), e146–e603.
- Brown, J. M., Stewart, J. C., Stump, T. E., & Callahan, C. M. (2011). Risk of coronary heart disease events over 15 years among older adults with depressive symptoms. *American Journal of Geriatric Psychiatry*, 19(8), 721–729.
- Brunner, E. J., Shipley, M. J., Britton, A. R., Stansfeld, S. A., Heuschmann, P. U., Rudd, A. G., ... Kivimaki, M. (2014). Depressive disorder, coronary heart disease, and stroke: Dose-response and reverse causation effects in the Whitehall II cohort study. *European Journal of Preventive Cardiology*, 21(3), 340–346.
- Burg, M. M., Edmondson, D., Shimbo, D., Shaffer, J., Kronish, I. M., Whang, W., ... Davidson, K. W. (2013). The ‘perfect storm’ and acute coronary syndrome onset: Do psychosocial factors play a role? *Progress in Cardiovascular Diseases*, 55(6), 601–610.
- Carroll, A. J., Carnethon, M. R., Liu, K., Jacobs, D. R., Colangelo, L. A., Stewart, J. C., ... Hitsman, B. (2017). Interaction between smoking and depressive symptoms with subclinical heart disease in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Health Psychology*, 36(2), 101–111.
- Chaiton, M., Cohen, J., O’Loughlin, J., & Rehm, J. (2009). A systematic review of longitudinal studies on the association between depression and smoking in adolescents. *BMC Public Health*, 9(1), 356.
- Daskalopoulou, M., George, J., Walters, K., Osborn, D. P., Batty, G. D., Stogiannis, D., ... Hemingway, H. (2016). Depression as a risk factor for the initial presentation of twelve cardiac, cerebrovascular, and peripheral arterial diseases: Data linkage study of 1.9 million women and men. *PLoS One*, 11(4), e0153838.
- Davidson, K. W. (2012). Depression and coronary heart disease. *ISRN Cardiology*, 743813(10), 22.
- Devantier, T. A., Norgaard, B. L., Sand, N. P., Mols, R. E., Foldager, L., Diederichsen, A. C., ... Videbech, P. (2013). Lack of correlation between depression and coronary artery calcification in a non-selected Danish population. *Psychosomatics*, 54(5), 458–465.
- Diez Roux, A. V., Ranjit, N., Powell, L., Jackson, S., Lewis, T. T., Shea, S., & Wu, C. (2006). Psychosocial factors and coronary calcium in adults without clinical cardiovascular disease. *Annals of Internal Medicine*, 144(11), 822–831.
- Greenland, P., Bonow, R. O., Brundage, B. H., Budoff, M. J., Eisenberg, M. J., Grundy, S. M., ... Society of Cardiovascular Computed Tomography. (2007). ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: A report of the American College of Cardiology Foundation Clinical Expert Consensus Task Force (ACCF/AHA Writing Committee to update the 2000 expert consensus document on electron beam computed tomography). *Circulation*, 115(3), 402–426.
- Hamer, M., Kivimaki, M., Lahiri, A., Marmot, M. G., & Steptoe, A. (2010). Persistent cognitive depressive symptoms are associated with coronary artery calcification. *Atherosclerosis*, 210(1), 209–213.
- Harte, C. B., Liverant, G. I., Sloan, D. M., Kamholz, B. W., Rosebrock, L. E., Fava, M., & Kaplan, G. B. (2013). Association between smoking and heart rate variability among individuals with depression. *Annals of Behavioral Medicine*, 46(1), 73–80.
- Hitsman, B., Papandonatos, G. D., McChargue, D. E., DeMott, A., Herrera, M. J., Spring, B., ... Niaura, R. (2013). Past major depression and smoking cessation outcome: A systematic review and meta-analysis update. *Addiction*, 108(2), 294–306.
- Hogarth, L., He, Z., Chase, H. W., Wills, A. J., Troisi, J., 2nd, Leventhal, A. M., ... Hitsman, B. (2015). Negative mood reverses devaluation of goal-directed drug-seeking favouring an incentive learning account of drug dependence. *Psychopharmacology*, 232(17), 3235–3247.
- Janssen, I., Powell, L. H., Matthews, K. A., Cursio, J. F., Hollenberg, S. M., Sutton-Tyrrell, K., ... SWAN Study. (2011). Depressive symptoms are related to progression of coronary calcium in

- midlife women: The Study of Women's Health Across the Nation (SWAN) Heart Study. *American Heart Journal*, 161(6), 1186–1191.
- Janssen, I., Powell, L. H., Matthews, K. A., Jasielec, M. S., Hollenberg, S. M., Bromberger, J. T., ... Everson-Rose, S. A. (2016). Relation of persistent depressive symptoms to coronary artery calcification in women aged 46 to 59 years. *The American Journal of Cardiology*, 117, 1884–1889.
- Jones, D. J., Bromberger, J. T., Sutton-Tyrrell, K., & Matthews, K. A. (2003). Lifetime history of depression and carotid atherosclerosis in middle-aged women. *Archives of General Psychiatry*, 60(2), 153–160.
- Joynt, K. E., Whellan, D. J., & O'Connor, C. M. (2003). Depression and cardiovascular disease: Mechanisms of interaction. *Biological Psychiatry*, 54(3), 249.
- Kubzansky, L. D., Davidson, K. W., & Rozanski, A. (2005). The clinical impact of negative psychological states: Expanding the spectrum of risk for coronary artery disease. *Psychosomatic Medicine*, 67(Suppl. 1), S10–S14.
- Leventhal, A. M., & Zvolensky, M. J. (2015). Anxiety, depression, and cigarette smoking: A transdiagnostic vulnerability framework to understanding emotion-smoking comorbidity. *Psychological Bulletin*, 141(1), 176–212.
- Lichtman, J. H., Froelicher, E. S., Blumenthal, J. A., Carney, R. M., Doering, L. V., Frasure-Smith, N., ... American Heart Association Statistics Committee of the Council on Epidemiology and Prevention and the Council on Cardiovascular and Stroke Nursing. (2014). Depression as a risk factor for poor prognosis among patients with acute coronary syndrome: Systematic review and recommendations: A scientific statement from the American Heart Association. *Circulation*, 129(12), 1350–1369.
- Lloyd-Jones, D. M., Hong, Y., Labarthe, D., Mozaffarian, D., Appel, L. J., Van Horn, L., ... American Heart Association Strategic Planning Task Force and Statistics Committee. (2010). Defining and setting national goals for cardiovascular health promotion and disease reduction: The American Heart Association's strategic impact goal through 2020 and beyond. *Circulation*, 121(4), 586–613.
- Lyons, M., Hitsman, B., Xian, H., Panizzon, M. S., Jerskey, B. A., Santangelo, S., ... Tsuang, M. T. (2008). A twin study of smoking, nicotine dependence, and major depression in men. *Nicotine & Tobacco Research*, 10(1), 97–108.
- Matthews, K. A., Owens, J. F., Edmundowicz, D., Lee, L., & Kuller, L. H. (2006). Positive and negative attributes and risk for coronary and aortic calcification in healthy women. *Psychosomatic Medicine*, 68(3), 355–361.
- McEvoy, J. W., Blaha, M. J., Rivera, J. J., Budoff, M. J., Khan, A. N., Shaw, L. J., ... Nasir, K. (2012). Mortality rates in smokers and nonsmokers in the presence or absence of coronary artery calcification. *JACC: Cardiovascular Imaging*, 5(10), 1037–1045.
- Mulle, J. G., & Vaccarino, V. (2013). Cardiovascular disease, psychosocial factors, and genetics: The case of depression. *Progress in Cardiovascular Diseases*, 55(6), 557–562.
- Nicholson, A., Kuper, H., & Hemingway, H. (2006). Depression as an aetiological and prognostic factor in coronary heart disease: A meta-analysis of 6362 events among 146 538 participants in 54 observational studies. *European Heart Journal*, 27(23), 2763–2774.
- O'Neil, A., Fisher, A. J., Kibbey, K. J., Jacka, F. N., Kotowicz, M. A., Williams, L. J., ... Pasco, J. A. (2016a). The addition of depression to the Framingham Risk Equation model for predicting coronary heart disease risk in women. *Preventive Medicine*, 87, 115–120.
- O'Neil, A., Fisher, A. J., Kibbey, K. J., Jacka, F. N., Kotowicz, M. A., Williams, L. J., ... Pasco, J. A. (2016b). Depression is a risk factor for incident coronary heart disease in women: An 18-year longitudinal study. *Journal of Affective Disorders*, 196, 117–124.
- Pratt, L. A., & Brody, D. J. (2010). Depression and smoking in the U.S. household population aged 20 and over, 2005–2008. *NCHS Data Brief*, 34, 1–8.
- Richards, C. S., Cohen, L. M., Morrell, H. E., Watson, N. L., & Low, B. E. (2013). Treating depressed and anxious smokers in smoking cessation programs. *Journal of Consulting and Clinical Psychology*, 81(2), 263–273.

- Shaw, L. J., Raggi, P., Callister, T. Q., & Berman, D. S. (2006). Prognostic value of coronary artery calcium screening in asymptomatic smokers and non-smokers. *European Heart Journal*, 27(8), 968–975.
- Stewart, J. C., Perkins, A. J., & Callahan, C. M. (2014). Effect of collaborative care for depression on risk of cardiovascular events: Data from the IMPACT randomized controlled trial. *Psychosomatic Medicine*, 76(1), 29–37.
- Stewart, J. C., Rand, K. L., Muldoon, M. F., & Kamarck, T. W. (2009). A prospective evaluation of the directionality of the depression-inflammation relationship. *Brain, Behavior, and Immunity*, 23(7), 936–944.
- Taylor, G., McNeill, A., Girling, A., Farley, A., Lindson-Hawley, N., & Aveyard, P. (2014). Change in mental health after smoking cessation: Systematic review and meta-analysis. *BMJ*, 348, g1151.
- USDHHS. (2014). *The health consequences of smoking—50 years of progress: A report of the surgeon general*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.
- Vulser, H., Wiernik, E., Tartour, E., Thomas, F., Pannier, B., Czernichow, S., ... Lemogne, C. (2015). Smoking and the association between depressive symptoms and absolute neutrophil count in the investigations preventives et cliniques cohort study. *Psychosomatic Medicine*, 77, 955.
- World Health Organization. (2013). *Global action plan for the prevention and control of noncommunicable diseases 2013-2020*. Geneva: Author.

Suggested Reading

- Goldstein, B. I., Carnethon, M. R., Matthews, K. A., McIntyre, R. S., Miller, G. E., Raghuveer, G., ... Hypertension and Obesity in Youth Committee of the Council on Cardiovascular Disease in the Young. (2015). Major depressive disorder and bipolar disorder predispose youth to accelerated atherosclerosis and early cardiovascular disease: A scientific statement from the American Heart Association. *Circulation*, 132(10), 965–986.
- Nicassio, P. M., Meyerowitz, B. E., & Kerns, R. D. (2004). The future of health psychology interventions. *Health Psychology*, 23(2), 132–137.
- Xian, H., Scherrer, J. F., Franz, C. E., McCaffery, J., Stein, P. K., Lyons, M. J., ... Kremen, W. S. (2010). Genetic vulnerability and phenotypic expression of depression and risk for ischemic heart disease in the Vietnam era twin study of aging. *Psychosomatic Medicine*, 72(4), 370–375.

Neurocognitive Disorders and Health Psychology

Claudia Drossel

*Department of Psychology, Eastern Michigan University College of Arts and Sciences,
Ypsilanti, MI, USA*

Neurocognitive disorder (NCD) describes a persistent pattern of difficulties remembering, thinking, reasoning, or problem solving that represents a decline from baseline functioning (APA, 2013). This functional decline can be due to a range of underlying conditions, some of which are reversible or stable, while others irreversible or progressive. Heterogeneous etiologies (e.g., NCD due to Alzheimer's and vascular disease) are common (see The Lancet series, "Dementia—not all about Alzheimer's," 2015). In addition to etiological subtype, NCDs are distinguished by their level of severity. When interference with activities of daily living is subtle and compensatory strategies allow independent completion of tasks, NCDs are classified as mild. Major NCDs—the focus of this encyclopedia entry—are customarily known as dementias and denote a significant disruption of activities of daily living secondary to neurodegenerative processes.

In the course of neurodegeneration, changes occur in multiple domains vital to everyday functioning, such as perception (e.g., loss of olfaction, diminished visuospatial skills, auditory processing deficits), cognition (e.g., deficits in complex attention, planning and execution of multistep actions, and learning or memory), or language (e.g., restricted conversational topics, difficulties naming common objects). Decreased motor skills and gait disturbances put the individual at risk of falls. In addition, sleep–wake rhythms may shift. While patterns of domain-specific strengths and weaknesses depend on the underlying neurological disorder as well as the person's distinct premorbid repertoire, in general, everyday tasks whose execution did not require effort or problem solving become increasingly puzzling and arduous. Adults younger than 60 years of age, whose most frequent conditions underlying the decline are alcohol-related NCD, frontotemporal lobar degeneration, or early-onset Alzheimer's disease, and their families also experience loss of employment, income, and social status.

Regardless of etiology, individuals with major NCD must cope with a progressive and insidious loss of skill sets, a narrowing life space, and concomitant changes in relationships that can seem perplexing and worrisome. Because coping repertoires, available social supports, and health status other than the neurological condition vary from person to person, physiology intersects with contextual and potentially modifiable factors to create psychological presentations unique to each individual. In other words, the manner with which major NCD manifests in the life of a particular person and the specific emotional or behavioral changes it produces greatly depend upon the individual's medical and psychosocial history and his or her current circumstances. Kahn (1965) coined the term "excess disability" to describe functional impairment in excess of what would be expected based upon a person's neurological status alone. Even after more than half a century of clinical research efforts to better support affected individuals and their family and professional supports, excess disability continues to be common in NCD and indicative of care situations that inadvertently hasten decline.

Individuals with major NCD eventually depend on others for virtually all tasks of daily living, including the most basic such as eating and toileting. In the United States, responsibility for care rests upon family members (hereinafter termed "family care partners," to underline the dyadic nature of care situations). The degree to which these family care partners are prepared to understand and effectively compensate for functional loss and idiosyncratic emotional or behavioral sequelae, and to establish a 24/7 care system including respite in the form of third-party assistance, directly affects quality of care. It also affects care partners' physical and emotional health in the long run.

Given the prevalence of NCD, excess disability, and the care responsibility of families, the role of health psychology in NCD is threefold:

- i To mitigate factors that contribute to excess disability when NCD is present.
- ii To promote the health of care partners whose responsibility for part or full-time care places them at risk for adverse physical and emotional sequelae.
- iii To prevent the onset of major NCD through policy initiatives and health behavior interventions that address known risk factors.

While age is a known risk factor for many progressive NCDs, such as late-onset Alzheimer's disease, health status and physical fitness in midlife are important predictors of later functional status and thus notable points of primary prevention. The general role of health psychology related to risk factors for major NCD—exogenous (acquired brain injury via exposure to neurotoxic substances or trauma), endogenous (vascular, metabolic), and behavioral (e.g., substance use, sedentary behavior)—is addressed elsewhere in this volume. For this reason, the current entry will focus on (1) and (2) above: the prevention of excess disability and the promotion of care partner health when NCD is present.

Prevention of Excess Disability in NCD

A subtle loss of verbal skills, including self-description and self-expression but also complex reasoning and comprehension, puts individuals with major NCD at risk of unmet physical and emotional needs as well as social withdrawal. Already in 1996, the American Academy of Neurology Ethics and Humanities Subcommittee emphasized that prevention and management of factors aggravating NCD are at the heart of care (Bernat et al., 1996). Accordingly, NCD-appropriate care includes the following:

- 1 Optimizing basic functioning as the foundation for quality of life by establishing:
 - a Eating and hydration routines, (i) to maintain adequate nutrition given altered olfactory and taste perception and (ii) to prevent urinary tract infections, constipation, and increased confusion due to dehydration.
 - b Routines for urinary and bowel functioning and implementation of voiding schedules to prevent incontinence episodes and related agitated or irritable behavior.
 - c Proper detection and correction of sensory loss as decreased ability to hear or see may increase confusion, social isolation, and fear. Because cognitive decline may interfere with the effectiveness of corrective devices (e.g., individuals may develop difficulties using bi- or trifocal glasses and sophisticated binaural hearing aids or may lose their ability to switch batteries when indicated), removing barriers to correction of sensory loss is important. Often, simpler solutions are indicated, such as pocket amplifiers or glasses that only correct for myopia.
 - d Access to regular physical activity, to prevent restlessness and facilitate sleep.
 - e Routines that promote restful sleep and maintain the sleep–wake cycle (Cipriani, Lucetti, Danti, & Nuti, 2015).
- 2 Detecting and managing comorbidities. As medical providers generally rely on patients' self-report, diminishing and inadequate self-descriptive skills place individuals with NCD at risk of undetected acute illnesses and unmanaged chronic conditions. Whereas these conditions commonly reduce the cognitive efficiency of a healthy person, they tend to produce significant cognitive, emotional, and behavioral changes in individuals whose neurological functioning is compromised. Family care partners and providers may misattribute these precipitous changes to the NCD and focus on correlated emotional and behavioral changes to the detriment of detecting medical conditions that—when unmanaged—exacerbate cognitive difficulties and associated behavior problems.

As an example, individuals with cognitive decline and diabetes may continue to be solely responsible for their diabetes medication administration and their blood glucose monitoring. Alternatively, family care partners may receive the information for diabetes management from the person with NCD. Behavioral and emotional changes may overshadow long-term hyperglycemia and short-term but potentially serious hypoglycemic episodes that are more common in individuals with diabetes and NCD. Apathy, irritability, and agitation—together with increased confusion the hallmarks of poor glycemic control in older adults—can function as active barriers to care partners assisting the person with diabetes-related self-care and requesting access to preventive medical visits. Indeed, some care partners may discount the need for diabetes care and prioritize management of behavioral and affective changes commonly associated with NCD, yet often due to preventable or manageable medical conditions.

Other common comorbid conditions that exacerbate cognitive decline are acute and chronic pain (e.g., from urinary tract infections, toothaches, headaches, or rheumatoid arthritis), hypertension, and chronic lung and heart diseases. Because seemingly neuropsychiatric presentations can hinder the detection of acute or chronic illnesses, including cancer, health psychology plays a central role in the advocacy for proper rule-outs and adherence to chronic disease management at the intersection of physical and neuropsychiatric health.

- 3 Closely monitoring medication effects. Adverse events related to medications are common and may manifest as emotional or behavioral disturbances, particularly in older adults and those with already compromised central nervous system functioning (Campanelli, 2012). Individuals with NCD due to Lewy body disease, for example, are exceptionally sensitive

to treatment with antipsychotics. Referral to a geriatric pharmacist for medication review may be indicated when the person with NCD is on more than five medications, when there are multiple prescribers and potentially redundant prescriptions, when the medication regimen is suspected to worsen cognitive functioning or includes medications that are inappropriate for older adults or individuals with NCD, and when there have been changes in cognitive, emotional, or behavioral functioning that are uncharacteristic of the person and comorbid medical conditions have been ruled out. Of note, medications prescribed for the management of NCD (e.g., cholinesterase inhibitors) are known to precipitate prescribing cascades, in which the prescription drug's adverse events are diagnosed as novel conditions treated with additional medications.

- 4 Preventing and addressing depression and anxiety. Depression and anxiety frequently precipitate cognitive decline, potentially signaling subtle physiological changes of yet insufficient severity to disrupt activities of daily living. Once cognitive decline begins to interfere with everyday functioning, task or psychosocial demands exceed the person's skill set, and self-protective responses emerge. First among these self-protective responses are frequently avoidance of activities and general disengagement. Accordingly, depression and anxiety are the most common neuropsychiatric complaints and further exacerbate or accelerate the progressive effects of the neurodegenerative disease. Appropriate behavioral activation, that is, arranging opportunities for meaningful activities that match the person's lifetime interests and current skill level, provides the shared activities that are important to a sense of belonging and produces positive health outcomes in NCD. Inclusion of the person with NCD in daily activities, to the best of his or her abilities, is vital to maintaining quality of life.
- 5 Detecting and reducing substance use. NCDs are prevalent among older adults with substance use disorders, and differential diagnosis of the etiology underlying cognitive decline is difficult. Avoidant coping style, history of substance use, and psychosocial factors such as social isolation or family conflict may further increase vulnerability to substance use when cognitive decline is present (Kuerbis, Sacco, Balzer, & Moore, 2014).
- 6 Recognizing and managing delirium superimposed on dementia. The presence of an NCD predisposes individuals to delirium, associated with precipitous decline, hospitalization or institutionalization, and increased mortality. The practice of health psychology in inpatient medical settings often involves alerting the medical team to the rule-out of delirium and implementing non-pharmacological management strategies (Inouye, Westendorp, & Saczynski, 2014).

In summary, NCDs are complex conditions that tend to occur in medical and psychosocial contexts whose combined influence on the progression of NCDs is profound (Mast, 2011). In the United States, the everyday implementation of recommendations for care and the prevention of excess disability falls to family members, mostly women.

Promoting the Health of Family Care Partners

As family care partners assume responsibility for monitoring the safety and well-being of the person with NCD, arranging compensatory and supportive settings, assisting with basic and instrumental activities of daily living, and managing emotional and behavioral reactions to cognitive loss, family care partners' physical and mental health conditions affect how well they can meet the heavy emotional, organizational, problem-solving, and physical demands inherent in caring for a person with NCD. Family care partners' physical status and risk for

clinical depression have been linked to potentially harmful caregiver behaviors, as have greater care needs and cognitive impairment of the person with NCD (Beach et al., 2005). In addition, situational constraints imposed by the need to provide supervision for safety often lead to loss of income, reduced access to preferred activities, increased social isolation, sleep deprivation, and poor health behaviors including inadequate healthcare utilization. In general, while also described as meaningful, the provision of care within the family is associated with an elevated risk of negative physical and mental health outcomes for the primary care partner, including cardiovascular disease and depression (e.g., Dassel & Carr, 2016).

Most programs to date have been developed to support family care partners in the implementation of care recommendations and do not intervene directly with the person with NCD. These programs can have single or multiple components, consisting of psychoeducation about NCD and associated skills training to enhance safety and to compensate for cognitive loss, introduction to behavioral management techniques to address behavioral or emotional changes, enhancement of problem solving and coping, or stress management including exercise. Psychotherapeutic strategies, such as cognitive behavioral therapies for depression, dialectical behavior therapy skills training, or mindfulness-based stress reduction, have also been applied. Outcome measures mostly have focused on factors that affect the ability to provide care, that is, care partner's mental and physical status. Not surprisingly, intervention effects are domain specific and vary depending on the target of the intervention. Care partners often are asked to report the frequency and the perceived impact of the difficulties exhibited by the person with NCD. It is assumed that reported reductions in frequency and/or impact of problematic behaviors translate to direct benefits of the intervention for the person with NCD. Reviews of the evidence have questioned this assumption and called for further systematic studies of patient functioning (Griffin et al., 2015; see also Schulz et al., 2002).

The progression of NCDs and the health of care partners interact with dyad or person-specific modifiable psychosocial, biobehavioral, and environmental factors within the purview of health psychology. In general, effective interventions for care partners that reduce burden and enhance mood and well-being are multifaceted and tailored to individual needs, and they require active participation (e.g., McCurry & Drossel, 2011). In addition, these interventions remove barriers to soliciting assistance (e.g., from friends or third-party agencies) and provide long-term support (Sörensen & Conwell, 2011).

Author Biography

Claudia Drossel is an assistant professor at Eastern Michigan University. Her work focuses on developing functional assessments and implementing behavioral interventions for individuals and families to increase quality of life and prevent or reduce emotional and behavioral changes associated with neurocognitive disorders.

References

- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders (5th edition): DSM-5*. Washington, DC: Author.
- Beach, S. R., Schulz, R., Williamson, G., Miller, L. S., Weiner, M. F., & Lance, C. E. (2005). Risk factors for potentially harmful informal caregiver behavior. *Journal of the American Geriatrics Society*, 53(2), 255–261. doi:10.1111/j.1532-5415.2005.53111.x

- Bernat, J. L., Beresford, H. R., Celesia, G. G., Cranford, R. E., McQuillen, M. P., Pellegrino, T. R., ... Wichman, A. (1996). Ethical issues in the management of the demented patient: The American Academy of Neurology Ethics and Humanities Subcommittee. *Neurology*, 46(4), 1180–1183. doi:10.1212/WNL.46.4.1180
- Campanelli, C. M. (2012). American Geriatrics Society updated Beers Criteria for potentially inappropriate medication use in older adults. *Journal of the American Geriatrics Society*, 60(4), 616–631. doi:10.1111/j.1532-5415.2012.03923.x
- Cipriani, G., Lucetti, C., Danti, S., & Nuti, A. (2015). Sleep disturbances and dementia. *Psychogeriatrics*, 15, 65–74. doi:10.1111/psyg.12069
- Dassel, K. B., & Carr, D. C. (2016). Does dementia caregiving accelerate frailty? Findings from the Health and Retirement Study. *The Gerontologist*, 56(3), 444–450. doi:10.1093/geront/gnu078
- Griffin, J. M., Meis, L. A., Greer, N., MacDonald, R., Jensen, A., Rutks, I., ... Wilt, T. J. (2015). Effectiveness of caregiver interventions on patient outcomes in adults with dementia or Alzheimer's disease: A systematic review. *Gerontology and Geriatric Medicine*, 1, 1–17. doi:10.1177/2333721415595789
- Inouye, S. K., Westendorp, R. G., & Saczynski, J. S. (2014). Delirium in elderly people. *The Lancet*, 383, 911–922. doi:10.1016/S0140-6736(13)60688-1
- Kahn, R. S. (1965). Comments. In *Proceedings of the York House Institute on the mentally impaired aged*. Philadelphia: Philadelphia Geriatric Center.
- Kuerbis, A., Sacco, P., Balzer, D. G., & Moore, A. A. (2014). Substance use among older adults. *Clinics in Geriatric Medicine*, 30(3), 629–654. doi:10.1016/j.cger.2014.04.008
- Mast, B. T. (2011). *Whole person dementia assessment*. Baltimore, MD: Health Professions Press, Inc.
- McCurry, S., & Drossel, C. (2011). *Treating dementia in context: A step-by-step guide to working with individuals and families*. Washington, DC: American Psychological Association.
- Orrell, M., & Brayne, C. (Eds.) (2015). Dementia—Not all about Alzheimer's. *The Lancet*, 386(10004), 1600.
- Schulz, R., O'Brien, A., Czaja, S., Ory, M., Norris, R., Martire, L. M., ... Stevens, A. (2002). Dementia caregiver intervention research: In search of clinical significance. *The Gerontologist*, 42(5), 589–602. doi:10.1093/geront/42.5.589
- Sörensen, S., & Conwell, Y. (2011). Issues in dementia caregiving: Effects on mental and physical health, intervention strategies, and research needs. *American Journal of Geriatric Psychiatry*, 19(6), 491–496. doi:10.1097/JGP.0b013e31821c0e6e

Suggested Reading

- Inouye, S. K., Westendorp, R. G., & Saczynski, J. S. (2014). Delirium in elderly people. *The Lancet*, 383, 911–922. doi:10.1016/S0140-6736(13)60688-1
- Mast, B. T. (2011). *Whole person dementia assessment*. Baltimore, MD: Health Professions Press, Inc.
- McCurry, S., & Drossel, C. (2011). *Treating dementia in context: A step-by-step guide to working with individuals and families*. Washington, DC: American Psychological Association.

Depression and Comorbidity in Health Contexts

C. Steven Richards¹ and Lee M. Cohen²

¹*Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA*

²*Department of Psychology, College of Liberal Arts, The University of Mississippi,
University, MS, USA*

Depression is associated with many chronic health conditions and diseases (Richards & O'Hara, 2014a). In fact, depressive comorbidity is observed so frequently in health contexts that it is important for healthcare providers to be aware of the presentation of the symptoms of depression, how to quickly assess if this is a concern with their patients, and how to structure treatment for multiple concerns simultaneously. It is also important to have an understanding of how depression can influence the course of a variety of physical illnesses (Richards & O'Hara, 2014b). In this brief entry, examples of the concurrence of symptoms of depression with chronic health conditions are provided, followed by a discussion of the implications of this body of research and scholarship.

Cancer and Depression

There is often a strong association between being diagnosed with cancer and experiencing significant symptoms of depression (e.g., see reviews by Miller & Massie, 2010; Nezu, Nezu, Greenberg, & Salber, 2014). In fact, there are large numbers of randomized controlled trials examining multiple approaches to treating depressed cancer patients. For example, Manne, Siegel, Heckman, and Kashy (2016) examined 302 female patients with early-stage breast cancer, along with their spouses. For the most distressed patients, a couple-focused and supportive version of group therapy appeared to be the most effective intervention. In contrast, among less distressed patients, a structured and skill-based version of group therapy appeared the most effective. This study successfully addressed some of the common research concerns

in this area, such as including a relatively large sample and a broad array of measures. It also highlights the need for individualized treatment, based on the specific needs of the patient.

Another recent study by Desautels, Savard, Ivers, Savard, and Caplette-Gingras (2018) illustrates an alternative approach to evaluating treatment options among depressed cancer patients. This study compared the efficacy of treating breast cancer patients that had considerable depressive symptoms with either cognitive therapy or bright light therapy. The design was a randomized controlled trial, with 62 breast cancer patients, and it included pre-, post-, and 6-month follow-up assessments. Although caution is warranted because of the relatively small sample of 62 participants, the cognitive therapy group appeared to be efficacious for this group of cancer patients, compared with a wait-list control group. The bright light therapy showed some promise for reducing depressive symptoms in these patients, but it was not as consistently efficacious across all depression measures when compared with those who participated in the cognitive therapy group. Extensions of this treatment approach to larger and more diverse cancer patient samples, and with longer follow-up assessments, should prove interesting. This bright light therapy approach may be particularly helpful in circumstances where the well-tested approaches of cognitive therapy or cognitive behavior therapy are not available, or not acceptable, to cancer patients.

Hopko and his colleagues have conducted several studies suggesting that behavioral activation may be an efficacious intervention for psychosocial variables, such as depression, in samples of depressed breast cancer patients (e.g., Ryba, Lejuez, & Hopko, 2014). For instance, Ryba et al. (2014) conducted a study suggesting that the level of patient compliance with behavioral activation homework is strongly associated with reduced severity in symptoms of depression among cancer patients. The quality of the completed homework, however, appears to be less strongly associated with outcomes following this behavioral activation intervention. Hopko et al. have also found support for problem-solving interventions in this context, as have others using various methodologies among diverse samples of cancer patients (e.g., Nezu, Nezu, Felgoise, McClure, & Houts, 2003).

Many additional studies have appeared in elite academic journals, such as the *Journal of Consulting and Clinical Psychology*, *Health Psychology*, *Annals of Behavioral Medicine*, *The Journal of the American Medical Association (JAMA)*, and *The New England Journal of Medicine*, that focus on depressive comorbidity among a multitude specific diseases such as cancer, heart disease, diabetes, arthritis, and neurological disease. The frequency in which this topic is included across these multidisciplinary journals underlines the importance of the topic and regularity of the concern in healthcare settings. Further, research in this area offers important treatment information and implications.

For instance, there is a rapidly expanding literature on interventions to improve several psychosocial aspects of being diagnosed with cancer, caring for someone with cancer, or functioning effectively at home and work while a family member is being treated for cancer. The treatment goals often include a reduction in diagnosable clinical depression or depressive symptoms. Thus, there are large studies supporting the efficacy of different forms of treatment among this population. Specifically, cognitive behavioral therapy and relaxation training interventions have been shown to be effective in this area (e.g., Gudenkauf et al., 2015). Mindfulness and hope components may also enhance interventions aimed at the psychosocial components of living with cancer and fears of cancer recurrence (e.g., Thornton et al., 2014).

Additionally, there are studies that also focus on the impact of cancer (and cancer treatment) on family, friends, and caregivers of cancer patients. Cancer caregivers have been shown to experience serious depressive symptoms after a loved one's cancer diagnosis. For instance, Kim, Shaffer, Carver, and Cannady (2014) illustrated that social support and caregiving stress may have implications for several years after the initial cancer diagnosis. Furthermore, these

investigators continue to find that caregivers may benefit from interventions for better adjustment to long-term caregiving, even 5 years after the initial diagnosis. Finally, a randomized controlled trial by Lewis et al. (2015) suggests that an intervention of educational counseling sessions may reduce the burden of cancer and the level of negative affect for mothers with cancer. Not only did this study find that treatment helped mothers' mood, but also it helped with their parenting skills and the behavioral and emotional adjustment of their children (range of 8–12 years old).

In summary, the few examples provided, selected from a very long list of potential examples, illustrate the important progress that is being made regarding depressive comorbidity and cancer.

Treating Depressed Smokers in Smoking Cessation Programs

As another brief example relevant to the topic of this entry, we mention the rapidly growing literature on treating depressed (and anxious) smokers as part of smoking cessation interventions. Smoking and nicotine use are typically listed among the most commonly preventable causes of disease and death (e.g., Richards, Cohen, Morrell, Watson, & Low, 2013). Moreover, high rates of depressive (and anxious) symptoms are quite prevalent among patients seeking smoking cessation treatment and can in fact impede treatment progress. Therefore, research on efficacious interventions for smoking cessation that includes one or more modules on depressive symptom reduction is important (see Richards et al., 2013, for a review of treating depressed and anxious smokers in smoking cessation programs).

A recent example of this type of research is a study of the synergistic effect of a change in moderate depressive symptoms (dysphoria) and anxiety sensitivity, on post-quit tobacco withdrawal symptoms (Bakhshaie et al., 2018). This prospective research trial included 198 treatment-seeking adult smokers, who were enrolled in a smoking cessation treatment study. This large study included a broad array of measures and a 12-week post-quit assessment. The results indicated that reducing participant levels of depressive symptoms (dysphoria) and anxiety sensitivity before quitting is strongly related to the reduction withdrawal symptoms (which are highly associated with relapse to smoking) after a quit attempt. In turn, numerous studies suggest that reducing withdrawal symptoms after a quit attempt among participants in smoking cessation programs is often associated with better long-term treatment outcomes (e.g., Baker et al., 2007). Furthermore, less depression (and anxiety) post-quit attempt is also associated with a lesser probability of smoking relapse (Richards et al., 2013).

In summary, there is a rapidly developing research literature on depressive comorbidity and health-compromising behaviors such as smoking, particularly in the context of healthcare settings and treatment programs.

Depressive Comorbidity and Diversity

It is also important to discuss a couple examples of research that addresses depressive comorbidity as it relates to ethnic diversity in health contexts. For example, Le, Perry, and Stuart (2011) conducted a randomized controlled trial that illustrates the potential of a culturally sensitive cognitive behavior therapy intervention for a sample of 217 Latinas that were classified as high risk for comorbid challenges of perinatal depression, chronic health problems, and distressed relationships with their children and family. The majority of the participants were low-income immigrants from Central and South America. The cognitive behavior therapy intervention was

tailored to the precise needs of this sample. Specifically, the intervention was delivered in Spanish by bilingual and bicultural staff, and it was developed to be culturally sensitive to the ethnic and economic circumstances of these participants. The participants were randomized into the adapted cognitive behavior therapy condition (eight group sessions, plus three booster sessions) or a usual-care condition. The results suggested that the cognitive behavior therapy intervention significantly reduced depressive symptoms. Major depressive episodes, however, which are more severe and rare in this sample, were not significantly reduced by this brief intervention, compared with usual care. This study illustrates that a culturally sensitive version of cognitive behavior therapy offers promise for reducing depressive symptoms in high-risk, low-income Latina women who were at risk for chronic health problems, during their pregnancy.

Another relevant example that considers the challenge of depressive comorbidity, among a diverse sample, is a study on physical activity and depressive symptoms after treatment for breast cancer (Brunet, O'Loughlin, Gunnell, & Sabiston, 2018). The early research in this area has been limited to samples of almost entirely Caucasian women, within a small age range, who were relatively well educated and mostly married or living with a partner. In contrast, this large study of 201 women with breast cancer reflected efforts to recruit a more diverse sample across a multitude of demographic variables. The investigators note that while they were somewhat successful, even more diverse samples will be important for future research. One of the major findings from this study is that more physical exercise was associated with less frequent depressive symptoms, measured in a cross-sectional manner, among women post-treatment for breast cancer. This said, not all of the predicted relationships of depression and exercise held up longitudinally.

Discussion

Depressive comorbidity is pervasive in health contexts. This has important implications for assessment, treatment, follow-up care, and long-term survivorship. Although the fields of health psychology and behavioral medicine have recently embraced the merits of recruiting diverse samples in their studies, it is clear that depressive comorbidity is frequently present in underserved populations, high-risk groups, and ethnically diverse minority populations. Unfortunately, the implications of depressive comorbidity are serious and negative. These implications include assessment and evaluation complexities, threats to effective treatment planning, poorer adherence to treatment regimens, increased rates of relapse and recurrence after treatment, more disruptions and distress in day-to-day functioning, and a more negative overall prognosis for long-term morbidity and mortality. Despite these challenges, considerable progress has been made regarding the theory, research, and practice of effectively dealing with depressive comorbidity in health contexts. Clearly, depression or depressive symptoms present concurrently with a majority of the chronic health problems and diseases that clinicians help patients cope with daily.

Included below are a few “take-home messages” that may be culled from this entry and the much broader research literature on depression and comorbidity in health contexts:

- This area struggles with clarifications and distinctions between “depressive symptoms” versus “severe clinical depression,” which is defined as meeting the diagnostic criteria of major depressive disorder in the *DSM-5* (American Psychiatric Association, 2013).
- Some wonderfully ambitious and elegant studies have been designed and completed in this area. This said, any scientific, psychological, medical, and health area can be improved. For instance, future studies must include large, more diverse samples to more accurately reflect the world's population.

- The specifics of depressive comorbidity in health contexts are complex, as researchers and clinicians move from one type of comorbidity to the next, one problem to the next, and one context to the next. Therefore, extra effort and consideration may be warranted for assessment and treatment.
- The challenges of depressive comorbidity in health contexts argue for considering the merits of inter- and multidisciplinary efforts. Therefore, this seems to be an area where different disciplines working effectively together is critical.
- It appears that depression presents with almost all chronic health problems and diseases in health contexts. Therefore, it will be helpful to screen for depressive comorbidity, plan accordingly, and treat thoroughly.

Author Biographies

C. Steven Richards, PhD, is a professor of psychological sciences at Texas Tech University. His research interests include depression, clinical health psychology and behavioral medicine, comorbidity of health problems and psychopathology, self-control, and relapse prevention for depression. He has held 15 administrative positions during his faculty appointments at Texas Tech University, Syracuse University, and the University of Missouri, Columbia. Dr. Richards earned his PhD in clinical psychology at the State University of New York at Stony Brook (now Stony Brook University).

Lee M. Cohen, PhD, is dean of the College of Liberal Arts and professor in the Department of Psychology at the University of Mississippi. Dean Cohen came to UM from Texas Tech University, where he served in a number of administrative roles including the director of the nationally accredited doctoral program in clinical psychology, and the chair of the Department of Psychological Sciences. As a faculty member, he received several university-wide awards for his teaching and academic achievement. As a researcher, he received more than \$1.5 million from funding agencies, including the US Department of Health and Human Services, the National Science Foundation, and the National Institutes of Health/National Institute on Drug Abuse. His research program examines the behavioral and physiological mechanisms that contribute to nicotine use, and he worked to develop optimal smoking cessation treatments. He is a fellow of the American Psychological Association and received his PhD in clinical psychology from Oklahoma State University.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed., DSM-5). Washington, DC: Author.
- Baker, T. B., Piper, M. E., McCarthy, D. E., Bolt, D. M., Smith, S. S., Kim, S. Y., ... Toll, B. A. (2007). Time to first cigarette in the morning as an index of ability to quit smoking: Implications for nicotine dependence. *Nicotine & Tobacco Research*, 9(Suppl. 4), S555–S570.
- Bakhshaie, J., Kulesz, P. A., Garey, L., Langdon, K. J., Businelle, M. S., Leventhal, A. M., ... Zvolensky, M. J. (2018). A prospective investigation of the synergistic effect of change in anxiety sensitivity and dysphoria on tobacco withdrawal. *Journal of Consulting and Clinical Psychology*, 86, 69–80.
- Brunet, J., O'Loughlin, J. L., Gunnell, K. E., & Sabiston, C. M. (2018). Physical activity and depressive symptoms after breast cancer: Cross-sectional and longitudinal relationships. *Health Psychology*, 37, 14–23.
- Desautels, C., Savard, J., Ivers, H., Savard, M.-H., & Caplette-Gingras, A. (2018). Treatment of depressive symptoms in patients with breast cancer: A randomized controlled trial comparing cognitive therapy and bright light therapy. *Health Psychology*, 37, 1–13.

- Gudenkauf, L. M., Antoni, M. H., Stagl, J. M., Lechner, S. C., Jutagir, D. R., Bouchard, L. C., ... Carver, C. S. (2015). Brief cognitive-behavioral and relaxation training interventions for breast cancer: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 83*, 677–688.
- Kim, Y., Shaffer, K. M., Carver, C. S., & Cannady, R. S. (2014). Prevalence and predictors of depressive symptoms among cancer caregivers 5 years after the relative's cancer diagnosis. *Journal of Consulting and Clinical Psychology, 82*, 1–8.
- Le, H. N., Perry, D. F., & Stuart, E. A. (2011). Randomized controlled trial of a preventive intervention for perinatal depression in high-risk Latinas. *Journal of Consulting and Clinical Psychology, 79*, 135–141.
- Lewis, F. M., Brandt, P. A., Cochrane, B. B., Griffith, K. A., Grant, M., Haase, J. E., ... Shands, M. E. (2015). The enhancing connections program: A six-state randomized clinical trial of a cancer parenting program. *Journal of Consulting and Clinical Psychology, 83*, 12–23.
- Manne, S. L., Siegel, S. D., Heckman, C. J., & Kashy, D. A. (2016). A randomized clinical trial of a supportive versus a skill-based couple-focused group intervention for breast cancer patients. *Journal of Consulting and Clinical Psychology, 84*, 668–681.
- Miller, K., & Massie, M. J. (2010). Depressive disorders. In J. C. Holland, W. S. Breitbart, P. B. Jacobsen, M. S. Lederberg, M. J. Loscalzo, & R. McCorkle (Eds.), *Psycho-oncology* (2nd ed., pp. 311–318). New York, NY: Oxford University Press.
- Nezu, A. M., Nezu, C. M., Felgoise, S. H., McClure, K. S., & Houts, P. S. (2003). Project genesis: Assessing the efficacy of problem-solving therapy for distressed adult cancer patients. *Journal of Consulting and Clinical Psychology, 71*, 1036–1048.
- Nezu, A. M., Nezu, C. M., Greenberg, L. M., & Salber, K. E. (2014). Cancer and depression. In C. S. Richards & M. W. O'Hara (Eds.), *The Oxford handbook of depression and comorbidity* (pp. 302–318). New York, NY: Oxford University Press.
- Richards, C. S., Cohen, L. M., Morrell, H. E. R., Watson, N. L., & Low, B. E. (2013). Treating depressed and anxious smokers in smoking cessation programs. *Journal of Consulting and Clinical Psychology, 81*, 263–273.
- Richards, C. S., & O'Hara, M. W. (Eds.) (2014a). *The Oxford handbook of depression and comorbidity*. New York, NY: Oxford University Press.
- Richards, C. S., & O'Hara, M. W. (Eds.) (2014b). Introduction. In *The Oxford handbook of depression and comorbidity* (pp. 1–10). New York, NY: Oxford University Press.
- Ryba, M. M., Lejuez, C. W., & Hopko, D. R. (2014). Behavioral activation for depressed breast cancer patients: The impact of therapeutic compliance and quantity of activities completed on symptom reduction. *Journal of Consulting and Clinical Psychology, 82*, 325–335.
- Thornton, L. M., Cheavens, J. S., Heitzmann, C. A., Dorfman, C. S., Wu, S. M., & Anderson, B. L. (2014). Test of mindfulness and hope components in a psychological intervention for women with cancer recurrence. *Journal of Consulting and Clinical Psychology, 82*, 1087–1100.

Suggested Reading

- Bakhshaie, J., Kulesz, P. A., Garey, L., Langdon, K. J., Businelle, M. S., Leventhal, A. M., ... Zvolensky, M. J. (2018). A prospective investigation of the synergistic effect of change in anxiety sensitivity and dysphoria on tobacco withdrawal. *Journal of Consulting and Clinical Psychology, 86*, 69–80.
- Desautels, C., Savard, J., Ivers, H., Savard, M.-H., & Caplette-Gingras, A. (2018). Treatment of depressive symptoms in patients with breast cancer: A randomized controlled trial comparing cognitive therapy and bright light therapy. *Health Psychology, 37*, 1–13.
- Manne, S. L., Siegel, S. D., Heckman, C. J., & Kashy, D. A. (2016). A randomized clinical trial of a supportive versus a skill-based couple-focused group intervention for breast cancer patients. *Journal of Consulting and Clinical Psychology, 84*, 668–681.
- Richards, C. S., & O'Hara, M. W. (Eds.) (2014a). *The Oxford handbook of depression and comorbidity*. New York, NY: Oxford University Press.

Depression and Relapse in Health Contexts

C. Steven Richards¹ and Lee M. Cohen²

¹*Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA*

²*Department of Psychology, College of Liberal Arts, The University of Mississippi, University, MS, USA*

In 1978, Richards and Perri noted, in the context of a treatment maintenance study, that psychosocial treatment effects often do not last (Richards & Perri, 1978). Unfortunately, four decades later, this is still the case. Hence, depression is an excellent example of the complex challenges faced when working with individuals to prevent relapse and recurrence (Richards & Perri, 2010a). Further, it often presents along with many health conditions and diseases (Richards & O'Hara, 2014a). In fact, depressive comorbidity is so frequently observed in healthcare settings that healthcare providers from all backgrounds need to have some understanding of this disorder as well as the impact it has on the primary medical concern for which their patients are seeking treatment (Richards & O'Hara, 2014b). In this entry, examples of depressive relapse and recurrence, concurrent with serious health conditions, are presented. Implications of this research and scholarship, as well as recommendations for preventing and reducing depressive relapse, are also discussed.

Depression: Definition and Prevalence

Depression is a chronically relapsing and recurring condition (American Psychiatric Association [APA], 2013). It is also quite prevalent, with approximately 7–10% of the adult population in the United States and other industrialized countries experiencing depression or a closely related mood disorder at a given point in time (Gotlib & Hammen, 2015; Kessler & Ustun, 2008). For the purpose of this entry, the term “depression” will be used as the general equivalent for major depressive disorder, as defined in the *DSM-5* (APA, 2013). This said, it is important to note that this use of the term “depression” is not universal. In fact, some investigators use

“depression” to indicate diagnostically subthreshold symptoms and signs, such as elevated symptom levels on a self-report measure, rather than the interview-based observational assessments that are the base of *DSM-5* diagnoses. In research settings, mingling the concepts of self-reported “subthreshold depressive symptoms,” “depression,” and interview-based “major depressive disorder” is somewhat controversial (e.g., see discussions in APA, *DSM-5*, 2013; Gotlib & Hammen, 2015; Richards & Perri, 2002; Watson & O’Hara, 2017).

In summary, depression is a serious and debilitating disorder, with negative implications for day-to-day functioning, disruptions in close relationships at work and at home, deterioration in chronic health conditions and diseases, and an overall negative impact on morbidity and mortality (Kessler, Scott, Shahly, & Zaslavsky, 2014). Indeed, most large national and international surveys, such as those conducted by the US National Institute of Mental Health and the World Health Organization, have indicated that serious clinical depression, such as major depressive disorder, is always high on the list of causes for day-to-day disruptions in functioning and increased disability (Gotlib & Hammen, 2015; Kessler & Ustun, 2008). Moreover, there is a large literature associating depression, and depressive relapse, with a variety of chronic health conditions and diseases. Therefore, it is important that depressive relapse is assessed over time, and when necessary, treated in health contexts in accordance with approved clinical interventions.

Studies on Relapse Prevention for Depression

Frank, Kupfer, Reynolds, and their colleagues have conducted a very ambitious series of randomized controlled trials examining relapse prevention for depression (e.g., Frank et al., 1990; Reynolds et al., 2006). These studies include long-term follow-ups and comparison of treatment maintenance interventions. Specifically, after successful completion of treatment (often 12–20 weeks), Frank and her colleagues compared various combinations of a psychosocial intervention and an antidepressant medication. The psychosocial intervention studied during both acute treatment and maintenance therapy was interpersonal psychotherapy (IPT) for depression, which is a brief form of psychotherapy that focuses on an array of interpersonal issues and skills (see Weissman, Markowitz, & Klerman, 2017 for a review of IPT). The antidepressant medications used across these studies were FDA-approved antidepressant medications (e.g., imipramine [*Tofranil*], a tricyclic antidepressant medication, or a selective serotonin reuptake inhibitor [SSRI] such as paroxetine [*Paxil*]). A review of this group’s extensive body of research along with other relevant studies on IPT is available in O’Hara, Schiller, and Stuart (2010).

Several conclusions can be drawn from this important work, which utilized various combinations of IPT and antidepressant medications as relapse-prevention strategies for depressed adults. First, the continuation of maintenance treatment for depression following successful acute treatment, with IPT, antidepressant medications, or both, usually leads to reductions in the rate of depressive relapse and recurrence over multiyear follow-ups (e.g., Frank et al., 1990, 2007; Reynolds et al., 1999, 2006). For example, the Reynolds et al. study (1999) with depressed older adults found that 90% of the participants relapsed across a 3-year follow-up in the maintenance group that received a placebo during follow-up. However, only 20% of participants relapsed in the maintenance group that received IPT and antidepressant medication during follow-up. Adding to this impressive finding is the fact that it was with older adults, a population that often has chronic health conditions. This is an extremely important finding (however, compare with Reynolds et al., 2006, with even older-old adults, over 70 years of age, where the maintenance effect of long-term IPT was not particularly powerful).

Second, there is support for maintenance IPT as a stand-alone relapse-prevention strategy among adult women, which is important, as some women may be reluctant to take long-term antidepressants due to pregnancy or postpartum breastfeeding (Frank et al., 2007). Since depression and depressive relapse are highly prevalent, and frequently comorbid with other health concerns, approaches to reducing depressive relapse are very important.

Third, given findings from several randomized controlled trials and/or more naturalistic, low-cost intervention trials in point-of-healthcare clinics with depressed postpartum women, IPT interventions in the acute phase of depression treatment, and/or during maintenance treatment, appear to be helpful for depressive relapse prevention with women caring for young children (e.g., Nylén et al., 2010; O'Hara, Stuart, Gorman, & Wenzel, 2000; Serge, Brock, & O'Hara, 2015). Therefore, even in economically challenged and underserved populations, IPT apparently has potential as both an acute and maintenance treatment for depression (e.g., Serge et al., 2015).

In addition to the promising research examining the effectiveness of IPT, there is also an extensive research literature evaluating the benefits of cognitive behavior therapy (CBT), or more cognitively focused cognitive therapy (CT), as a relapse-prevention strategy in depressed adults. As with the treatment maintenance and depressive relapse-prevention research with IPT, the studies with CBT and CT have often been quite promising in support of this approach (e.g., for studies see Hollon, DeRubeis, et al., 2005; Jarrett et al., 2001; for a review see Hollon, Stewart, & Strunk, 2005). In summary, there are few, if any, evidence-based psychotherapy for depression approaches with as much evaluation using large randomized controlled trials as CBT and CT. Overall, this well-researched psychosocial intervention has also shown strong potential as a relapse-prevention strategy for depressed adults.

Finally, there has also been encouraging research investigations of mindfulness-based versions of CT (and CBT) in the area of depressive relapse prevention. Recent meta-analytic reviews of this research indicate considerable potential for mindfulness-based therapy for relapse prevention of depression. For example, a meta-analysis of over 1,200 individuals with recurrent depression, collected across nine research trials, suggested that patients receiving mindfulness-based interventions (sometimes combined with antidepressant medication) showed less depressive relapse by about 23% compared with patients who were continued on antidepressant medication but who did not receive the mindfulness therapy (Kuyken et al., 2016). Researchers, scholarly reviewers, and clinicians who work with individuals diagnosed with depression typically consider a 23% reduction in depressive relapse to be an important clinical finding (Gotlib & Hammen, 2015; Richards & Perri, 2010a).

These studies on mindfulness-based interventions were also typically conducted considering the constraints of real-world conditions, such as the use of time-limited psychological treatment. Specifically, the study designs compared 8 weeks of a mindfulness version of CT with "usual care," where "usual care" primarily consisted of antidepressant medication treatment and did not include an evidence-based psychotherapy. Therefore, this research by Kuyken et al. (2016) offers hope that mindfulness-based therapies, as with the more traditional CT and CBT therapies developed before them, may help depressed patients significantly decrease depressive relapse rates.

Examples of Depressive Relapse in the Health Context

There are many potential examples of depression and depressive relapse in the health context. Moreover, this research and clinical literature is expanding rapidly, attracting increased

attention from investigators, editors, and clinicians. For example, a recent “special section” of the *Journal of Consulting and Clinical Psychology* (Davila & Safren, 2017) focused on “sexual and gender minority health.” For instance, an ambitious study presented in this special section, by Choi, Batchelder, Ehlinger, Safren, and O’Cleirigh (2017), illustrates recent work on depression and depressive relapse in the context of important health behaviors. The investigators used network analysis to investigate the comorbidity of depression, PTSD, and sexual risk behaviors in the context of sexual minority men who had histories of trauma. Their sample included 296 sexual minority men who were HIV negative and living in urban environments. The findings suggested that comorbid depression and PTSD, and relapse for either disorder, were strongly related to increases in sexual risk behaviors, which in turn are often related to significant health problems and diseases (Choi et al., 2017).

Another example is a recent study by Desautels, avar, Ivers, Savard, and Caplette-Gingras (2018), which evaluated treatment options in anticipation of enhancing relapse prevention, among depressed cancer patients. This study compared the efficacy of treating breast cancer patients that had considerable depressive symptoms with either CT or bright light therapy. The design was a randomized controlled trial, with 62 breast cancer patients, and it included pre-, post-, and 6-month follow-up assessments. Although caution is warranted because of the relatively small sample of 62 participants, CT appeared to be efficacious for this group of cancer patients, compared with a wait list control group. The bright light therapy showed some promise for *briefly* reducing depressive symptoms among these patients, but it was not as consistently efficacious as CT across all depression measures or at the follow-up assessments. Moreover, bright light therapy appeared to be more vulnerable, than CT, to the enrolled patients having experiences with depressive relapse during follow-up. Extensions of this treatment approach to larger and more diverse cancer patient samples, and with longer follow-up assessments, could prove interesting. This bright light therapy approach may be particularly helpful in circumstances where the well-tested approaches of CT or CBT are not available, or not acceptable, to cancer patients.

Finally, some of the recent research that is relevant to depressive relapse investigates depression and other important health-related behaviors in the context of longitudinal research in the natural environment, rather than the randomized controlled trials that were highlighted earlier. An interesting example is the study by Trucco, Villafuerte, Hussong, Burmeister, and Zucker (2018). These investigators explored the longitudinal pathways to alcohol use and abuse among 426 adolescents who had genetic risk factors for addiction (e.g., their biological father had been charged with drunk driving and met diagnostic criteria for alcohol use disorder), which the researchers predicted would be moderated by depression and depressive relapse during the last wave of the study (adolescents aged 15–17 years). Thus, they predicted that depression would be related to more substance use and would essentially facilitate the expression of the genetic risk factors. Trucco et al. (2018) also predicted that ineffective coping methods for stress would be a moderator, related to greater substance use. It was found that ongoing depression (or depressive relapse), along with ineffective coping methods, appears to be a pathway that allows genetic risk factors to influence substance use and abuse. This study also represents some of the research trends toward excellence in clinical health psychology and behavioral medicine, noted in other entries in this volume. These trends include use of a large sample ($N = 426$, plus a comparison sample), which was followed longitudinally over many years (ages 3–17, in 5 waves), using multimodal and diverse measures (multi-method and multi-source) that included biological and psychological variables.

Discussion

Overview

Treatment effects often do not last. Patients and their healthcare providers are frequently surprised, and disappointed, that the psychological improvements and associated health benefits that may occur during, and soon after, acute psychological treatment will disappear over longer periods of time. Depression and depressive relapse are clear examples of this problem. Without evidence-based care, along with thorough attention to empirically supported relapse-prevention strategies, most depressed patients will experience relapse or recurrence within a few years of treatment completion. Clinical depression, such as major depressive disorder, is a chronic and relapsing condition. Recent studies suggest that depressive relapse can be reduced, even in high-risk populations such as older adults, through a combination of evidence-based therapies such as IPT, CBT, and antidepressant medication, along with maintenance follow-up care that can last several years.

In sum, four implications can be culled from this entry and the much broader research literature on depression and relapse in health contexts. Noted below are also a few of the specific relapse-prevention strategies for depression that are supported by empirical research (see Gotlib & Hammen, 2015; Hollon, Stewart, et al., 2005; Richards & Perri, 2010a, for reviews of this literature):

- This area struggles with clarifications and distinctions between “depressive symptoms” versus “severe clinical depression” meeting the diagnostic criteria of major depressive disorder in the *DSM-5* (APA, 2013; see Watson & O’Hara, 2017, for a recent discussion of assessment and diagnostic issues regarding depression). Patients, clinicians, and researchers will find it helpful to keep these distinctions in mind.
- Many of the randomized controlled trials and naturalistic longitudinal studies in this area are models of excellent research in terms of large and diverse samples, psychometrically sophisticated and multi-method measures, and long-term follow-up evaluations. This excellent research is helpful and inspiring.
- Research on relapse prevention for depression is growing rapidly. Examples of journals that regularly publish sophisticated randomized controlled trials on this topic include *Journal of Consulting and Clinical Psychology*, *JAMA Psychiatry* (formerly *Archives of General Psychiatry*), *American Journal of Psychiatry*, *British Journal of Psychiatry*, and general medical journals such as the *New England Journal of Medicine*, *JAMA*, and *The Lancet*.
- It appears that depressive relapse is likely. It seems that depression is associated with many health problems, and as such, screening for risk regarding depressive relapse, building in relapse prevention and maintenance treatment, and assessing for patient progress and durable improvement during follow-up evaluations are warranted.

Five Relapse-Prevention Strategies for Depression

- 1 *Teach patients effective coping skills.* Patients with severe depression will usually find it helpful to think about their personal problems more positively, regulate their emotion in a better manner, and behave more adaptively and strategically. Effective coping skills can accomplish these goals. Moreover, these coping skills may become durable (e.g., see studies by Hollon, DeRubeis, et al., 2005; Reynolds et al., 1999, 2006; for reviews see Hollon, Stewart, et al., 2005; Nezu, Nezu, Greenberg, & Salber, 2014; Richards & Perri, 2010a, 2010b).

- 2 *Accommodate the patient's circumstances, diversity, and membership in special populations.* As with any treatment, relapse-prevention strategies for depression will be most effective if they are tailored to the specific circumstances, diversity, and membership in special populations. For instance, a 75-year-old widowed Latina grandmother with chronic health problems, who is living in a nursing home but has frequent visits from her Latina daughter and grandchildren, may want a different relapse-prevention focus than a 29-year-old single Caucasian male, in relatively good physical health, but with chronic depression and alcohol abuse challenges. The vast research literatures on depression and health psychology include many examples where diversity, context, and special population membership make a huge difference (e.g., Davila & Safren, 2017, *JCCP* special issue; for reviews see Gotlib & Hammen, 2015; Smith, 2010).
- 3 *Assess carefully and follow-up thoroughly for the chronic health problems that are often associated with depression and depressive relapse.* As mentioned several times throughout this entry, depression is associated with other problems, including chronic health conditions and diseases such as coronary heart disease, cancer, arthritis, chronic pain, and neurological conditions such as multiple sclerosis. Depressive comorbidity with these health conditions usually has a negative impact on the patient, including more complicated treatment regimens, poorer adherence to medications and health promotion strategies, more risk for relapse and recurrence, and negative predictions for morbidity and mortality. Depressive relapse and recurrence will also have many negative impacts. Therefore, the comorbidity of depressive relapse and chronic health problems should be carefully assessed, thoroughly treated, monitored throughout follow-up, and considered for long-term continuation interventions, after acute treatment, which may prevent relapse (e.g., for studies see Frank et al., 1990, 2007; Nylén et al., 2010; O'Hara et al., 2000; e.g., for reviews see O'Hara et al., 2010; Richards, Cohen, Morrell, Watson, & Low, 2013; Smith, 2010).
- 4 *Carefully evaluate and intervene, as much as possible, with the depressed patient's social-support network.* Depression is typically a "socially based disorder," with disruption and dysfunction in close relationships at work and home being quite common in the more severe forms of depression. If a depressed patient's social world stays chaotic and not reinforcing, then it is almost inevitable that the patient will experience depressive relapse and recurrence. Depression disrupts close relationships, and dysfunctional relationships can lead to symptoms of depression (e.g., for reviews see Richards & O'Hara, 2014a; Richards & Perri, 2010a, 2010b; Stroud, Feinstein, Bhatia, Hershenberg, & Davila, 2014).
- 5 *Consider the patient's treatment preferences in relapse-prevention strategies.* Some patients prefer antidepressant medication regimens and consider the side effects minor or at a minimum tolerable. However, some patients dislike antidepressant medication and find the side effects very difficult to manage. A similar like-dislike relationship to treatment types can be observed with evidence-based psychotherapies. For example, many patients appreciate the strong evidence base of CBT, the relatively simple set of principles and goals, and the ease with which it can be combined with additional treatment strategies like activity scheduling, problem solving, skill building, and role-playing of challenging social interactions. Some patients, however, do not like the frequent homework assignments, the focus on cognitive errors and schema change, and the minimizing of some classic psychotherapeutic issues such as childhood and family psychodynamic issues, insight into personality styles, and transference. An analogous line of pros and cons, with different details, could be given for other evidence-based therapies, such as IPT. What is clear from the literature is that if a patient does not like their treatment, they are unlikely

to keep doing it after acute treatment ends (and this assumes they are engaged during acute treatment, which is often not the case). Thus, depressive relapse is likely to follow (e.g., for reviews see Gotlib & Hammen, 2015; Nezu et al., 2014; Richards & Perri, 2010a, 2010b).

Author Biographies

C. Steven Richards, PhD, is a professor of psychological sciences at Texas Tech University. His research interests include depression, clinical health psychology and behavioral medicine, comorbidity of health problems and psychopathology, self-control, and relapse prevention for depression. He has held 15 administrative positions during his faculty appointments at Texas Tech University, Syracuse University, and the University of Missouri–Columbia. Dr. Richards earned his PhD in clinical psychology at the State University of New York at Stony Brook (now Stony Brook University).

Lee M. Cohen, PhD, is dean of the College of Liberal Arts and professor in the Department of Psychology at the University of Mississippi. Dr. Cohen came to the University of Mississippi from Texas Tech University, where he served in a number of administrative roles including the director of the nationally accredited doctoral program in clinical psychology and the chair of the Department of Psychological Sciences. As a faculty member, he received several university-wide awards for his teaching and academic achievement. As a researcher, he received more than \$1.5 million from funding agencies, including the US Department of Health and Human Services, the National Science Foundation, and the National Institutes of Health/National Institute on Drug Abuse. His research program examines the behavioral and physiological mechanisms that contribute to nicotine use, and he worked to develop optimal smoking cessation treatments. Dr. Cohen is a fellow of the American Psychological Association and the Society of Behavioral Medicine. He received his PhD in clinical psychology from Oklahoma State University.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed., *DSM-5*). Washington, DC: Author.
- Choi, K. W., Batchelder, A. W., Ehlinger, P. P., Safren, S. A., & O’Cleirigh, C. (2017). Applying network analysis to psychological comorbidity and health behavior: Depression, PTSD, and sexual risk in sexual minority men with trauma histories. *Journal of Consulting and Clinical Psychology, 85*, 1158–1170.
- Davila, J., & Safren, S. A. (Eds.). (2017). Introduction to the special section on sexual and gender minority health. *Journal of Consulting and Clinical Psychology, 85*, 1109–1110. (Special section in *JCCP*, 2017, pp. 1109–1181)
- Desautels, C., Savard, J., Ivers, H., Savard, M.-H., & Caplette-Gingras, A. (2018). Treatment of depressive symptoms in patients with breast cancer: A randomized controlled trial comparing cognitive therapy and bright-light therapy. *Health Psychology, 37*, 1–13.
- Frank, E., Kupfer, D. J., Buysse, D. J., Swartz, H. A., Pilkonis, P. A., Houck, P. R., ... Stapf, D. M. (2007). Randomized trial of weekly, twice-monthly, and monthly interpersonal psychotherapy as maintenance treatment for women with recurrent depression. *American Journal of Psychiatry, 164*, 761–767.

- Frank, E., Kupfer, D. J., Perel, J. M., Cornes, C., Jarrett, D. B., Mallinger, A. G., ... Grochocinski, V. J. (1990). Three-year outcomes for maintenance therapies in recurrent depression. *Archives of General Psychiatry*, 47, 1093–1099.
- Gotlib, I. H., & Hammen, C. L. (Eds.) (2015). *Handbook of depression* (3rd ed.). New York, NY: Guilford Press.
- Hollon, S. D., DeRubeis, R. J., Shelton, R. C., Amsterdam, J. D., Salomon, R. M., O'Reardon, J. P., ... Gallop, R. (2005). Prevention of relapse following cognitive therapy vs. medications in moderate to severe depression. *Archives of General Psychiatry*, 62, 417–422.
- Hollon, S. D., Stewart, M. O., & Strunk, D. (2005). Enduring effects for cognitive behavior therapy in the treatment of depression and anxiety. *Annual Review of Psychology*, 57, 285–315.
- Jarrett, R. B., Kraft, D., Doyle, J., Foster, B. M., Eaves, G. G., & Silver, P. C. (2001). Preventing recurrent depression using cognitive therapy with and without a continuation phase. *Archives of General Psychiatry*, 58, 381–388.
- Kessler, R. C., Scott, K. M., Shahly, V., & Zaslavsky, A. M. (2014). The big picture. In C. S. Richards & M. W. O'Hara (Eds.), *The Oxford handbook of depression and comorbidity* (pp. 599–614). New York, NY: Oxford University Press.
- Kessler, R. C., & Ustun, T. B. (2008). *The WHO World Mental Health Surveys: Global perspectives on the epidemiology of mental disorders*. New York, NY: Cambridge University Press.
- Kuyken, W., Warren, F. C., Taylor, R. S., Whalley, B., Crane, C., Bondolfi, G., ... Dalgleish, T. (2016). Efficacy of mindfulness-based cognitive therapy in prevention of depressive relapse: An individual patient data meta-analysis from randomized trials. *JAMA Psychiatry*, 73, 565–574.
- Nezu, A. M., Nezu, C. M., Greenberg, L. M., & Salber, K. E. (2014). Cancer and depression. In C. S. Richards & M. W. O'Hara (Eds.), *The Oxford handbook of depression and comorbidity* (pp. 302–318). New York, NY: Oxford University Press.
- Nylen, K. J., O'Hara, M. W., Brock, R., Moel, J., Gorman, L., & Stuart, S. (2010). Predictors of the longitudinal course of post-partum depression following interpersonal psychotherapy. *Journal of Consulting and Clinical Psychology*, 78, 757–763.
- O'Hara, M. W., Schiller, C. E., & Stuart, S. (2010). Interpersonal psychotherapy and relapse prevention for depression. In C. S. Richards & M. G. Perri (Eds.), *Relapse prevention for depression* (pp. 77–97). Washington, DC: American Psychological Association.
- O'Hara, M. W., Stuart, S., Gorman, L. L., & Wenzel, A. (2000). Efficacy of interpersonal psychotherapy for postpartum depression. *Archives of General Psychiatry*, 57, 1039–1045.
- Reynolds, C. F., Dew, M. A., Pollock, B. G., Mulsant, B. H., Frank, E., Miller, M. D., ... Kupfer, D. J. (2006). Maintenance treatment of major depression in old age. *New England Journal of Medicine*, 354, 1130–1138.
- Reynolds, C. F., Frank, E., Perel, J. M., Imber, S. D., Cornes, C., Miller, M. D., ... Kupfer, D. J. (1999). Nortriptyline and interpersonal psychotherapy as maintenance therapies for recurrent major depression: A randomized controlled trial in patients older than 59 years. *JAMA*, 281, 39–45.
- Richards, C. S., Cohen, L. M., Morrell, H. E. R., Watson, N. L., & Low, B. E. (2013). Treating depressed and anxious smokers in smoking cessation programs. *Journal of Consulting and Clinical Psychology*, 81, 263–273.
- Richards, C. S., & O'Hara, M. W. (Eds.) (2014a). *The Oxford handbook of depression and comorbidity*. New York, NY: Oxford University Press.
- Richards, C. S., & O'Hara, M. W. (2014b). Introduction. In C. S. Richards & M. W. O'Hara (Eds.), *The Oxford handbook of depression and comorbidity* (pp. 1–10). New York, NY: Oxford University Press.
- Richards, C. S., & Perri, M. G. (1978). Do self-control treatments last? An evaluation of behavioral problem solving and faded counselor contact as treatment maintenance strategies. *Journal of Counseling Psychology*, 25, 376–383.
- Richards, C. S., & Perri, M. G. (2002). *Depression*. Thousand Oaks, CA: Sage Publications.
- Richards, C. S., & Perri, M. G. (Eds.) (2010a). *Relapse prevention for depression*. Washington, DC: American Psychological Association.

- Richards, C. S., & Perri, M. G. (2010b). Epilogue: Top 10 guidelines for practitioners. In C. S. Richards & M. G. Perri (Eds.), *Relapse prevention for depression* (pp. 271–281). Washington, DC: American Psychological Association.
- Serge, L. S., Brock, R. L., & O'Hara, M. W. (2015). Depression treatment for impoverished mothers by point-of-care providers: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 83, 314–324.
- Smith, T. W. (2010). Depression in chronic medical illness: Implications for relapse prevention. In C. S. Richards & M. G. Perri (Eds.), *Relapse prevention for depression* (pp. 199–225). Washington, DC: American Psychological Association.
- Stroud, C. B., Feinstein, B. A., Bhatia, V., Hershenberg, R., & Davila, J. (2014). Intimate relationships. In C. S. Richards & M. W. O'Hara (Eds.), *The Oxford handbook of depression and comorbidity* (pp. 441–459). New York, NY: Oxford University Press.
- Trucco, E. M., Villafuerte, S., Hussong, A., Burmeister, M., & Zucker, R. A. (2018). Biological underpinnings of an internalizing pathway to alcohol, cigarette, and marijuana use. *Journal of Abnormal Psychology*, 127, 79–91.
- Watson, D., & O'Hara, M. W. (2017). *Understanding the emotional disorders: A symptom-level approach based on the IDAS—II*. New York, NY: Oxford University Press.
- Weissman, M. M., Markowitz, J. C., & Klerman, G. L. (2017). *The guide to interpersonal psychotherapy* (Updated ed.). New York, NY: Oxford University Press.

Suggested Reading

- Kuyken, W., Warren, F. C., Taylor, R. S., Whalley, B., Crane, C., Bondolfi, G., ... Dalgleish, T. (2016). Efficacy of mindfulness-based cognitive therapy in prevention of depressive relapse: An individual patient data meta-analysis from randomized trials. *JAMA Psychiatry*, 73, 565–574.
- Reynolds, C. F., Frank, E., Perel, J. M., Imber, S. D., Cornes, C., Miller, M. D., ... Kupfer, D. J. (1999). Nortriptyline and interpersonal psychotherapy as maintenance therapies for recurrent major depression: A randomized controlled trial in patients older than 59 years. *JAMA*, 281, 39–45.
- Richards, C. S., & Perri, M. G. (Eds.) (2010). *Relapse prevention for depression*. Washington, DC: American Psychological Association.

Eating Disorders

Eliot Dennard

Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Eating disorders (EDs) refer to a group of disorders that are characterized by abnormal thoughts, feelings, and behaviors related to food and weight. These disorders often begin in adolescence and are much more common in females than males (American Psychiatric Association [APA], 2013). Individuals with EDs often struggle with other psychological concerns, including depression, anxiety, and substance use. Furthermore, EDs are also associated with medical complications. These factors make the treatment of EDs more complicated. This chapter includes an overview of EDs, including risk factors, comorbidity, complications, and treatment.

Types of Eating Disorders

Anorexia Nervosa

Anorexia nervosa (AN) describes a disorder characterized by self-starvation and body image disturbance. To be diagnosed with AN, an individual must meet the following criteria according to the *Diagnostic and Statistical Manual of Mental Disorders-5* (DSM-5): restriction of caloric intake leading to a low body weight, a fear of gaining weight, and body image disturbance (APA, 2013). AN is a relatively rare psychiatric disorder. Prevalence estimates of AN vary, but one recent nationally representative survey found the lifetime prevalence rates of AN to be 0.9% for women and 0.3% for men (Hudson, Hiripi, Pope, & Kessler, 2007).

Bulimia Nervosa

Bulimia nervosa (BN) is characterized by episodes of binge eating followed by purging. According to the APA (2013), to be diagnosed with BN, an individual must meet the following criteria: frequent episodes of binge eating followed by engaging in “compensatory

behaviors,” and self-evaluation that is heavily dependent on one’s weight and size. To be considered an eating binge, the individual must feel a loss of control over eating, which results in the individual eating an unusually large amount of food in short period of time. Compensatory behaviors are designed to rid the individual of the calories just consumed and can include behaviors such as self-induced vomiting, laxative and diuretic use, and excessive exercise. These symptoms must have occurred at least once a week over the course of 3 months (APA, 2013). The lifetime prevalence estimates for BN are 1.5% among women and 0.5% among men (Hudson et al., 2007).

Binge Eating Disorder

Binge eating disorder (BED) is characterized by eating binges without the use of compensatory behaviors. There also must be distress experienced related to the disordered eating (APA, 2013). The disordered eating must have occurred at least twice a week for 6 months to be classified as BED (APA, 2013). Lifetime prevalence estimates of BED are 3.5% for women and 2.0% for men, which is higher than that of AN and BN (Hudson et al., 2007).

Other Feeding and Eating Disorders

There are several other feeding and EDs described in the DSM-5 (APA, 2013). Pica involves the ingestion of “nonnutritive, nonfood substances” (APA, 2013, p. 329). Rumination disorder involves the regurgitation of food that is either rechewed and swallowed or spit out. Avoidant/restrictive food intake disorder is characterized by an avoidance of food, including sensory stimulation related to food and concern about negative consequences of eating food. This often results in malnutrition, weight loss, and the need for nutritional supplementation. Avoidant/restrictive food intake disorder is distinguished from AN in that with avoidant/restrictive food intake disorder, there is not the same fear of weight gain nor is there body image disturbance (APA, 2013). These other feeding and EDs, particularly Pica, are often comorbid with intellectual disabilities. For the remainder of this chapter, the primary focus will be on AN, BN, and BED.

Subthreshold Levels of Eating Disorders

The current method of defining EDs in the DSM-5 uses a categorical approach, which means that individuals either meet diagnostic criteria or they do not. However, both in research and in clinical practice, individuals presenting with eating concerns often do not meet full diagnostic criteria but still experience significant distress related to food and body image. As such, disordered eating is often conceptualized as a continuum of behaviors and feelings about weight and food, rather than the traditional categorical approach. By taking a continuum approach to understanding disordered eating, EDs can be understood as all of the gray area between normal eating and severe, diagnosable EDs. In the research literature, varying levels of disordered eating presentations are often described as subthreshold EDs and partial EDs. For instance, according to Stice, Marti, Shaw, and Jaconis (2009), a subthreshold ED is defined by meeting all of the criteria for a diagnosable ED, except not at the levels specified by the diagnostic criteria. For example, an individual who engages in bingeing and purging behaviors less than once a week (as opposed to the once a week required by diagnostic standards) would be considered to have subthreshold BN. Similarly, an individual who only meets a

subset of the required symptoms for diagnosis would be considered to have a partial ED (Stice et al., 2009).

A substantial portion of individuals that struggle with disordered eating have subthreshold and partial levels of EDs, as compared with the relatively small amount of individuals with diagnosable EDs. For example, Stice et al. (2009) found that 12% of adolescents fall somewhere on the continuum of disordered eating, which is substantially more than the prevalence of full threshold EDs, which is around 2%. Therefore, it is important for clinicians and researchers to consider the full continuum of disordered eating in both research and practice.

Risk Factors

The research literature has identified a number of factors that contribute to the development of disordered eating symptoms. Understanding different risk factors can hopefully lead to more targeted prevention efforts, as well as better treatment approaches. Although a plethora of research literature has described different risk factors for developing disordered eating, only a small portion of that is presented here.

Biological and Genetic Risk Factors

Like many psychological disorders, EDs also seem to have biological and genetic factors that contribute. In Jacobi, Hayward, de Zwaan, Kraemer, and Stewart Agras' (2004) review article of risk factors of EDs, several twin studies are cited, almost all of which found a higher concordance rate of EDs in monozygotic twins, as compared with dizygotic twins. These finds suggest that there is a genetic component to EDs (i.e., EDs are not solely caused by environmental factors). Recent advances with gene mapping have found several genes that seem linked with EDs (Jacobi et al., 2004). There will undoubtedly be further advances in understanding EDs from a biological perspective, which may lead to advances in pharmacological treatment of EDs.

Psychological, Social, and Cultural Risk Factors

The research literature provides evidence for a number of psychological and social risk factors that contribute to disordered eating. Western culture in particular prizes the female body and strongly values thinness (Striegel-Moore & Bulik, 2007). In addition, having weight concerns has been found to predict subthreshold and full threshold EDs (Killen et al. as cited by Keel & Forney, 2013). Some models even posit that cultural ideals surrounding thinness lead to having weight concerns and body dissatisfaction, which then leads to disordered eating (e.g., see Striegel-Moore & Bulik, 2007). However, individuals from other cultural backgrounds, including Latina women, also develop EDs (Marques et al., 2011). Thus the relationships among cultural variables, body dissatisfaction, weight concerns, and disordered eating are complicated and certainly not fully understood.

Several other risk factors have been supported in the literature. Both negative affect/negative emotionality and perfectionistic tendencies have been found to predict disordered eating (Keel & Forney, 2013). High levels of depressive symptoms have also been found to predict disordered eating (Dennard & Steven Richards, 2013).

Trauma has also been found to be a significant risk factor for developing disordered eating. Brewerton's (2007) review of the literature suggested that childhood sexual abuse, as well as

other forms of abuse and neglect, is significantly associated with EDs, particularly BN. In addition, trauma increases comorbidity in those with EDs (Brewerton, 2007). There are many more hypothesized risk factors for the development of an ED, ranging from dysfunctional family-of-origin experiences to cognitive rigidity (see Jacobi et al., 2004 for a more complete overview). Understanding different risk factors will hopefully lead to more successful prevention and treatment efforts.

Diversity Considerations

Gender and cultural factors are an important consideration to explore in their relation to EDs. Several studies have found that the prevalence of disordered eating is similar in women of different ethnicities (e.g., Marques et al., 2011). However, the expression and cluster of symptoms may be impacted by gender and/or cultural factors. For example, one study utilized Caucasian, Latina, and African American female undergraduate students (Gordon, Castro, Sitnikov, & Holm-Denoma, 2010). Participants were asked their perception of the ideal body type for members of their cultural group. Results indicated that Caucasian women perceived that slimmer body types were ideal for their ethnicity than both Latinas and African Americans. Latinas indicated that their cultural group preferred a slimmer body than the African American participants. Participants also indicated their personal ideal body type (as opposed to ideal body types of the culture at large). There were no differences between Caucasian and Latina women on personal ideal body type. African American women had larger personal ideal body types than did the other two groups (Gordon et al., 2010). This study highlights how cultural considerations may affect body image concerns.

In addition, acculturation levels and acculturative stress may play a role in EDs in individuals from ethnic minority groups. In a sample of Mexican American women with and without EDs, a stronger orientation toward “Anglo American” culture was predictive of disordered eating, whereas a stronger identification with and orientation toward Mexican culture was not associated with disordered eating (Cachelin, Phinney, Schug, & Striegel-Moore, 2006). Acculturative stress, which is defined “as the difficulties and psychological toll that arise from the process of adapting to a new cultural context,” is also an important factor to consider (Warren & Rios, 2012, p. 4). In a sample of Latino men, acculturative stress was correlated with body image disturbance, while acculturation was not (Warren & Rios, 2012). Therefore, it may be important to consider how acculturation and acculturative stress may contribute to the development of an ED.

Gender differences also seem to impact EDs. One indicator of an ED is an individual’s dissatisfaction with his or her weight and body shape. However, this particular symptom may present somewhat differently in men than it does in women. For example, in a sample of undergraduate men and women with and without EDs, Ousley, Cordero, and White (2008) found that both men and women with EDs showed concern about body shape and tone. The women with EDs showed greater concern with weight and fat, while the men showed greater concern with muscle tone.

Gender differences are also evident in individuals hospitalized for AN. Over the period of 16 years, data from both male and female patients admitted to a hospital-based treatment program were analyzed (Gueguen et al., 2012). The males in this study presented for treatment later, had higher mortality rates, were more likely to have been overweight in the past, and were less likely to have a history of suicidality. The females in the study were more likely to be involved in a romantic relationship and living with a partner. Therefore, there may be differences in the interpersonal functioning between men and women with EDs (Gueguen et al., 2012).

Among males with EDs, sexual orientation is correlated with disordered eating. One study found that the rates of EDs are higher among men that identify as gay or bisexual than among men that identify as heterosexual (Feldman & Meyer, 2007). Further research will contribute to the understanding of how diversity-related factors impact both the development and treatment of EDs.

Complications

EDs are often associated with significant health consequences, which often necessitates monitoring by a physician. AN, in particular, often has very severe health consequences associated with malnutrition. Cardiac complications, including sinus bradycardia, decreased heart rate, and mitral valve prolapse, are often serious and are associated with at least one-third of the deaths related to EDs (Katzman, 2005). Issues with bone mass, including osteopenia and osteoporosis, are also associated with AN. The effects of losing bone mass are often permanent (Katzman, 2005). Several endocrine consequences, including amenorrhea, hypoglycemia, and thyroid abnormalities, are also potential health consequences of AN (Mehler & Brown, 2015). AN is also associated with reproductive consequences, including an increased risk of miscarriage, lower weight babies, and infertility (Mehler & Brown, 2015). Finally, in cases of severe AN, changes in brain volume can be seen using MRI technology. These changes are presumed to be a result of malnutrition (Mehler & Brown, 2015).

Cardiovascular issues, such as arrhythmias, hypotension, and bradycardia, are also associated with BN (Casiero & Frishman, 2006). Esophageal complications are also associated with BN and can even become life threatening in the case of esophageal rupture (Mehler, 2011). Many methods of purging, including laxative and diuretic use, often produce electrolyte imbalances, which require medical intervention (Mehler, 2011).

BED is also associated with health complications. One potential implication of binge eating is an increase in body weight, which in itself is associated with negative health outcomes. In one study, obese women that engaged in binge eating had higher rates of medical disorders (e.g., hypertension) than their obese counterparts that did not engage in binge eating (Bulik, Sullivan, & Kendler, 2002). One longitudinal study found that those with BED are at greater risk of developing components of metabolic syndrome (i.e., type 2 diabetes, hypertension, dyslipidemia) when compared with a comparison group individuals of similar body mass index (Hudson et al., 2010). Thus, BED seems to have health complications above and beyond the health risks of being overweight.

Individuals with EDs also have a higher mortality rate as a result of these health consequences and an increased rate of suicide. In fact, AN has the highest mortality rate of any psychiatric disorder. Mortality rates for AN have been found to be around 5% (Steinhausen, 2002). Because of the significant health consequences of EDs, early intervention and medical monitoring are crucial.

Comorbidity

EDs are associated with increased rates of comorbidity with other psychiatric diagnoses, including mood disorders, anxiety disorders, and substance use disorders. Understanding and evaluating individuals for comorbid conditions is especially important because it likely impacts both the severity of the ED and the treatment approach used. For instance, one study of

adolescent females with EDs found that the participants with comorbid depression and anxiety had significantly more hospitalizations, suicide attempts, and higher scores on subscales of self-reported measures (e.g., eating concerns scores, body dissatisfaction scores, perfectionism, etc.) (Brand-Gothelf, Leor, Apter, & Fenning, 2014).

Rates of anxiety disorders in individuals with EDs are higher than in individuals without EDs. In one study, 63.5% of participants with EDs “were diagnosed with at least one lifetime anxiety disorder” (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004, p. 2217). Obsessive-compulsive disorder was the most frequently diagnosed among the sample (40%), and social anxiety disorder was the next most frequently diagnosed anxiety disorder in the sample (20%). The rates of different anxiety disorders did not vary depending on ED diagnosis (i.e., AN and BN) (Kaye et al., 2004).

Many studies have found a strong association between depressive and disordered eating symptoms. The rates of comorbidity between eating and mood disorders, particularly depression, may be as high as 94% (Blinder, Cumella, & Sanathara, 2006). Among high school students, strong positive correlations have also been found between disordered eating and depression (Santos, Steven Richards, & Kathryn Bleckley, 2007). Depressive symptoms have also been found to predict disordered eating in a sample of college females (Dennard & Steven Richards, 2013).

Substance use disorders are often common among individuals with EDs, particularly among those with BN. One study found that individuals with BN were three times more likely to have substance use concerns than individuals with AN (Blinder et al., 2006). This comorbidity is particularly important, as alcohol and substance use disorders are a significant predictor of mortality for individuals with AN (Keel et al., 2003).

Treatment

Conventional wisdom suggests that utilizing a treatment team approach to treat EDs is the most beneficial. EDs are complex disorders that affect an individual psychologically, nutritionally, and medically. As such, EDs are often best treated by a team of professionals collaborating to provide holistic care. Typical members of a treatment team consist of a therapist or psychologist, nutritionist or dietician, and a physician. Therefore, individuals with EDs often receive psychotherapy, nutritional counseling, and medical monitoring. Treatment is generally not only provided on an outpatient basis but can also include partial hospitalization and inpatient/residential treatment.

Psychotherapy

The research supporting psychological therapies for the treatment of EDs, particularly AN, is disappointingly sparse. This is likely due to the low prevalence of the disorder, medical complications requiring hospitalization that interfere with study completion, extended length of time needed for treatment, and individual’s negative attitudes related to recovery (Wilson, Grilo, & Vitousek, 2007). However, recent studies are beginning to show promise in treating both AN and BN with psychotherapy.

Anorexia Nervosa

Few studies have examined the efficacy of psychological therapies for AN. Many of the studies on the treatment of AN have found low recovery rates and often inconclusive results. High

levels of attrition within these studies are another complication of conducting randomized controlled trials with this population (Wilson et al., 2007). Despite the limited research on treating EDs, there are several psychological therapies often used, and some of those therapies have empirical support.

One randomized controlled trial compared interpersonal therapy (IPT), cognitive behavior therapy (CBT), and nonspecific supportive clinical management therapy for women with AN. The nonspecific supportive clinical management provided support, praise, advice, and education related to healthy eating behaviors. This treatment also focused on fostering a strong therapeutic alliance between the patient and clinician. The results of the study showed that the group of patients receiving the nonspecific supportive clinical management did as well as, and even better than, those treated with interpersonal or cognitive behavioral therapy (McIntosh et al., 2005).

Another randomized controlled trial compared a novel therapy for AN, termed the Maudsley model of AN treatment for adults (MANTRA), with “specialist supportive clinical management” (SSCM) for adults with AN (Schmidt et al., 2015). The MANTRA treatment utilized elements of both CBT and IPT to target traits such as obsessiveness, avoidance, and anxiousness that are often central in AN. The treatment also incorporated aspects of motivational interviewing. The other treatment arm of the study utilized SSCM, which included clinical management (e.g., providing advice and psychoeducation) and supportive therapy. Participants completed outcome measures at baseline, 6, and 12 months. Both groups showed substantial improvement in body weight, mood, and other psychological factors at the completion of treatment. There were no differences in body mass index in either treatment group following treatment. Many of the other study outcomes, including assessments related to shape/weight concern, depression, and anxiety, were similar in both treatment groups. However, those in the MANTRA group reported higher treatment acceptability and credibility than those in the SSCM group. Furthermore, MANTRA seemed more effective in increasing body mass index in the most severely underweight participants. The results of this study suggest that a variety of therapeutic approaches may be helpful in treating AN (Schmidt et al., 2015).

Following weight restoration in underweight patients with AN, outpatient CBT has been shown to have some benefits. Pike, Walsh, Vitousek, Wilson, and Bauer (2003) utilized a sample of women who had completed inpatient treatment for AN and achieved a weight gain of at least 90% of an ideal body weight. Participants were randomly assigned to one of two treatment conditions, either CBT or nutritional counseling. Both treatments were administered individually for 50 sessions. The results found that the patients receiving CBT relapsed later in treatment (if at all) than those receiving nutritional counseling. Furthermore, the number of patients prematurely ending treatment before session 10 was higher in the nutritional counseling group. Thus, it appears that CBT is beneficial and is delaying or preventing relapse in individuals who previously completed inpatient treatment for AN (Pike et al., 2003).

Family therapy is often the treatment of choice for adolescents with AN, and it has also received considerable empirical support. One such study compared family-based treatment (FBT) with adolescent-focused therapy for adolescents with AN (Lock et al., 2010). The FBT was conducted in three phases, including a weight restoration phase, followed by a transition phase when decisions related to food and weight were given back to the adolescent, and ending with a phase designed to enhance the relationship between the adolescent and the parent. The adolescent-focused therapy arm of the study focused on helping adolescents understand, identify, and tolerate emotions, as opposed to using starvation as a means of regulating emotion. Rates of symptom remission were similar between the two therapies by the end of

the study. However, at follow-up evaluations (6- and 12-month posttreatment), the FBT group had superior outcomes (Lock et al., 2010).

Despite the limited empirical support for treating AN, the American Psychiatric Association's (APA) Practice Guidelines for treating AN include "psychodynamically informed" therapy during the initial refeeding and weight gain process. Therapy should incorporate "empathic understanding, explanations, praise for positive efforts, coaching, support, encouragement, and other positive behavioral reinforcement" (p. 16). Providing these elements in therapy may increase the patient's comfort in therapy, which could potentially reduce premature termination of therapy. For children and adolescents with AN, the guidelines recommended the Maudsley model of family therapy, which is similar to the FBT previously described (APA, 2006). This approach generally requires around 20 sessions over 12 months. The initial phase of treatment helps restore weight in the patient with AN by having the parents control and regulate their child's caloric intake. In subsequent phases of treatment, the patient is gradually allowed more autonomy in his/her eating choices (Pike, Gianini, Loeb, & Le Grange, 2015). Following weight restoration, the guidelines recommend CBT to prevent relapse and note that IPT and psychodynamically oriented therapy are also often used with success in clinical practice (APA, 2006).

Bulimia Nervosa

For BN, both CBT and IPT have empirical support. Generally, CBT for BN requires 16–20 sessions. Aims of the treatment include CBT approaches to increase motivation, modify eating patterns, reduce shape/weight concerns, and reduce likelihood of relapse (Wilson et al., 2007). CBT for BN eliminates bingeing and purging behaviors in approximately 30–50% of cases (Wilson et al., 2007).

Several interventions are often used in CBT for BN. Initial stages of therapy often focus on increasing patient motivation and engagement in the treatment process. Self-monitoring of eating behaviors and associated behaviors, thoughts, and feelings is another initial component of CBT for BN. Another essential element of CBT for BN is psychoeducation. During this process, the clinician provides the patient with information about the harmful effects of purging, laxative use, etc., as well as how those behaviors are ineffective weight management strategies. Subsequent stages of treatment focus on addressing shape and weight concerns, dietary rules, low self-esteem, and interpersonal concerns (Murphy, Straebl, Cooper, & Fairburn, 2010).

CBT has also been shown to be superior to psychoanalytic therapy in treating BN. For example, a recent randomized controlled trial assigned 70 patients with BN to receive either 2 years of weekly psychoanalytic psychotherapy or 20 sessions of CBT (Poulsen et al., 2014). Although both treatments resulted in improvement, those receiving CBT fared much better than those receiving psychoanalytic psychotherapy. Abstinence from bingeing and purging behaviors was achieved after 5 months for 42% of the patients in the CBT group, compared with 6% of the patients in the psychoanalytic group. At the end of the 2-year study, abstinence of bingeing and purging was maintained by 44% of patients in the CBT group and only 15% in the psychoanalytic group (Poulsen et al., 2014).

The results found in randomized clinical trials using CBT for BN seem to translate to clinical practice as well. For example, Waller et al. (2014) utilized CBT to treat individuals with BN in an outpatient therapy clinic. There were few exclusion criteria, and the implementation of the therapy was less manualized than in typical randomized controlled trials. The results of the study showed a remission rate of close to 50%, which is similar to the results observed in randomized clinical trials (Waller et al., 2014). Thus, CBT seems effective in "real-world" practice as well.

Dialectical behavior therapy (DBT), which is often considered a “third-wave CBT,” also shows some promise in the treatment of BN. For example, in a small study, 30 participants with BN were assigned to either 20 sessions of DBT or a 20-week wait list group. The DBT used was specifically designed to teach emotion-regulation skills. Those in the DBT group showed much greater reductions in bingeing and purging episodes than did their wait list group counterparts (Shafer, Telch, & Agras, 2001).

There is also empirical support for the use of IPT in treating BN. IPT was originally developed for treating depressive disorders and focuses on modifying interpersonal patterns that contribute to disordered eating (Wilson et al., 2007). One study compared a group of participants that completed CBT with a group that had completed IPT. The results showed no significant differences between the groups at the 1-year follow-up. However, measurements taken at the end of treatment showed greater improvement in the group treated with CBT (Agras, Walsh, Fairburn, Terrance Wilson, & Kraemer, 2000). Thus, IPT seems to have equivalent long-term results and may also be an effective choice for treating BN, but CBT may show quicker results.

The APA’s Practice Guidelines (2006) for treating BN suggest using CBT to address acute symptoms. IPT is also recommended as a possible initial therapy, or for patients that do not respond to CBT. In adolescents, family therapy is suggested, and couple’s therapy is suggested for adults experiencing conflict in romantic relationships (APA, 2006).

Medical Management

Medical monitoring by a qualified medical professional with experience treating EDs is another component of treatment. A medical provider’s role includes assessing degree of malnutrition and providing medical supervision and monitoring during the individual’s treatment process (Golden et al., 2015). This includes assessment and management of common physical complications of EDs (e.g., cardiac complications, electrolyte imbalances). More severe EDs often require inpatient medical treatment. Some possible reasons for hospitalization include severe electrolyte disturbances, hypothermia, food refusal, extreme low weight (i.e., weighing less than 75% of an ideal body weight), physiological instability, and suicidal ideation (Golden et al., 2015).

In addition to medical monitoring, pharmacotherapy may also be beneficial for individuals with BN. Flament, Bissada, and Spettigue (2012) reviewed randomized controlled trials utilizing monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs), and selective serotonin reuptake inhibitors (SSRIs). Results showed that all three classes of medication were more effective than a placebo in reducing binge eating (Flament et al., 2012). Topiramate, an antiepileptic drug, has been shown to reduce binge eating in individuals with BED. When compared with group of participants receiving a placebo, the individuals receiving topiramate showed a significant reduction in several outcome measures, including binge frequency and weight (McElroy et al., 2003).

Nutritional Therapy

A registered dietician is another integral part of the treatment team. Registered dietitians can provide nutritional therapy to individuals with EDs and focus on food-related issues faced by the individual with an ED. The goals of nutritional therapy are to provide the individual with psychoeducation related to nutrition, develop a meal plan, and begin to develop healthier eating patterns. Dietitians can also be instrumental in monitoring the possible effects of malnutrition (Henry & Ozier, 2006).

Partial Hospitalization

Partial hospitalization programs (PHP) are also an option for the treatment of EDs. In these programs, patients are usually provided with 8–12 hr/day of treatment that usually includes individual therapy, group therapy, nutritional therapy, and medical monitoring. In addition, patients are supervised during meal times while at the program. Patients return home on the evenings and weekends. Alternatively, some programs offer “supportive housing” during the patient’s time in the PHP and while transitioning out of PHP (Tantillo, MacDowell, Anson, Taillie, & Cole, 2009).

Residential Treatment

Residential treatment programs offer more intensive treatment in a nonhospital setting. Most treatment centers adopt an integrative approach to treatment, incorporating CBT, IPT, and DBT. Individuals receive therapy in a variety of formats while in residential treatment, which includes both individual and group therapy. Many programs also implement 12-step work, art therapy, yoga, and meditation. One potential disadvantage of residential treatment is that it often requires a significant financial commitment and is sometimes not covered by health insurance (Frisch, Herzog, & Franko, 2006).

Prognosis

Although there are numerous treatment options for EDs, the prognosis is often discouraging. One longitudinal outcome study of individuals with AN found that 47.5% of the individuals in the study still met criteria for AN, BN, or ED-NOS at 12-year follow-up (Fitcher, Quadflieg, & Hedlund, 2006). Early intervention is especially important in treating EDs. As research advances, more knowledge will possibly help facilitate better methods of treating EDs.

Conclusion

Although EDs have been described as far back as the dark ages and into the Renaissance, research related to etiology and treatment of EDs is still somewhat lacking. However, research has determined several potential contributors to developing disordered eating, including biological, social, cultural, and psychological correlates. As scientific research advances, there will likely be a deeper understanding of what causes EDs, which will likely lead to more efficacious treatments. Currently, several treatment strategies indicate the possibility of a good prognosis for individuals suffering from EDs. Having professionals from multiple fields working with individuals with EDs seems to be the best approach, as these individuals have psychological, physical, and nutritional needs that must be addressed.

Author Biography

Eliot Dennard, PhD, is a counseling psychologist in private practice. She received her undergraduate degree from the University of Oklahoma and her masters and doctoral degrees from Texas Tech University. Although she primarily works in clinical practice, her previous research focused on predictors of disordered eating and gender and cultural differences in eating disorders.

References

- Agras, W. S., Walsh, T., Fairburn, C. G., Terrance Wilson, G., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry*, 57, 459–466.
- American Psychiatric Association. (2006). *Practice guidelines for the treatment of eating disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.
- Blinder, B. J., Cumella, E. J., & Sanathara, V. A. (2006). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine*, 68, 454–462. doi:10.1097/01.psy.00000221254.77675.f5
- Brand-Gothelf, A., Leor, S., Apter, A., & Fenning, S. (2014). The impact of comorbid depressive and anxiety disorders on severity of anorexia nervosa in adolescent girls. *The Journal of Nervous and Mental Disease*, 202, 759–762. doi:10.1097/NMD.0000000000000194
- Brewerton, T. D. (2007). Eating disorders, trauma, and comorbidity: Focus on PTSD. *Eating Disorders*, 15, 285–304. doi:10.1080/10640260701454311
- Bulik, C. M., Sullivan, P. F., & Kendler, K. S. (2002). Medical and psychiatric morbidity in obese women with and without binge eating. *International Journal of Eating Disorders*, 32, 72–80. doi:10.1002/eat.10072
- Cachelin, F. M., Phinney, J. S., Schug, R. A., & Striegel-Moore, R. H. (2006). Acculturation and eating disorders in a Mexican American community sample. *Psychology of Women Quarterly*, 30, 340–347. doi:10.1111/j.1471.6402.2006.00309.x
- Casiero, D., & Frishman, W. H. (2006). Cardiovascular complications of eating disorders. *Cardiology in Review*, 14, 227–231. doi:10.1097/01.crd.0000216745.96062.7c
- Dennard, E. E., & Richards, C. S. (2013). Depression and coping in subthreshold eating disorders. *Eating Behaviors*, 14, 325–329. doi:10.1016/j.eatbeh.2013.05.011
- Feldman, M. B., & Meyer, I. H. (2007). Eating disorders in diverse lesbian, gay, and bisexual populations. *International Journal of Eating Disorders*, 40, 218–226. doi:10.1002/eat.20360
- Fitcher, M. M., Quadflieg, N., & Hedlund, S. (2006). Twelve-year course and outcome predictors of anorexia nervosa. *International Journal of Eating Disorders*, 39, 87–100. doi:10.1002/eat.20215
- Flament, M. F., Bissada, H., & Spettigue, W. (2012). Evidence-based pharmacotherapy of eating disorders. *International Journal of Neuropsychopharmacology*, 15, 189–207. doi:10.1017/S1461145711000381
- Frisch, M. J., Herzog, D. B., & Franko, D. L. (2006). Residential treatment for eating disorders. *International Journal of Eating Disorders*, 39, 434–442. doi:10.1002/eat.20255
- Golden, N. H., Katzman, D. K., Sawyer, S. M., Ornstein, R. M., Rome, E. S., Garber, A. K., ... Kreipe, R. E. (2015). Update on medical management of eating disorders in adolescents. *Journal of Adolescent Health*, 56, 370–375. doi:10.1016/j.jadohealth.2014.11.020
- Gordon, K. H., Castro, Y., Sitnikov, L., & Holm-Denoma, J. M. (2010). Cultural body shape ideals and eating disorder symptoms among white, Latina, and black college women. *Cultural Diversity and Ethnic Minority Psychology*, 16, 135–143. doi:10.1037/a0018671
- Gueguen, J., Godart, N., Chambry, J., Brun-Eberentz, A., Foulon, C., Divac, S. M., ... Huas, C. (2012). Severe anorexia nervosa in men: Comparison with severe AN in women and analysis of mortality. *International Journal of Eating Disorders*, 45(537), 545. doi:10.1002/eat.20987
- Henry, B. W., & Ozier, A. D. (2006). Position of the American Dietetic Association: Nutrition intervention in the treatment of anorexia nervosa, bulimia nervosa, and other eating disorders. *Journal of the American Dietetic Association*, 106, 2073–2082. doi:10.1016/j.jada.2011.06.016
- Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey replication. *Journal of Biological Psychiatry*, 61, 348–358. doi:10.1016/j.biopsych.2006.03.040
- Hudson, J. I., Lalonde, J. K., Coit, C. E., Tsuang, M. T., McElroy, S. L., Crow, S. J., ... Pope, H. G. (2010). Longitudinal study of the diagnosis of components of the metabolic syndrome in individuals

- with binge eating disorder. *The American Journal of Clinical Nutrition*, 91, 1568–1573. doi:10.3945/ajcn.2010.29203
- Jacobi, C., Hayward, C., de Zwaan, M., Helena, K., & Stewart Agras, W. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin*, 130, 19–65. doi:10.1037/0033-2909.130.1.19
- Katzman, D. K. (2005). Medical complications in adolescents with anorexia nervosa: A review of the literature. *International Journal of Eating Disorders*, 37, S52–S59. doi:10.1002/eat.20118
- Kaye, W. H., Bulik, C. M., Thornton, L., Barbarich, N., & Masters, K. (2004). Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *American Journal of Psychiatry*, 161, 2215–2221. doi:10.1176/appi.ajp.161.12.2215
- Keel, P. K., Dorner, D. J., Eddy, K. T., Franko, D., Charatan, D. L., & Herzog, D. B. (2003). Predictors of mortality in eating disorders. *Archives of General Psychiatry*, 60, 179–183. doi:10.1001/archpsyc.60.2.179
- Keel, P. K., & Jean Forney, K. (2013). Psychosocial risk factors for eating disorders. *International Journal of Eating Disorders*, 46, 433–439. doi:10.1002/eat.22094
- Lock, J., Le Grange, D., Stewart Agras, W., Moye, A., Bryson, S. W., & Jo, B. (2010). Randomized clinical trial comparing family-based treatment with adolescent focused individual therapy for adolescents with anorexia nervosa. *Archives of General Psychiatry*, 67, 1025–1032. doi:10.1001/archgenpsychiatry.2010.128
- Marques, L., Algeria, M., Becker, A. E., Chen, C.-n., Fang, A., Chosak, A., & Diniz, J. B. (2011). Comparative prevalence, correlates of impairment, and service utilization for eating disorders across US ethnic groups: Implications for reducing ethnic disparities in health care access for eating disorders. *International Journal of Eating Disorders*, 44, 412–420. doi:10.1002/eat.20787
- McElroy, S. L., Arnold, L. M., Shapira, N. A., Keck, P. E., Rosenthal, N. R., Rezauil Karim, M., & Hudson, J. I. (2003). Topiramate in the treatment of binge eating disorder associated with obesity: A randomized, placebo-controlled trial. *American Journal of Psychiatry*, 160, 255–261. doi:10.1176/appi.ajp.160.2.255
- McIntosh, V. V. W., Jordan, J., Carter, F. A., Luty, S. E., McKenzie, J. M., Bulik, C. M., ... Joyce, P. R. (2005). Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *American Journal of Psychiatry*, 162, 741–747. doi:10.1176/appi.ajp.162.4.741
- Mehler, P. S. (2011). Medical complications of bulimia nervosa and their treatments. *International Journal of Eating Disorders*, 44, 95–104. doi:10.1002/eat.20825
- Mehler, P. S., & Brown, C. (2015). Anorexia nervosa-medical complications. *Journal of Eating Disorders*, 3, 1–8. doi:10.1186/s40337-015-0040-8
- Murphy, R., Straebl, S., Cooper, Z., & Fairburn, C. G. (2010). Cognitive behavioral therapy for eating disorders. *Psychiatric Clinics of North America*, 33, 611–627. doi:10.1016/j.psc.2010.04.004
- Ousley, L., Cordero, E. D., & White, S. (2008). Eating disorders and body image of undergraduate men. *Journal of American College Health*, 56, 617–621. doi:10.3200/JACH.56.6.617-622
- Pike, K. M., Walsh, B. T., Vitousek, K., Wilson, G. T., & Bauer, J. (2003). Cognitive behavior therapy in posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry*, 160, 2046–2049. doi:10.1176/appi.ajp.160.11.2046
- Pike, K. M., Gianini, L. M., Loeb, K. L., & Le Grange, D. (2015). Treatments for eating disorders. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 641–658). New York, NY: Oxford University Press.
- Poulsen, S., Lunn, S., Daniel, S. I. F., Folke, S., Mathiesen, B. B., Katznelson, H., & Fairburn, C. G. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry*, 171, 109–116. doi:10.1176/appi.ajp.2013.12121511
- Santos, M., Steven Richards, C., & Kathryn Bleckley, M. (2007). Comorbidity between depression and disordered eating in adolescents. *Eating Behaviors*, 8, 440–449. doi:10.1016/j.eatbeh.2007.03.005
- Schmidt, U., Magill, N., Renwick, B., Keyes, A., Kenyon, M., Dejong, H., ... Landau, S. (2015). The Maudsley Outpatient Study of treatments for anorexia nervosa and related conditions (MOSAIC):

- Comparison of the Maudsley Model of Anorexia Nervosa Treatment for Adults (MANTRA) with Specialist Supportive Clinical Management (SSCM) in outpatients with broadly defined anorexia nervosa: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 83, 796–807. doi:10.1037/ccp0000019
- Shafer, D. L., Telch, C. F., & Agras, W. S. (2001). Dialectical behavior therapy for bulimia nervosa. *American Journal of Psychiatry*, 158, 632–634. doi:10.1176/appi.ajp.158.4.632
- Steinhausen, H.-C. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, 159, 1284–1293. doi:10.1176/appi.ajp.159.8.1284
- Stice, E., Nathan Marti, C., Shaw, H., & Jaconis, M. (2009). An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology*, 118, 587–597. doi:10.1037/a0016481
- Striegel-Moore, R. H., & Bulik, C. M. (2007). Risk factors for eating disorders. *American Psychologist*, 62, 181–198.
- Tantillo, M., MacDowell, S., Anson, E., Taillie, E., & Cole, R. (2009). Combining supported housing and partial hospitalization to improve eating disorder symptoms, perceived health status, and health related quality of life for women with eating disorders. *Eating Disorders*, 17, 385–399. doi:10.1080/10640260903210172
- Waller, G., Gray, E., Hinrichsen, H., Mountford, V., Lawson, R., & Patient, E. (2014). Cognitive-behavioral therapy for bulimia nervosa and atypical bulimic nervosa: Effectiveness in clinical settings. *International Journal of Eating Disorders*, 47, 13–17. doi:10.1002/eat.22181
- Warren, C. S., & Rios, R. M. (2012). The relationships among acculturation, acculturative stress, endorsement of the Western media, social comparison, and body image in Hispanic male college students. *Psychology of Men & Masculinity*, 14(192), 201. doi:10.1037/a0028505
- Wilson, G. T., Grilo, C. M., & Vitousek, K. M. (2007). Psychological treatment of eating disorders. *American Psychologist*, 62, 199–216. doi:10.1037/0003066X.62.3.199

Suggested Reading

- American Psychiatric Association. (2006). *Practice guidelines for the treatment of eating disorders* (3rd ed.). Washington, DC: Author.
- Halmi, K. A. (2013). Salient components of a comprehensive service for eating disorders. *World Psychiatry*, 8, 150–155.
- Hay, P. (2013). A systematic review of evidence for psychological treatments of eating disorders: 2005-2012. *International Journal of Eating Disorders*, 46, 462–469.
- Wilson, G. T., Grilo, C. M., & Vitousek, K. M. (2007). Psychological treatment of eating disorders. *American Psychologist*, 62, 199–216.

Headaches

Amy Wachholtz, Adam Harris, and Amy Frers

*Department of Psychology and University Health Sciences Center,
University of Colorado Denver, Denver, CO, USA*

Worldwide, headache disorders are a painful and disabling condition. According to the World Health Organization, headache disorders are the third leading cause of worldwide years lost due to disability (Institute for Health Metrics and Evaluation, 2013). The experience of a headache can fall into one of two categories. The first category is called a “primary” headache diagnosis. This is due to the headache as the sole diagnosis (e.g., migraine, tension, cluster). These forms of headaches are painful, and can be very disabling, but generally are benign. In the other category, the headache is “secondary” because rather than being the primary focus of treatment, the headache is a symptom of another illness, injury, or event. There are hundreds of reasons why someone may experience a secondary headache, from benign allergy-related sinus pressure to the more serious condition of stroke.

Tension Headaches

Epidemiology

Tension-type headache (TTH) is the second most prevalent of all disorders (Vos et al., 2012). The most commonly used diagnostic criteria (see Table 1 for diagnostic criteria) for TTH is the International Classification of Headache Disorders (ICDH), created by the International Headache Society (Headache Classification Committee of the International Headache Society, 2013). The annual prevalence of episodic tension-type headache (ETTH) is estimated at 38%, and the annual prevalence of chronic tension-type headache (CTTH) is 2–3% (Schwartz, Stewart, Simon, & Lipton, 1998). Its peak prevalence is between the ages of 30 and 39, and it is slightly more common among women than men (ratio of 5 : 4) (Chowdhury, 2012).

For the 5.4% of workers in the United States who reported losing productive time due to headache, TTH accounted for about 70% of this reduction. This translates to an average of 2.9 hours per week of reduced performance at work and an annual estimated cost of

Table 1 Tension-type headache diagnostic criteria.

	<i>Infrequent episodic TTH</i>	<i>Frequent episodic TTH</i>	<i>Chronic TTH</i>
Frequency	10+ /year; <1/month	1–14 days/month for more than 3 months	15+ days/month for more than 3 months
Duration	30 min to 7 days	30 min to 7 days	Hours to days, or unremitting
Additional criteria	At least two of the following four characteristics: 1. Bilateral location 2. Pressing or tightening (non-pulsating) quality 3. Mild or moderate intensity 4. Not aggravated by routine physical activity such as walking or climbing stairs Both of the following: 1. No nausea or vomiting 2. No more than one of photophobia or phonophobia	At least two of the following four characteristics: 1. Bilateral location 2. Pressing or tightening (non-pulsating) quality 3. Mild or moderate intensity 4. Not aggravated by routine physical activity such as walking or climbing stairs Both of the following: 1. No nausea or vomiting 2. No more than one of photophobia or phonophobia	At least two of the following four characteristics: 1. Bilateral location 2. Pressing or tightening (non-pulsating) quality 3. Mild or moderate intensity 4. Not aggravated by routine physical activity such as walking or climbing stairs Both of the following: 1. Neither moderate or severe nausea nor vomiting 2. No more than one of photophobia, phonophobia, or mild nausea

\$19 billion, adjusted for inflation (Schwartz, Stewart, & Lipton, 1997). The annual estimated cost of TTH for adults in the European Union is 21 billion Euros (M. Linde et al., 2012).

Etiology

A study of headache sufferers found that the most common precipitating factors they reported were stress/tension, not eating on time, lack of sleep, and fatigue (Spierings, Ranke, & Honkoop, 2001). However, these factors were reported at similar rates by both patients diagnosed with TTH and those diagnosed with migraine, so the authors note that they should be considered general causes of headache rather than causes of TTH specifically. (Permission to reproduce granted by: _____ [copyright holder, on _____ [date].)

Stress was the most frequently reported precipitating factor, and a stressful task can induce headaches in TTH sufferers at a far greater rate than it induces headaches in those without a diagnosis of TTH (Cathcart, Petkov, Winefield, Lushington, & Rolan, 2010). This difference may be due to increased pain sensitivity in people with TTH. In studies of the effects of stress-inducing tasks on headache, CTTH patients had lower pain thresholds than healthy participants at the beginning of the experiment, and they also experienced a greater reduction in pain threshold after the stressful task (Cathcart et al., 2010; Cathcart, Petkov, & Pritchard, 2008).

Tenderness of the pericranial (head and neck) muscles is thought to play a large role in TTH. Patients with TTH have increased pericranial muscle tenderness (Aaseth, Grande, Lundqvist, & Russell, 2014). It is possible that poor posture contributes to pericranial muscle

tenderness (Freitag, 2013). Additionally, there are myofascial (connective) tissue trigger points in the head and neck that may be more electrically active (and therefore more sensitive to pain) in people with TTH (Fernández-De-Las-Peñas, Cuadrado, & Pareja, 2007).

The temporomandibular (jaw) joint has also been implicated in TTH. People with temporomandibular joint dysfunction (TMJD) are more likely to have TTH (Speciali & Dach, 2015). Further, TMJD patients treated with botulinum toxin A (Botox) have displayed a reduction in TTH symptoms (Pihut, Ferendiuk, Szewczyk, Kasprzyk, & Wieckiewicz, 2016).

Treatments

Biofeedback training has been studied extensively for the treatment of TTH. A meta-analysis by Nestoriuc, Rief, and Martin (2008) analyzed 53 studies of patients with any form of TTH that were treated with biofeedback training. The interventions consisted of an average of 11 sessions. The studies used structured headache diaries to assess their primary outcome measure, headache pain, which consists of headache frequency, duration, and intensity. The analysis found a medium-to-large effect size for all studies—that is, comparisons between biofeedback and any control. There was a medium-to-large effect size when biofeedback was compared with no treatment, a medium effect size compared with placebos, and a small effect size compared with relaxation interventions. In addition, 18 studies measured follow-up headache pain. Follow-ups ranged from 3 to 60 months, and the average follow-up was 14.6 months. The authors found a medium-to-large effect size of biofeedback treatment. Some secondary measures were assessed as well: with biofeedback treatment, medication intake was reduced with a small-to-medium effect size, and there were low dropout rates that indicate a high level of acceptability for biofeedback treatment.

Relaxation training involves bringing awareness to tense muscles in the body (Sun-Edelstein & Mauskop, 2012). There are two main types of relaxation training: progressive relaxation training (PRT) and autogenic training. PRT involves intentionally tensing and releasing individual muscle groups one by one. Autogenic training uses imagery and body awareness to relax the muscles. A systematic review by Verhagen, Damen, Berger, Passchier, and Koes (2009) found mixed results for the efficacy of relaxation training for the treatment of TTH. Two of the studies reviewed found significant improvement of TTH compared with placebo and no treatment groups, and four studies found no significant difference.

Evidence is mixed for the efficacy of cognitive behavioral therapy (CBT) for the treatment of TTH, but several studies have demonstrated that CBT is more efficacious than no treatment or placebo and that it can reduce TTH by 30–50% (Christiansen, Jürgens, & Klinger, 2015). It has been suggested that CBT is most effective for patients with psychological or environmental stressors that contribute to their TTH, and some studies have found that it is more effective when combined with biofeedback and relaxation training (Freitag, 2013; Sun-Edelstein & Mauskop, 2012).

Manual therapy (e.g., massage, chiropractic therapy) is commonly used for a variety of types of pain. Lozano López, Mesa Jiménez, de la Hoz Aizpurúa, Pareja Grande, and Fernández de las Peñas (2016) conducted a systematic review of manual therapy for patients with frequent or chronic TTH. There were two primary types of manual therapy used: articulatory techniques that involve moving a joint through its full range of motion and soft tissue therapy where pressure is applied to the body as in massage. Some studies used both. There was a significant decrease in headache frequency in all studies. Headache intensity decreased in 8 of the 10 studies. Analgesic use decreased in only one of five studies. Follow-up periods were relatively short in some of the studies, limiting conclusions about the lasting effects of manual therapy.

A Cochrane review by K. Linde et al. (2016) investigated the efficacy of acupuncture for the treatment of chronic and episodic TTH. Twelve studies (2,349 participants) were included. The interventions included between 6 and 15 sessions, and the sessions occurred at least once per week. Several studies found that acupuncture reduced headache frequency and intensity for 9–29% more people than did sham treatments and control conditions. However, the studies that compared acupuncture with other forms of treatment were judged to be too low of quality to draw strong conclusions. Therefore, the research is inconclusive about the effectiveness of acupuncture compared with other treatments for TTH. A review by Hao, Xue, Dong, and Zheng (2013) similarly found mixed and inconclusive results. However, they found that electroacupuncture, in which an electrical current is passed through the acupuncture needles, may be more efficacious than manual acupuncture.

Acetaminophen (paracetamol) is a commonly used over-the-counter drug that is taken orally for headaches. A Cochrane review by Stephens, Derry, and Moore (2016) examined the results of studies of patients with frequent ETTH, most of whom took the standard adult dose of 1,000 mg. Only 5–15% more patients received a benefit from using acetaminophen compared with those who took a placebo, across 23 studies (8,079 participants). Eleven studies (5,605 participants) found no significant difference in adverse events between participants taking acetaminophen and a placebo. These findings indicate that a small but consistent group of people get a benefit from taking acetaminophen. It is unclear why this group benefits while others do not.

Nonsteroidal anti-inflammatory drugs (NSAIDs) are another type of over-the-counter medication commonly taken for headaches. A Cochrane review by Derry, Wiffen, Moore, and Bendtsen (2015) examined the effects of a 400 mg dose of ibuprofen, a type of NSAID, on patients with frequent ETTH. Only 7–17% more patients received a benefit from using ibuprofen compared with those who took a placebo, across 12 studies (3,094 participants). Eight studies (1,645 participants) found no significant difference in adverse events between participants taking ibuprofen and a placebo. These findings are similar to those for acetaminophen and suggest that NSAIDs not only may be effective for slightly more people than acetaminophen but also not substantially better than behavioral treatments.

Selective serotonin reuptake inhibitors (SSRIs) and serotonin–norepinephrine reuptake inhibitors (SNRIs) are antidepressants commonly used to treat depression and anxiety. They are also sometimes prescribed for the treatment of chronic TTH. A Cochrane review by Banzi et al. (2015) evaluated the results of 8 studies (412 participants) that used an SSRI or SNRI to treat patients with chronic TTH. The SSRIs used were citalopram, sertraline, fluoxetine, paroxetine, and fluvoxamine; the SNRI was venlafaxine. With the exception of reduced analgesic use compared with placebo, there was no greater benefit from SSRIs compared with placebo or other antidepressants. However, all of the included studies were rated as low quality; therefore it is still unclear if these drugs reduce the frequency of TTH.

TTH is a very common disorder for people of all ages across the globe. Presently, biofeedback training, CBT, and manual therapy appear to be the most effective in reducing the frequency, duration, and intensity of TTH episodes, while acetaminophen and NSAIDs are effective for acute treatment of TTH pain for only a small percentage of TTH sufferers.

Migraine Headaches

Migraine headaches affect many people throughout the United States and the world, having a global prevalence rate of 11% (Stovner et al., 2007) and affecting 17.3% of women and 5.7% of men (Buse, Silberstein, Manack, Papapetropoulos, & Lipton, 2013). Migraines are

among the top causes of reduced productivity at work (Stewart, Ricci, Chee, Morganstein, & Lipton, 2003) and contribute 1.4% of all years of life lost to disability (World Health Organization, 2001). Additionally, the amount of disability associated with migraine has been found to be comparable with that of dementia or two days after a heart attack (Steiner, Stovner, & Birbeck, 2013).

Migraine Process

There are three phases of a migraine. The first is the premonitory phase that can be thought of as the “warning” phase of a migraine and can occur several hours before the onset of the headache. About 33% of migraine sufferers experience this, and common symptoms include fatigue, gastrointestinal distress, and/or mood changes (Kelman, 2004). Those who experience the premonitory phase appear to have more headache-related complications, such as nausea and a longer-lasting headache.

The second phase is the aura phase that is experienced by about a third of people with migraines. During this period people experience visual, auditory, or motor symptoms (McCance, Huether, Brashers, & Rote, 2010). The aura may last up to an hour or longer, and roughly 90% of those who experience aura develop the proceeding headache on the same side of the head. Additionally, migraines with aura are often more mild than those without aura (Russell, Iversen, & Olesen, 1994). According to the International Headache Society diagnostic criteria, migraines can occur with or without an aura (Headache Classification Committee of the International Headache Society, 2013).

The last phase in migraine headaches is the headache phase during which actual headache symptoms occur and is characterized by unilateral, throbbing pain that worsens with movement. They are often accompanied by nausea/vomiting, and the sufferer usually has some kind of sensory sensitivity to light, smell, and/or noise. Migraines can be defined as either episodic (less than 15 attacks per month) or chronic (15 attacks or more per month for at least 3 months) (Headache Classification Committee of the International Headache Society, 2013).

Migraine Triggers

There are various triggers common in patients with migraine. Stress, weather changes, certain foods, skipping meals, too much or too little sleep or caffeine, bright sunlight, alcohol, strong odors, and menstruation can all be potential triggers for people (Wöber & Wöber-Bingöl, 2010). Additionally, certain food additives and food dyes have also been noted as potential triggers in some migraineurs (Frieri & Kettelhut, 1999).

Pathophysiology

There are many aspects of the pathophysiology of migraine headaches that are still unknown. The widely accepted theory is that during an attack, cortical spreading depression occurs, where a wave of electrical activity spreads from the occipital lobe to the cerebral cortex, which is then followed by a suppression of brain activity (Charles & Baca, 2013). This is thought to cause the aura phase of migraine; however, the reasons for migraine symptomology are still under debate. This cortical spreading depression has been found to cause increased blood flow in the middle meningeal artery that is largely involved in trigeminal and parasympathetic pathways (Bolay et al., 2002). Furthermore, cortical spreading depression has been shown to activate dural nociceptors (nerve cells that respond to painful stimuli) (Zhang et al., 2010), which is believed to cause the headache.

The etiology of migraines is thought to be largely genetic. More specifically, it seems that migraines with aura are more likely to be genetic, with children of a parent who has migraine with aura being 1.9 times more likely to develop them, compared with children of parents with migraine without aura being 4 times more likely to develop them. Interestingly, the spouse of a person with aura migraines is 1.5 times more likely to also have them, whereas there is no increased risk if the spouse has migraine without aura, indicating the possibility of more environmental factors impacting the likelihood of developing migraine with aura (Russell, Iselius, & Olesen, 1996).

Treatments

Pharmacological

Analgesics have proven to be effective for treating acute migraine attacks in many clinical trials. The typical analgesic treatments for migraine headaches include triptans, NSAIDs, and muscle relaxants (Cameron et al., 2015), and in some cases contraceptives for women who have migraines due to hormonal fluctuations (Nappi et al., 2013). Additionally, many over-the-counter medications have caffeine in them as an active ingredient. SSRIs have also been used for migraine prophylaxis; however, while they may be effective for treating anxiety/depression in people with migraines, a recent review concluded that there is not sufficient evidence that these work better than placebo for migraines (Banzi et al., 2015).

It should be noted that too much medication use can result in medication overuse headaches, a form of chronic migraine. These occur at least 15 times per month, for at least 3 months, resulting from taking triptans or opioids more than 10 days per month (or acetaminophen/NSAIDs more than 15 days per month) (Headache Classification Committee of the International Headache Society, 2013). Often, these headaches turn episodic migraines into chronic migraines, but by weaning off of the overused medication and establishing medication limits, they may be remedied. Therefore, it is recommended that patients with migraines limit medication use to two or fewer days per week, avoid opioids and butalbital, and always use the proper dosage (Tepper, 2012).

In addition to more traditional forms of pharmacological treatments, Botulinum toxin A has also shown efficacy at reducing the number of headaches in those with chronic migraines (Jackson, Kuriyama, & Hayashino, 2012). However, this has not been shown to be an effective method for episodic migraine reduction, and the associated costs are quite large compared with other prophylactic treatments.

Transcranial Magnetic Stimulation

There is some evidence that transcranial magnetic stimulation (TMS) is effective. A review by Lipton and Pearlman (2010) concluded that single-pulse TMS is effective for the treatment of migraines with aura, and a portable device manufactured by Neuralieve has been made available for the most efficient administration of TMS. It should be noted that while two studies have shown this device to be effective in treating migraines with aura, the authors were funded by Neuralieve and thus the results may warrant cautious interpretation. Furthermore, although these findings show positive results for the treatment of migraine, research has been inconclusive as to whether or not this method is effective for the prevention of migraine attacks.

Biofeedback and Psychotherapy Treatments

Biofeedback training is a type of therapy in which patients are taught to become aware of and mentally regulate physiological responses such as heart rate, skin conductance, and body temperature. Biofeedback has been recognized as an effective treatment for migraines. A review of 55 studies showed that it is most effective when home training is incorporated and blood-volume-pulse feedback appears to be more effective than peripheral skin temperature feedback and electromyography feedback. Furthermore, effect sizes are moderately large, and the effects seem to sustain over time (Nestoriuc & Martin, 2007), making this a viable treatment option for those wishing to avoid medications.

Along the same vein, there are other potential options for migraine sufferers wishing to treat their migraines behaviorally. Therefore, it is important that patients also address any mental health concerns with a trained professional. CBT focuses on challenging thoughts and changing behaviors that contribute to a person's distress. One systematic review concluded that many studies have shown that CBT can be an effective way to reduce the physical symptoms associated with migraines, although the authors note that there is not sufficient evidence for CBT to be considered an established treatment due to some methodological issues in existing studies (Harris, Loveman, Clegg, Easton, & Berry, 2015). Despite this, migraines have been correlated with several mental disorders, including depression and anxiety (Buse et al., 2013), and it seems that a large proportion of patients with chronic migraine do not utilize mental health services. Furthermore, these psychological comorbidities may go unrecognized in patients who simply attribute these disorders to their migraines (Wachholtz, Malone, & Bhowmick, 2015). Therefore, CBT may certainly improve quality of life in those suffering from migraines.

Mindfulness-based methods that focus on teaching moment to moment awareness and separation of emotions from thoughts have also found success in the treatment of migraine headaches. A recent review indicated that mindfulness-based interventions are able to reduce pain intensity (Reiner, Tibi, & Lipsitz, 2013). Additionally, it is important to note that mindfulness is a lifelong skill; therefore, pain reduction effects may compound over time (provided a person practices).

Acupuncture

Acupuncture is a treatment in which needles are inserted at various trigger points on the body. Acupuncture has been tested in several studies, and a recent review showed that it is at least as effective as prophylactic drug treatments for the prevention of migraine (Linde et al., 2009). Furthermore, fewer adverse effects have been found with acupuncture compared with drug therapy.

Emerging Treatments

There is emerging evidence that links may exist between the microbiota of a person and migraines. There are several studies that link gastrointestinal disorders with migraines (for a review, see van Hemert et al., 2014), and one recent study showed that introducing probiotics into the diets of 29 migraine sufferers significantly reduced both occurrence and intensity of migraines over a period of 12 weeks (de Roos et al., 2015). Although this was a small nonrandomized pilot study, the results warrant further research into the possibility of treating migraine with probiotics or with fecal transplantation.

Cluster Headaches

There is another class of headaches known as cluster headaches. They affect men more than women and occur in less than 1% of the population (Russell, 2004). They are characterized by severe unilateral pain, restlessness, and a recurrent pattern of attacks that may occur several times per day and last for 6–12 weeks on average, followed by a period of remission. There is also a tendency for abnormal bodily sensations to occur on the same side as the headache, such as a drooping eyelid and sweating (May, 2005). Researchers are still investigating the best treatment options for this type of headache. Neurostimulation has shown promise, but researchers are still working to determine the most effective, least invasive methods (Pedersen, Barloese, & Jensen, 2013).

Conclusion

Headaches are common, painful, often repetitive, disorders that can cause personal and professional hardship. While primary headache disorders are largely benign, headache as a secondary syndrome can be indicative of a serious disorder. Empirical comprehensive reviews identify psychobehavioral treatments as particularly effective relative to pharmacological approaches to reduce the frequency, severity, or duration of headache pain, and additional novel pharmacological and non-pharmacological treatments are in development.

Author Biographies

Dr. Amy Wachholtz, PhD, MDiv, MS is an assistant professor of psychology at the University of Colorado Denver. Dr. Wachholtz holds graduate degrees in theology, bioethics, clinical psychology, and psychopharmacology. Her research and clinical interests focus on (a) bio-psycho-social-spiritual model of chronic pain disorders and (b) the complexities of treating of comorbid pain and opioid addiction in both acute pain and chronic pain situations.

Adam Harris is a doctoral student in the Clinical Health Psychology Program at the University of Colorado Denver. His research interests include the relationship between health behaviors and mindfulness and their impact on mental health.

Amy Frers is a doctoral student at the University of Colorado Denver in the Clinical Health Psychology Program. She is interested in chronic pain and disease management, mindfulness, and the efficacy of third wave therapies for improving quality of life in these patient populations.

References

- Aaseth, K., Grande, R. B., Lundqvist, C., & Russell, M. B. (2014). Pericranial tenderness in chronic tension-type headache: The Akershus population-based study of chronic headache. *The Journal of Headache and Pain*, 15(1), 58.
- Banzi, R., Cusi, C., Randazzo, C., Sterzi, R., Tedesco, D., Moja, L., & Banzi, R. (2015). Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) for the prevention of migraine in adults. *Cochrane Database of Systematic Reviews*, (4).
- Bolay, H., Reuter, U., Dunn, A. K., Huang, Z., Boas, D. A., & Moskowitz, M. A. (2002). Intrinsic brain activity triggers trigeminal meningeal afferents in a migraine model. *Nature Medicine*, 8(2), 136–142.

- Buse, D. C., Silberstein, S. D., Manack, A. N., Papapetropoulos, S., & Lipton, R. B. (2013). Psychiatric comorbidities of episodic and chronic migraine. *Journal of Neurology*, 260(8), 1960–1969.
- Cameron, C., Kelly, S., Hsieh, S. C., Murphy, M., Chen, L., Kotb, A., ... Wells, G. (2015). Triptans in the acute treatment of migraine: A systematic review and network meta-analysis. *Headache*, 55(Suppl 4), 221–235.
- Cathcart, S., Petkov, J., & Pritchard, D. (2008). Effects of induced stress on experimental pain sensitivity in chronic tension-type headache sufferers. *European Journal of Neurology*, 15(6), 552–558.
- Cathcart, S., Petkov, J., Winefield, A. H., Lushington, K., & Rolan, P. (2010). Central mechanisms of stress-induced headache. *Cephalalgia*, 30(3), 285–295.
- Charles, A. C., & Baca, S. M. (2013). Cortical spreading depression and migraine. *Nature Reviews Neurology*, 9(11), 637–644.
- Chowdhury, D. (2012). Tension type headache. *Annals of Indian Academy of Neurology*, 15(Suppl 1), S83–S88.
- Christiansen, S., Jürgens, T. P., & Klinger, R. (2015). Outpatient combined group and individual cognitive-behavioral treatment for patients with migraine and tension-type headache in a routine clinical setting. *Headache*, 55(8), 1072–1091.
- de Roos, N. M., Giezenaar, C. G., Rovers, J. M., Witteman, B. J., Smits, M. G., & van Hemert, S. (2015). The effects of the multispecies probiotic mixture Ecologic(R)Barrier on migraine: Results of an open-label pilot study. *Beneficial Microbes*, 6(5), 641–646.
- Derry, S., Wiffen, P. J., Moore, R. A., & Bendtsen, L. (2015). Ibuprofen for acute treatment of episodic tension-type headache in adults. *Cochrane Database of Systematic Reviews*, (7), CD003983.
- Fernández-De-Las-Peñas, C., Cuadrado, M. L., & Pareja, J. A. (2007). Myofascial trigger points, neck mobility, and forward head posture in episodic tension-type headache. *Headache*, 47(5), 662–672.
- Freitag, F. (2013). Managing and treating tension-type headache. *The Medical Clinics of North America*, 97(2), 281–292.
- Frieri, M., & Kettelhut, B. (1999). *Food hypersensitivity and adverse reactions: A practical guide for diagnosis and management*. New York, NY: Marcel Dekker, Inc.
- Hao, X., Xue, C. C., Dong, L., & Zheng, Z. (2013). Factors associated with conflicting findings on acupuncture for tension-type headache: Qualitative and quantitative analyses. *The Journal of Alternative and Complementary Medicine*, 19(4), 285–297.
- Harris, P., Loveman, E., Clegg, A., Easton, S., & Berry, N. (2015). Systematic review of cognitive behavioral therapy for the management of headaches and migraines in adults. *British Journal of Pain*, 9(4), 213–224.
- Headache Classification Committee of the International Headache Society. (2013). The international classification of headache disorders, 3rd edition (beta version). *Cephalalgia*, 33(9), 629–808.
- Institute for Health Metrics and Evaluation. (2013). *The global burden of disease: Generating evidence, guiding policy* (Vol. 2013). Seattle, WA: IHME.
- Jackson, J. L., Kuriyama, A., & Hayashino, Y. (2012). Botulinum toxin A for prophylactic treatment of migraine and tension headaches in adults: A meta-analysis. *JAMA*, 307(16).
- Kelman, L. (2004). The premonitory symptoms (prodrome): A tertiary care study of 893 migraineurs. *Headache: The Journal of Head and Face Pain*, 44(9), 865–872.
- Linde, K., Allais, G., Brinkhaus, B., Fei, Y., Mehring, M., Bc, S., ... Ar, W. (2016). Acupuncture for the prevention of tension-type headache. *Cochrane Database of Systematic Reviews*, (4).
- Linde, K., Allais, G., Brinkhaus, B., Manheimer, E., Vickers, A., & White, A. R. (2009). Acupuncture for migraine prophylaxis. *Cochrane Database of Systematic Reviews*, (1), CD001218.
- Linde, M., Gustavsson, A., Stovner, L. J., Steiner, T. J., Barré, J., Katsarava, Z., ... Andrée, C. (2012). The cost of headache disorders in Europe: The Eurolight project. *European Journal of Neurology*, 19(5), 703–711.
- Lipton, R. B., & Pearlman, S. H. (2010). Transcranial magnetic stimulation in the treatment of migraine. *Neurotherapeutics*, 7(2), 204–212.
- Lozano López, C., Mesa Jiménez, J., de la Hoz Aizpurúa, J. L., Pareja Grande, J., & Fernández de las Peñas, C. (2016). Efficacy of manual therapy in the treatment of tension-type headache. A systematic review from 2000 to 2013. *Neurología (English Edition)*, 31(6), 357–369.

- May, A. (2005). Cluster headache: Pathogenesis, diagnosis, and management. *The Lancet*, 366(9488), 843–855.
- McCance, K. L., Huether, S. E., Brashers, V. L., & Rote, N. S. (2010). *Pathophysiology: The biologic basis for disease in adults and children*. London, UK: Elsevier Health Sciences.
- Nappi, R. E., Terreno, E., Sances, G., Martini, E., Tonani, S., Santamaria, V., & Spinillo, A. (2013). Effect of a contraceptive pill containing estradiol valerate and dienogest (E2V/DNG) in women with menstrually-related migraine (MRM). *Contraception*, 88(3), 369–375.
- Nestoriuc, Y., & Martin, A. (2007). Efficacy of biofeedback for migraine: A meta-analysis. *Pain*, 128(1–2), 111–127.
- Nestoriuc, Y., Rief, W., & Martin, A. (2008). Meta-analysis of biofeedback for tension-type headache: Efficacy, specificity, and treatment moderators. *Consulting and Clinical Psychology*, 76(3), 379–396.
- Pedersen, J. L., Barloese, M., & Jensen, R. H. (2013). Neurostimulation in cluster headache: A review of current progress. *Cephalalgia*, 33(14), 1179–1193.
- Pihut, M., Ferendiuk, E., Szewczyk, M., Kasprzyk, K., & Wieckiewicz, M. (2016). The efficiency of botulinum toxin type A for the treatment of masseter muscle pain in patients with temporomandibular joint dysfunction and tension-type headache. *The Journal of Headache and Pain*, 17(1), 29–29.
- Reiner, K., Tibi, L., & Lipsitz, J. D. (2013). Do mindfulness-based interventions reduce pain intensity? A critical review of the literature. *Pain Medicine*, 14(2), 230–242.
- Russell, M. B. (2004). Epidemiology and genetics of cluster headache. *The Lancet Neurology*, 3(5), 279–283.
- Russell, M. B., Iselius, L., & Olesen, J. (1996). Migraine without aura and migraine with aura are inherited disorders. *Cephalalgia*, 16(5), 305–309.
- Russell, M. B., Iversen, H. K., & Olesen, J. (1994). Improved description of the migraine aura by a diagnostic aura diary. *Cephalalgia*, 14(2), 107–117.
- Schwartz, B. S., Stewart, W. F., & Lipton, R. B. (1997). Lost workdays and decreased work effectiveness associated with headache in the workplace. *Journal of Occupational & Environmental Medicine*, 39(4), 320–327.
- Schwartz, B. S., Stewart, W. F., Simon, D., & Lipton, R. B. (1998). Epidemiology of tension-type headache. *Journal of the American Medical Association*, 279(5), 381–383.
- Speciali, J. G., & Dach, F. (2015). Temporomandibular dysfunction and headache disorder. *Headache*, 55(S1), 72–83.
- Spierings, E. L. H., Ranke, A. H., & Honkoop, P. C. (2001). Precipitating and aggravating factors of migraine versus tension-type headache. *Headache*, 41(6), 554–558.
- Steiner, T. J., Stovner, L. J., & Birbeck, G. L. (2013). Migraine: The seventh disabler. *Headache*, 53(2), 227–229.
- Stephens, G., Derry, S., & Moore, R. A. (2016). Paracetamol (acetaminophen) for acute treatment of episodic tension-type headache in adults. *Cochrane Database of Systematic Reviews*, 2016(6).
- Stewart, W. F., Ricci, J. A., Chee, E., Morganstein, D., & Lipton, R. (2003). Lost productive time and cost due to common pain conditions in the US workforce. *JAMA*, 290(18), 2443–2454.
- Stovner, L., Hagen, K., Jensen, R., Katsarava, Z., Lipton, R., Scher, A., ... Zwart, J. A. (2007). The global burden of headache: A documentation of headache prevalence and disability worldwide. *Cephalalgia*, 27(3), 193–210.
- Sun-Edelstein, C., & Mauskop, A. (2012). Complementary and alternative approaches to the treatment of tension-type headache. *Current Pain and Headache Reports*, 16(6), 539–544.
- Tepper, S. J. (2012). Medication-overuse headache. *Continuum: Lifelong Learning in Neurology*, 18(4), 807–822.
- van Hemert, S., Breedveld, A. C., Rovers, J. M., Vermeiden, J. P., Witteman, B. J., Smits, M. G., & de Roos, N. M. (2014). Migraine associated with gastrointestinal disorders: Review of the literature and clinical implications. *Frontiers in Neurology*, 5, 241.

- Verhagen, A. P., Damen, L., Berger, M. Y., Passchier, J., & Koes, B. W. (2009). Behavioral treatments of chronic tension-type headache in adults: Are they beneficial? *CNS Neuroscience and Therapeutics*, 15(2), 183–205.
- Vos, T., Flaxman, A. D., Naghavi, M., Lozano, R., Michaud, C., Ezzati, M., ... Moradi-Lakeh, M. (2012). Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: A systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*, 380(9859), 2163–2196.
- Wachholtz, A., Malone, C., & Bhowmick, A. (2015). The chronic migraineur and health services: National survey results. *Journal of Pain Management & Medicine*, 1(1), 103.
- Wöber, C., & Wöber-Bingöl, Ç. (2010). Triggers of migraine and tension-type headache. *Handbook of Clinical Neurology*, 97, 161–172.
- World Health Organization. (2001). *Mental health: New understanding, new hope*. Geneva, Switzerland: Author.
- Zhang, X., Levy, D., Nosedá, R., Kainz, V., Jakubowski, M., & Burstein, R. (2010). Activation of meningeal nociceptors by cortical spreading depression: Implications for migraine with aura. *Journal of Neuroscience*, 30(26), 8807–8814.

Suggested Reading

- Freitag, F. (2013). Managing and treating tension-type headache. *The Medical Clinics of North America*, 97(2), 281–292.
- Gooriah, R., Nimeri, R., & Ahmed, F. (2015). Evidence-based treatments for adults with migraine. *Pain Research and Treatment*, 2015, 629382.
- Nestoriuc, Y., Rief, W., & Martin, A. (2008). Meta-analysis of biofeedback for tension-type headache: Efficacy, specificity, and treatment moderators. *Consulting and Clinical Psychology*, 76(3), 379–396.
- Smitherman, T. A., Wells, R. E., & Ford, S. G. (2015). Emerging behavioral treatments for migraine. *Current Pain and Headache Reports*, 19(4), 1–6.

Multiple Sclerosis, Walking, and Depression: Implications for Health Psychology

Ipek Ensari¹ and Robert W. Motl²

¹*Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA*

²*Department of Physical Therapy, University of Alabama at Birmingham, Birmingham, AL, USA*

Multiple sclerosis (MS) is an immune-mediated demyelinating disease of the central nervous system (CNS) with often co-occurring, interrelated manifestations. For example, there is evidence for the coupling of cognitive and motor functions in persons with MS such that those with worse information processing speed demonstrate worse upper extremity functioning (Benedict et al., 2011). This coupling is likely the result of damage within co-occurring regions of the CNS that subserve cognitive and motor functions, and it has clear implications for considering multicomponent interventions (Motl, Sandroff, & DeLuca, 2015). Another set of co-occurring, interrelated manifestations of MS includes walking impairment and depressive symptomology, and this has implications for intervention design and clinical practice in the field of health psychology.

Walking impairment is a common externally observable feature of MS and its progression and has meaningful consequences for daily living and mental health. The prevalence and degree of walking impairment in MS can be seen, for example, with the Expanded Disability Status Scale (EDSS) or scores on the Timed 25-Foot Walk (T25FW) and Multiple Sclerosis Walking Scale-12 (MSWS-12) (Motl & Learmonth, 2014). For example, there is consistent evidence that T25FW and 6-minute walk (6MW) performance are compromised in MS compared with controls (Motl & Learmonth, 2014). There further are epidemiological data indicating that 75% of persons with MS report significant walking impairment (Hobart, Riazi, Lamping, Fitzpatrick, & Thompson, 2003). Another study of MS patients in Europe indicated that nearly 50% reported experiencing mobility impairments within the 1st month of diagnosis

and more than 90% of patients reported experiencing mobility impairments within 10 years of diagnosis (van Asch, 2011). The impairment of walking can have many unwanted consequences among persons with MS, including interruptions of daily tasks (91%), worsening of self-esteem (84%), and interference with employment (83%) and driving (87%) (LaRocca, 2011). Walking impairment may further influence family and social life among people with MS (van Asch, 2011). Collectively, the prevalence and impact of walking in MS would seemingly have relevance for mental health outcomes including depression.

Depression (Feinstein, Magalhaes, Richard, Audet, & Moore, 2014) and depressive symptoms (Jones et al., 2012) are common psychological manifestations of MS. One British survey reported that approximately 47% of the 4,178 respondents from the UK MS Registry had elevated depressive symptoms based on a Hospital Anxiety and Depression Scale (HADS) score of 8 or more (Jones et al., 2012). That same study reported that respondents with MS had mean depression symptom scores approximately 1 standard deviation (SD) higher than a reference sample without MS (Jones et al., 2012). Depressive symptoms in MS are associated with a variety of other negative consequences including pain, stress, cognitive dysfunction, and poor disease coping (Feinstein et al., 2014). Depression has been indicated to be predictive of symptoms and prospects for recovery in a variety of other diseases. Depression over illness and treatment has been linked with suicide among the chronically ill, as well as among those with MS (Siegert & Abernethy, 2005). Collectively, this would set the stage for depression resulting in physical health manifestations, including walking difficulty.

There is increasing interest in the evolving bidirectional association between walking and depression in MS, and this relationship is depicted in Figure 1. To begin, poor physical functioning and health, particularly walking impairment, brought about by MS might result in depressive symptoms and episodes (see Path a in Figure 1). Walking has been identified as one

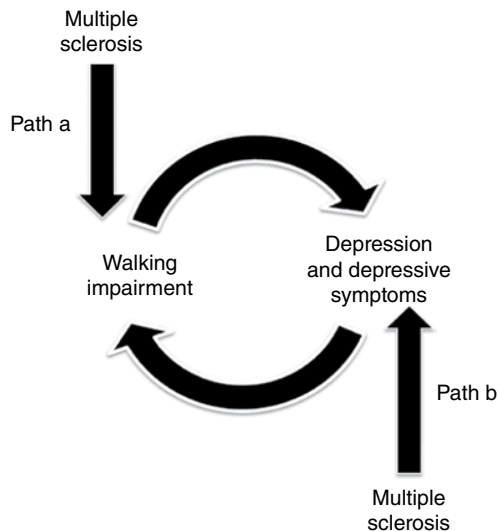


Figure 1 Proposed cyclical associations between walking impairment and depressive symptomology in multiple sclerosis (MS). MS is associated with walking impairment and depressive symptomology. Depressive symptoms might be the starting point for the worsening of disease course (e.g., walking impairment) (Path b). This might subsequently be further exacerbating depressive symptomology at a later time point (Path a).

of the most valued functions among persons with MS (LaRocca, 2011; van Asch, 2011), and its dysfunction could portend considerable consequences for mental health outcomes such as depressive symptoms. Walking impairment has been associated with loss of independence (Hirvensalo, Rantanen, & Heikkinen, 2000), and loss of independence could influence depression and depressive symptoms. Walking impairment has been associated with reduced quality of life in MS (LaRocca, 2011), and this might further reflect worse depressive symptoms. Those with MS who have walking dysfunction have reduced physical activity (Klaren, Motl, Dlugonski, Brian, & Lara, 2013), and this has been associated with depressive symptoms through a pathway mediated by mobility disability (Suh, Motl, & Mohr, 2010). Persons with MS often experience chronic pain that may interfere with walking ability and therefore increase depressive mood (Molton, Hirsh, Smith, & Jensen, 2013). Walking impairment can increase distress, and distress can result in increased depressive mood among individuals with MS (Senders, Sando, Wahbeh, Peterson Hiller, & Shinto, 2014).

On the other hand, there are multiple reasons why depressive symptoms brought about by MS might result in walking impairment (see Path b in Figure 1). Depressive symptoms have been associated with cardiovascular disease (CVD) (Joynt, Whellan, & O'Connor, 2003), and CVD has been identified as a comorbid condition associated with mobility disability in MS (Marrie et al., 2010). Depressive symptoms have been directly associated with walking impairment in large epidemiological studies of the general population of older adults, independent of sex, age, and education levels (Kivela & Pakkala, 2001). Depressive symptoms have been associated with poor patient activation (Goodworth et al., 2014), self-care (Fruewald, Loeffler-Stastka, Eher, & Baumhacki, 2001), and medication adherence in MS (Bruce, Hancock, Arnett, & Lynch, 2010), and this might influence outcomes such walking function. Depressive symptoms are further associated with reduced physical activity (Suh et al., 2010) and poorer health behaviors that are associated with walking impairment (Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999).

The possibility of an association between depressive symptoms and walking impairment in MS is supported empirically by previous literature conducted in other populations. For example, one prospective study reported that poor physical function at baseline predicted depression 3 years later in a sample of older adults, yet the opposite association was not statistically significant (Gayman, Turner, & Cui, 2008). Another study further reported that poor physical function at baseline predicted depressive symptoms after 1 year, regardless of improvements in physical function, in a cohort of 4,757 general practice attendees across Europe (Stegenga et al., 2012). An epidemiological study examined the association between depressive symptoms and physical function in older biracial individuals and reported that the relationship was unidirectional, whereby declining physical function predicted poorer depressive symptom scores but not the other way around (Everson-Rose et al., 2005). Collectively, those findings seemingly would support a directional association between walking impairment and depressive symptoms over time in MS, but we believe that there is a possibility of a dynamic, unfolding bidirectional association between these outcomes. That is, depressive symptoms might be associated with long-term changes in walking impairment, but this might involve intermediate steps involving a cyclical worsening of walking impairment and depressive symptoms.

Of note, the prospect of a bidirectional, cyclical association between walking and depressive symptoms is rooted in the Theory of Unpleasant Symptoms (TOUS) (Lenz & Pugh, 2003). The TOUS indicates that baseline symptoms such as depression predict performance outcomes such as walking impairments. The performance outcomes, in turn, have a reciprocal association with symptoms whereby change in walking impairment would predict change in depressive symptoms. The TOUS further identifies other variables that can predict symptoms

and performance outcomes, indicating the importance of controlling for external variables (e.g., age, sex, weight, physical activity, and disability status) that might confound the relationship between walking impairment and depressive symptoms. Importantly, researchers have experienced success in applying the TOUS for studying symptomatic and behavioral consequences of MS (Motl, Snook, & Shapiro, 2008), and this would seemingly strengthen the applicability of this model for proposing an association between depressive symptoms and walking in MS.

To date, we are aware of a limited number of studies examining the association between depressive symptoms and walking impairment in MS (e.g. Motl, Balantrapu, et al., 2013; Motl, McAuley, et al., 2013; Ensari, Adamson, & Motl, 2016). One study compared performance on the 6MW and symptoms of fatigue, pain, and depression between persons with MS and controls as a precursory step before examining those symptoms as correlates of 6MW performance (Motl, Balantrapu, et al., 2013; Motl, McAuley, et al., 2013). All participants in the study completed questionnaires on symptoms of fatigue, pain, and depression and then underwent the 6MW test. The results indicated that depressive symptoms and pain were significantly, but weakly, associated with 6MW performance in the overall sample that included MS and controls. Of note, depressive symptoms did not explain the variance in 6MW performance as well as disease status (i.e., MS vs. control) or fatigue. On the other hand, the results indicated that fatigue was the strongest correlate of 6MW performance overall and in the separate subsamples of individuals with MS and healthy control participants. Fatigue partially accounted for the difference in 6MW performance between those with MS and controls. The authors concluded that targeting fatigue might be a feasible way to improve walking in individuals with MS and depression is a main contributor into elevated fatigue in MS. One limitation of this study was the sample size ($N = 66$) that might have been too small to detect effects of depressive symptoms of walking outcomes.

The other study involved a longitudinal panel design and examined the associations between depressive symptoms and walking impairment over a period of 2 years among persons with MS (Ensari et al., 2016). The researchers specifically examined the possible reciprocal relationship between depressive symptoms and walking impairment over 2 sequential 12-month periods in a sample of 269 people with relapsing-remitting MS (RRMS). Participants self-reported impairments in walking using the MSWS-12 and depressive symptoms using the HADS-D. Self-reported measures, including demographic and clinical information, were collected via questionnaire packets. The data were examined with panel analysis in *Mplus*, and the model underlying the hypothesized associations is provided in Figure 2. The model hypothesized that depressive symptoms would be the starting point of this reciprocal unfolding relationship between walking impairment and depressive symptoms over time. As hypothesized, the results indicated that depressive symptoms at baseline (Time 1) predicted change in walking impairment at 1-year follow-up (Time 2), and change in walking impairment at 1-year follow-up predicted change in depressive symptoms at 2-year follow-up (Time 3) (Ensari et al., 2016). There was no evidence for the opposite pattern of unfolding relationships (i.e., walking impairment at baseline did not predict depressive symptoms at 1-year follow-up, and depressive symptoms at 1-year follow-up did not predict walking impairment at 2-year follow-up). The relationships were independent of age, sex, disease duration, disability status, BMI, and physical activity. This study provides the first evidence of a possible reciprocal relationship initiating between depressive symptoms and walking impairment over a 2-year period in a large sample of people with RRMS. The associations were independent of extraneous variables, and this partially validates the hypotheses with the TOUS model. Collectively, these findings point to a possible reciprocal association between depressive symptoms and walking impairment over

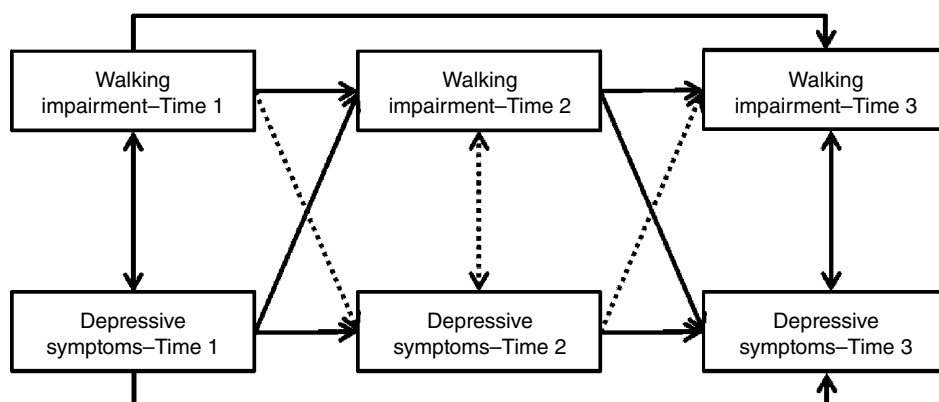


Figure 2 The tested model of longitudinal associations between depressive symptoms and walking impairment over three 1-year periods in 269 persons with multiple sclerosis. *Note:* Solid lines represent statistically significant path coefficients, whereas dashed lines represent nonsignificant path coefficients. *Source:* The figure has been reproduced from Ensari et al. (2016).

time in people with RRMS, and depressive symptoms might be the entry point for breaking this evolving pattern of associations.

The notion of physical and psychological symptoms of a chronic disease interacting with each other over time fits well within the biopsychosocial (BPS) model of health and illness (Taylor, 2012). This model was developed based on limitations of the biomedical approach that was considered a reductionist, linear cause–effect approach for explaining clinical phenomenon. The BPS model attempted to revise and improve our understanding of disease by adding the essential component of patient-related factors into the model. In essence, the BPS model maintains that biological, psychological, and social factors are all determinants and consequences of health and wellness. That is, the individual unique factors related to the patient might be exerting an influence on the symptoms and outcomes of a disease. There might be additional bidirectional influences between the biological, psychological, and social aspects of the disease as experienced by the patient. This is a more comprehensive model to explain the reciprocal relationships between psychological and somatic symptoms (i.e., depressive symptoms and walking impairment in MS). Accordingly, the possibility that changes in depressive symptoms and walking impairment interact with each other over time throughout the disease course makes sense from a theoretical perspective. Practically, it is therefore important that clinicians consider multiple interrelated manifestations when treating a patient, rather than isolating the somatic or psychological symptoms.

In a practical sense, then, it might be prudent that health psychologists working with patients who have MS consider the interactive nature between these seemingly distal (i.e., physical vs. psychological) symptoms in clinical practice. Indeed, some have recommended that screening for depressive symptoms become a routine feature of the medical management of all patients with MS (Siegert & Abernethy, 2005). Such an approach has potential benefits and is consistent with a BPS model. For example, monitoring and addressing depressive symptoms could improve treatment adherence, self-efficacy, and self-care behaviors, as well as help maintain physical activity levels in individuals with MS over time (Bruce et al., 2010; Fruewald et al., 2001; Goodworth et al., 2014; Penninx et al., 1999; Suh et al., 2010). Such improvements could aid in the prevention of future worsening of walking impairment in this population, and this might in turn reduce the likelihood of depressive symptomology worsening with disease progression.

The observation that changes in walking impairment can predict depressive symptoms over time independent of various other correlates is consistent with Lewinsohn's integrative model of depression. This model states that disease (i.e., MS) will be a risk factor for depression only when it results in functional impairment (i.e., walking impairment) and that impairment in the absence of disease is a risk factor for depression. Lewinsohn based this framework on the argument that disability and dependence, along with disease, can determine depressive symptomology and, therefore, that functional impairment must be distinguished from physical disease (Zeiss, Lewinsohn, Rohde, & Seeley, 1996). Evidently, this framework underscores the need to take into account the causal role of functional impairment when examining the causal relationship between depressive symptoms and physical disease (Zeiss et al., 1996). This model states that disease by itself only weakly increases the likelihood of depressive onset and functional impairment (e.g., walking impairment) is a stronger predictor of depressive onset. This model further suggests the presence of a linear positive association between the degree of functional impairment and the risk of depressive symptomology. Such an assumption would mean that physical disease increases the likelihood of depressive symptom onset only if it is associated with functional impairment. Indeed, previous studies have provided evidence for this in both samples of individuals with MS (Voss et al., 2002) and other samples such as older adults (Zeiss et al., 1996). Research involving older adults indicated that disease by itself was not a consistent predictor of depression. Once depressive symptoms are triggered, these mental health changes may have additional negative consequences, including further decline in functional ability (Zeiss et al., 1996). Collectively, the implications of the Lewinsohn's model combined with empirical research are in line with those of Ensari et al. (2016). This underscores the importance of screening for depressive symptoms in clinical practice when working with individuals with chronic diseases (e.g., MS), instead of only relying on monitoring the immediate consequences/impairments associated with the disease (e.g., walking impairments), as the former can initiate the worsening of the latter.

The present findings can inform the design of behavioral approaches that target depressive symptoms and walking impairment in individuals with MS and other neurological conditions. For example, based on the notion that depression might be the starting point for an unfolding cyclical relationship that involves walking impairment, researchers can test the efficacy of an intervention that primarily targets maintaining positive mood or improving depressive symptoms over a period of several months. Primarily, the changes in walking, and secondarily other walking-related functional outcomes (e.g., speed, velocity, gait), would then be the outcome measures of such an intervention. If these target outcomes improve or maintain baseline levels, then this would lend further support to the implementation of targeted interventions for treating depression and secondary physical outcomes among those with MS and other similar chronic diseases. This can subsequently provide individuals with MS more options for disease management and alleviate consequences of negative symptoms in a proactive way. Another possibility would be targeting walking in non-depressed individuals (i.e., those without clinically significant levels of depressive symptoms) to prevent/reduce the potential for depressive symptomology. This approach would be consistent with other researchers who have suggested exercise and physical activity as behavioral interventions that target walking impairment for reducing depressive symptoms in individuals with clinical and subclinical levels of depressive symptoms in the general population (e.g., Kandola & Hayes, 2020).

There are still questions that can be investigated regarding the associations between walking and depressive symptoms in individuals with MS. For example, it would be interesting to assess whether these same patterns manifest in MS patients who have concurrent formal depressive disorders. Accordingly, future studies would benefit from investigating these relationships in

individuals with a clinical diagnosis of a depressive disorder (e.g., major depression). Such investigation can lead the way to better monitoring and interventions of depressive symptoms in individuals with MS and comorbid depressive disorders to hopefully prevent the progression of gait and walking impairment over time that is seen in MS. Another obvious direction would be to assess these patterns of changes across levels of neurological disability and disease course. The current literature is limited mostly to the lowest range of neurological disability, and therefore it would be important to see if higher neurological disability status interacts differently with depressive symptoms and walking impairment over time. Most of the literature so far has focused on the RRMS, and it would be beneficial to study these patterns in other types of MS (e.g., primary progressive MS) wherein ambulatory function declines considerably over time. Finally, future studies would benefit from administering objective measures of walking and clinical diagnoses of depression and examine these two variables over longer periods of time in people with MS for a further assessment of this reciprocal relationship and its stability.

We propose the novel idea and provide supportive arguments as well as models of a cyclical, bidirectional association between psychological and physical functioning symptoms over time in MS. We further provide longitudinal data supporting that these relationships likely evolve over time in MS. The coupling of these two manifestations of MS presents considerable promise for clinical research and practice that embraces the notion of helping those with MS lead the best possible life. The evidence is clear that MS is driving the coupling of depressive symptoms and walking impairment and research that better articulates the nature of this coupling will inform decisions on managing depression and walking by clinicians and practitioners.

Author Biographies

Ipek Ensari is a postdoctoral research scientist at the Center for Behavioral Cardiovascular Health at Columbia University Medical Center. Her research focuses on the biological and behavioral mechanisms underlying the associations between physical activity, cardiovascular health, and negative emotions.

Robert W. Motl is a professor within the Department of Physical Therapy at the University of Alabama at Birmingham. Professor Motl's research focuses on physical activity and its measurement, predictors, and consequences in persons with multiple sclerosis, as well as design and delivery of behavioral interventions for increasing physical activity in MS.

References

- Benedict, R. H., Holtzer, R., Motl, R. W., Foley, F. W., Kaur, S., Hojnacki, D., & Weinstock-Guttman, B. (2011). Upper and lower extremity motor function and cognitive impairment in multiple sclerosis. *Journal of the International Neuropsychological Society*, 17(04), 643–653.
- Bruce, J. M., Hancock, L. M., Arnett, P., & Lynch, S. (2010). Treatment adherence in multiple sclerosis: Association with emotional status, personality, and cognition. *Journal of Behavioral Medicine*, 33(3), 219–227.
- Ensari, I., Adamson, B. C., & Motl, R. W. (2016). Longitudinal association between depressive symptoms and walking impairment in people with relapsing-remitting multiple sclerosis. *Journal of Health Psychology*, 21(11), 2732–2741. doi:10.1177/1359105315584837
- Everson-Rose, S. A., Skarupski, K. A., Bienias, J. L., Wilson, R. S., Evans, D. A., & de CFM, L. (2005). Do depressive symptoms predict declines in physical performance in an elderly, biracial population? *Psychosomatic Medicine*, 67(4), 609–615.

- Feinstein, A., Magalhaes, S., Richard, J. F., Audet, B., & Moore, C. (2014). The link between multiple sclerosis and depression. *Nature Reviews Neurology*, 10(9), 507–517.
- Fruewald, S., Loeffler-Stastka, H., Eher, R., & Baumhacki, U. (2001). Depression and quality of life in multiple sclerosis. *Acta Neurologica Scandinavica*, 104(5), 257–261.
- Gayman, M. D., Turner, R. J., & Cui, M. (2008). Physical limitations and depressive symptoms: Exploring the nature of the association. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 63(4), S219–S228.
- Goodworth, M. C. R., Stepleman, L., Hibbard, J., Johns, L., Wright, D., Hughes, M. D., & Williams, M. J. (2014). Variables associated with patient activation in persons with multiple sclerosis. *Journal of Health Psychology*, 21(1), 82–92.
- Hirvensalo, M., Rantanen, T., & Heikkinen, E. (2000). Mobility difficulties and physical activity as predictors of mortality and loss of independence in the community-living older population. *Journal of the American Geriatrics Society*, 48(5), 493–498.
- Hobart, J. C., Riaz, A., Lamping, D. L., Fitzpatrick, R., & Thompson, A. J. (2003). Measuring the impact of MS on walking ability The 12-Item MS Walking Scale (MSWS-12). *Neurology*, 60(1), 31–36.
- Jones, K. H., Ford, D. V., Jones, P. A., John, A., Middleton, R. M., Lockhart-Jones, H., ... Noble, J. G. (2012). A large-scale study of anxiety and depression in people with multiple sclerosis: A survey via the web portal of the UK MS register. *PLoS One*, 7(7), e41910.
- Joynt, K. E., Whellan, D. J., & O'Connor, C. M. (2003). Depression and cardiovascular disease: Mechanisms of interaction. *Biological Psychiatry*, 54(3), 248–261.
- Kandola, A., & Hayes, J. F. (March, 2020). Depressive symptoms and objectively-measured physical activity and sedentary behaviour throughout adolescence: A prospective cohort study. *Lancet—Psychiatry*, 7(3), 262–271.
- Kivela, S. L., & Pahlkala, K. (2001). Depressive disorder as a predictor of physical disability in old age. *Journal of the American Geriatrics Society*, 49(3), 290–296.
- Klaren, R. E., Motl, R. W., Dlugonski, D., Brian, M. S., & Lara, A. P. (2013). Objectively quantified physical activity in persons with multiple sclerosis. *Archives of Physical Medicine and Rehabilitation*, 94(12), 2342–2348.
- LaRocca, N. G. (2011). Impact of walking impairment in multiple sclerosis. *The Patient*, 4(3), 189–201.
- Lenz, E. R., & Pugh, L. C. (2003). The theory of unpleasant symptoms. In M. J. Smith & P. R. Liehr (Eds.), *Middle range theory for nursing* (pp. 69–90). New York, NY: Springer.
- Marrie, R. A., Rudick, R., Horwitz, R., Cutter, G., Tyry, T., Campagnolo, D., & Vollmer, T. (2010). Vascular comorbidity is associated with more rapid disability progression in multiple sclerosis. *Neurology*, 74(13), 1041–1047.
- Molton, I. R., Hirsh, A. T., Smith, A. E., & Jensen, M. P. (2013). Age and the role of restricted activities in adjustment to disability-related pain. *Journal of Health Psychology*, 19(8), 1025–1034.
- Motl, R. W., Balantrapu, S., Pilutti, L., Dlugonski, D., Suh, Y., Sandroff, B. M., ... Fernhall, B. (2013). Symptomatic correlates of six-minute walk performance in persons with multiple sclerosis. *European Journal of Physical and Rehabilitation Medicine*, 49(1), 59–66.
- Motl, R. W., & Learmonth, Y. C. (2014). Neurological disability and its association with walking impairment in multiple sclerosis: Brief review. *Neurodegenerative Disease Management*, 4, 491–500.
- Motl, R. W., McAuley, E., & Sandroff, B. S. (2013). Longitudinal change in physical activity and its correlates in relapsing-remitting multiple sclerosis. *Physical Therapy*, 93(8), 1037–1048.
- Motl, R. W., Sandroff, B. M., & DeLuca, J. (2015). Exercise training and cognitive rehabilitation a symbiotic approach for rehabilitating walking and cognitive functions in multiple sclerosis? *Neurorehabilitation and Neural Repair*. doi:10.1177/1545968315606993
- Motl, R. W., Snook, E. M., & Shapiro, R. T. (2008). Symptoms and physical activity behavior in individuals with multiple sclerosis. *Research in Nursing & Health*, 31(5), 466–475.
- Penninx, B. W., Leveille, S., Ferrucci, L., van Eijk, J. T., & Guralnik, J. M. (1999). Exploring the effect of depression on physical disability: Longitudinal evidence from the established populations for epidemiologic studies of the elderly. *American Journal of Public Health*, 89(9), 1346–1352.

- Senders, A., Sando, K., Wahbeh, H., Peterson Hiller, A., & Shinto, L. (2014). Managing psychological stress in the multiple sclerosis medical visit: Patient perspectives and unmet needs. *Journal of Health Psychology, 21*(8), 1676–1687.
- Siebert, R. J., & Abernethy, D. A. (2005). Depression in multiple sclerosis: A review. *Journal of Neurology, Neurosurgery & Psychiatry, 76*(4), 469–475.
- Stegenga, B. T., Nazareth, I., Torres-Gonzalez, F., Xavier, M., Svab, I., Geerlings, M. I., ... King, M. (2012). Depression, anxiety and physical function: Exploring the strength of causality. *Journal of Epidemiology and Community Health, 66*(7), e25.
- Suh, Y., Motl, R. W., & Mohr, D. C. (2010). Physical activity, disability, and mood in the early stage of multiple sclerosis. *Disability and Health Journal, 3*(2), 93–98.
- Taylor, S. (2012). *Health psychology* (8th ed.). New York, NY: McGraw-Hill.
- Van Asch, P. (2011). Impact of mobility impairment in multiple sclerosis 2-patients' perspectives. *European Neurological Review, 6*(2), 115–120.
- Voss, W. D., Arnett, P. A., Higginson, C. I., Randolph, J. J., Campos, M. D., & Dyck, D. G. (2002). Contributing factors to depressed mood in multiple sclerosis. *Archives of Clinical Neuropsychology, 17*(2), 103–115.
- Zeiss, A. M., Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1996). Relationship of physical disease and functional impairment to depression in older people. *Psychology and Aging, 11*, 572–581.

Suggested Reading

- Ensari, I., Adamson, B. C., & Motl, R. W. (2016). Longitudinal association between depressive symptoms and walking impairment in people with relapsing-remitting multiple sclerosis. *Journal of Health Psychology, 21*(11), 2732–2741. doi:10.1177/1359105315584837
- Motl, R. W., & Learmonth, Y. C. (2014). Neurological disability and its association with walking impairment in multiple sclerosis: Brief review. *Neurodegenerative Disease Management, 4*(6), 491–500.
- Suh, Y., Motl, R. W., & Mohr, D. C. (2010). Physical activity, disability, and mood in the early stage of multiple sclerosis. *Disability and Health Journal, 3*(2), 93–98.

The Impact of Exposure to Potentially Traumatic Events on Physical Health

Candice Presseau¹, Danielle S. Berke¹, Julie D. Yeterian¹,
and Brett Litz²

¹*Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC),
VA Boston Healthcare System, Boston, MA, USA*

²*Department of Psychology, Boston University & VA Boston Healthcare System,
Boston, MA, USA*

Exposure to potentially traumatic events (PTEs), such as combat, accidents, and sexual assault, is associated with negative alterations in biology that may create greater risk of cardiovascular, gastrointestinal, and immunological problems. Unlike physical injuries that cause direct insult to various biological systems, there is no direct causal link between psychological trauma and health problems. Rather, a variety of biopsychosocial processes—some immediate, some delayed, and most chronically present—increase the risk for physical health problems. In particular, there are two modifiable pathways in the behavioral medicine arena through which PTEs can negatively affect physical health: (a) enduring dysregulation of stress reactions and (b) use of emotional inhibition strategies for regulating distress. We also discuss the potential implications of these pathways for the assessment and treatment of health problems that may develop following exposure to PTEs.

Potentially Traumatic Events and Posttraumatic Stress Disorder

Events are not inherently traumatic. Situations that involve actual or threatened death, serious injury, or sexual violence toward self or others are *potentially* traumatic. A stressor is *traumatic* if it entails sufficiently intense and functionally impairing reactions to these experiences. It is a testament to human resilience that most people that are exposed to PTEs do not experience acute or enduring traumatic stress reactions. PTEs may be chronic and repeated (e.g., ongoing childhood abuse, living in a war zone, or repeated intimate partner violence) or single incidents

(e.g., a motor vehicle accident, natural disaster, or armed robbery). Rates of PTE exposure vary widely within and across specific populations (e.g., more common among male, individuals with preexisting behavioral disorders, Caucasian persons, and those of lower socioeconomic statuses; Amstadter, Aggen, Knudsen, Reichborn-Kjennerud, & Kendler, 2013; McLaughlin et al., 2013; Roberts, Gilman, Breslau, Breslau, & Koenen, 2011). However, up to 90% of the US population will experience one or more PTE(s) at some point in their lives (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Kilpatrick et al., 2013). As such, PTE exposure is the rule, rather than the exception, in American and other societies worldwide (e.g., Benjet et al., 2016).

In response to PTEs, individuals may develop psychopathological syndromes, including acute stress disorder, posttraumatic stress disorder (PTSD), adjustment disorders, and other trauma- and stressor-related disorders, including complex bereavement (American Psychiatric Association, 2013). Likewise, it is not uncommon for individuals who have been exposed to PTEs to experience difficulties with depression and/or substance use, which may be comorbid with trauma-related disorders (Mills, Teesson, Ross, & Peters, 2006; Rytwinski, Scur, Feeny, & Youngstrom, 2013). The most widely studied of the trauma-related disorders, PTSD, includes symptoms across four domains: (a) intrusions (i.e., dreams, intrusive thoughts and memories, strong emotional and physical reactions to trauma reminders), (b) avoidance (i.e., of thoughts, feelings, situations, or people that serve as reminders), (c) alterations in mood and cognition (e.g., loss of interest in activities, negative thoughts about self and others, difficulty experiencing positive emotions), and (d) hyperarousal (e.g., sleep problems, concentration difficulties, irritable behavior). To meet criteria for PTSD, symptoms must last for at least a month.

Lifetime prevalence of PTSD is estimated to be 5–10% in the general population (Friedman, 2015; Kilpatrick et al., 2013), meaning that roughly 1 in 10 people will meet diagnostic criteria for PTSD at some point in their lifetime. In a given year, around 8.5 million adults in the United States (or 3.5%) are estimated to meet criteria for PTSD (Kessler, Chiu, Demler, & Walters, 2005). Most individuals who are exposed to PTEs do not develop a mental illness, such as PTSD, but instead exhibit psychological and behavioral resilience (e.g., Bonanno, 2004). Resilience refers to, “the ability to *bounce back* from immediate understandably disrupted states and from initial pre-clinical responses and impairments” (Litz, 2014, p. 2).

Exposure to PTEs and Physical Health

In addition to mental and behavioral health sequelae, PTE exposure can also impact physical health. Epidemiological studies have found associations between PTEs and subjective perceptions of health and self-reported medical problems and medical records-based medical diagnoses (Pacella, Hruska, & Delahanty, 2013). Findings from the *Adverse Childhood Experiences Study* showed that the risk of developing diseases in adulthood, including cancer, diabetes, coronary artery disease, and chronic lung diseases, was highest for individuals with the greatest number of PTEs or extremely stressful experiences in childhood, even after controlling for age, gender, race, and education level (Felitti et al., 1998). Single PTEs, such as motor vehicle accidents, natural disasters, and sexual traumas, can also bring about stress reactions and subsequent health problems. For instance, sexual trauma in adulthood is associated with negative health consequences, including somatic complaints and chronic diseases (e.g., Campbell, Self, & Ahrens, 2003). Moreover, longitudinal research with older adults shows that PTEs across the life course are associated with physical health problems to varying

degrees (i.e., the death of a child associated with more chronic and acute health conditions; Krause, Shaw, & Cairney, 2004).

PTSD appears to be the key set of symptoms that mediates the relationships between PTE exposure and poor health (e.g., Schnurr & Green, 2004). However, exposure to PTEs is associated with worse physical health even when controlling for PTSD and trauma-related mental health symptoms (Scott et al., 2013; Spitzer et al., 2009). Indeed, a large multinational survey found that individuals with greater lifespan exposures to PTEs were more likely to suffer from physical health conditions such as chronic lung diseases, ulcers, heart disease, and pain conditions, even after controlling for 16 other mental health conditions, including PTSD, mood and anxiety disorders, and substance use disorders (Scott et al., 2013). The authors speculated that health risk behaviors such as binge eating, smoking, substance use, and risky sexual behaviors might explain, at least in part, the relationship between PTE exposure and poor health, as health risk behaviors were also found to be elevated among individuals with greater PTE exposure. Yet, other studies have shown that the relationship between PTE exposure and health consequences is present even when health risk behaviors are accounted for (e.g., Schnurr, Spiro, & Paris, 2000). Although health risk behaviors (e.g., smoking, diet, checkups, exercise) among individuals with lifespan exposure to PTEs should be targeted in prevention efforts within healthcare settings, they are not likely to be the sole mechanism through which health problems emerge following PTE exposure. Other possible explanations for the relationship between exposure to PTEs and negative physical health outcomes include distinct, albeit potentially co-occurring, pathways of dysregulated stress responses and inhibited emotional processing that can operate in conjunction with or independently of trauma-related mental health symptoms and poor health behaviors.

Dysregulated Stress Reactions

The experience of severe stress can lead to over-activation or dysregulation of normal stress responses, thereby increasing wear and tear demands on the body in both the short and longer term. During a stressful encounter, the brain activates regulatory processes to increase the body's ability to respond to the stressor (sympathetic nervous system) and afterward to assist the body in returning to a resting state when the stressor has dissipated (parasympathetic nervous system). The brain's evaluation of a stressor triggers a cascade of reactions across several interconnected systems, including the autonomic, neuroendocrine, and immunological systems, resulting in physiological changes (e.g., increased heart rate, shortness of breath, hormone secretion) and behavioral responses (flight, fight, freeze, dissociate) with implications for physical health functioning.

Neurons, the basic building blocks of the human brain's functioning, are organized in networks that transmit signals as new experiences are encountered in the environment. Neurons are the means of communication between the brain and body and are influenced both structurally and chemically by environmental factors. The ever-changing quality of neurons, while facilitating adaptation, also enables information storage, or memory. As such, the brain and body may learn through neural circuits to habitually operate in ways that lead to physical wear and tear (e.g., persistent rapid heart rate, shallow breathing).

Neural networks are organized primarily to facilitate the maintenance of stability, or equilibrium, of the brain and body in response to change, referred to as allostasis. Hormones, neurotransmitters, and other bodily tissues are mediators of allostasis and aid in the body's functioning during daily activities, like eating, sleeping, and exercising (McEwen, 2003). When individuals

are under extreme stress, the production and dissemination of allostatic mediators can lead to “allostatic load,” wherein excessive demands for change accumulate and cause deterioration of the brain and body.

Allostatic load is manifested primarily in the neuroendocrine system, which includes the sympathetic–adrenal–medullary (SAM) system and hypothalamic–anterior pituitary–adrenal cortex (HPA) axis, and is responsible for hormone production and regulation. The HPA axis is the primary regulatory system for glucocorticoids, mainly cortisol. Cortisol is perhaps most intimately linked with exposure to psychosocial stress and is involved in a variety of functions, such as energy release, immunity, mental vigilance, memory, and learning (Flinn & England, 1995).

Under conditions of chronic stress, such as in war or ongoing abuse and neglect, individuals are faced with persistent threats and elevated arousal for prolonged periods. Therefore, frequent exposure to PTEs places a heavy burden on bodily systems. Persistent over-activation of the HPA system is associated with poor health outcomes, including immune deficiencies, cognitive impairment, growth and maturity delays, and psychological difficulties (Flinn & England, 1995).

It may not only be *how often* one is exposed that determines vulnerability to health effects but also *when* in the life course such exposure occurs. There are time periods during which brain development and associated functions are especially vulnerable to extreme stress and adversity. PTE exposure during critical developmental periods can change the way the brain is organized and ultimately the way it functions (Perry, Pollard, Blaichley, Baker, & Vigilante, 1995). Specifically, PTE exposure occurring during formidable developmental periods provides a neural roadmap for the brain to integrate new sensory information, thereby establishing patterns of neural activities as well as sensitization to particular information inputs. Because the primary mode of the brain under such circumstances is survival, learning related to other sensory inputs is potentially stunted.

Furthermore, given that brain systems are developed in an exceedingly complex fashion (brain stem to cortex), limited development of less complex systems is detrimental to the brain’s overall functioning and capacity to engage in more sophisticated processing, such as regulation of mood and abstract thinking (Perry et al., 1995). Accordingly, state-dependent physiological and biochemical reactions can become personal attributes, or traits, that create additional vulnerabilities for neuropsychiatric symptoms (e.g., anxiety) and physical health problems (e.g., cardiovascular problems). Imagine a developing brain essentially hardwired to perceive threat and a body programmed to produce threat-related responses (e.g., heart rate, respiratory changes, and stress hormone secretion), and the potential for health problems becomes evident.

Inhibitory Emotion Regulation Styles

Exposure to PTEs may also contribute to negative health outcomes by way of inhibitory emotional coping strategies, which include attempts to avoid or escape internal experiences such as thoughts, emotions, bodily sensations, or memories (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Individuals who attempt to cope with trauma-related emotions, thoughts, and memories by suppressing these reactions may inadvertently generate physical distress. Although the link from PTE exposure to negative health outcomes by way of emotion inhibition remains speculative, a wealth of research suggests that use of inhibitory emotional coping strategies has the paradoxical effect of increasing physiological arousal (e.g., Gross, 2002).

Research shows that individuals who engage in strategies designed to inhibit, avoid, or escape internal experiences following PTE exposure are more likely to report psychological distress and impairment (Plumb, Orsillo, & Luterek, 2004) and may be at increased risk for

the onset of slow-developing multifactorial disease processes, such as cardiovascular disease (Gross, 2002). For example, in a longitudinal study of heart attack survivors divided into groups (combinations of high versus low inhibition and high versus low distress), individuals who scored highest on measures of distress and inhibition had a significantly higher mortality rate than other patients (Denollet et al., 1996). Moreover, PTE-exposed persons who attempt to avoid aversive memories by suppressing intrusive thoughts are more likely to report PTSD reexperiencing symptoms (Shipherd & Beck, 2005). Taken together, these findings suggest that use of inhibitory coping may thwart recovery from PTE exposure by perpetuating a cycle wherein individuals both experience increased distress in response to the event(s) and rely on suppressive coping styles that are physically exhausting to manage, or cope with, distress.

Because inhibition and suppression are characterized by a strategic and intentional withholding of emotional responses, such strategies require vigilant self-monitoring and the active stifling of automatic behaviors or bodily reactions (Gross, 2002). Active, effortful emotional inhibition can lead to *ego depletion*, a state in which the ability to alter responses is compromised (Baumeister, Bratslavsky, Muraven, & Tice, 1998). As such, individuals who rely on emotion suppression to cope with painful thoughts and memories of trauma may deplete their mental capacity to engage in other effortful processes, such as attention, encoding of memory, and the processing of mood inconsistent information. Arguably, the depleting effects of effortful inhibition can influence health by taxing cognitive processes essential for regulating health (Gross, 2002), including the ability to attend to subtle internal and external cues, to encode and remember important health guidelines (e.g., medication schedule), and to engage in mood inconsistent behavior (e.g., exercising when feeling apathetic or depressed).

Strategies that create distance between an individual and their emotional pain following PTEs (i.e., emotional suppression and inhibition) also have the potential to create distance between an individual and their social world. In this way, individuals who inhibit emotion in response to PTEs may experience negative health consequences due, in part, to their isolation from the health buffering effects of social support (D'Andrea, Sharma, Zelechowski, & Spinazzola, 2011). Additionally, individuals who are unresponsive to emotional cues have been shown to produce physiological activation in those with whom they socially interact. Suppression of emotional experiences has also been shown to reduce social rapport and impede relationship formation (Butler et al., 2003). These findings suggest that habitual inhibitory coping strategies may exact both personal and social consequences (Butler et al., 2003). As the absence or withdrawal of social support following PTE exposure has been shown to be one of the robust predictors of chronic psychological distress (Charuvastra & Cloitre, 2008), reliance on inhibitory coping strategies may exacerbate the physiological toll of PTE exposure on an individual's psychological and physical health by damaging social bonds. Collectively, the arguments outlined above suggest there may be complex and systemic influences of PTE exposure on physical health for individuals and those around them.

Summary, Clinical Implications, and Conclusions

In this chapter, we reviewed literature demonstrating a relationship between PTE exposure and physical health problems and identified two possible mechanisms through which PTE exposure may give rise to negative health outcomes: (a) dysregulated stress reactions and (b) inhibitory emotion regulation styles. Thus, providers may consider intervening on the mechanisms (physiological or psychological) following PTE exposure that can lead to health problems in addition to targeting health symptoms and behaviors themselves. Theory and

research that explores the PTE exposure–health relationship may have implications for the optimization of healthcare interventions, as targeting underlying processes may provide benefits above and beyond those offered by medication, exercise, and diet changes.

Evidence of PTE exposure should not necessarily prompt a referral to specialty trauma-focused services, as most individuals who are exposed to PTEs will be resilient to the experience and will not suffer long-term mental health consequences. Rather, gathering information about patients' PTE exposure can be used to develop a biopsychosocial conceptualization of health problems that is informed by an appreciation for the interplay between physiological and psychological processes that may be triggered by PTEs. Addressing the intersection of mental and physical health in the context of PTE exposure may serve to enhance the effectiveness of multidisciplinary care.

Healthcare systems have been shifting toward integrative and collaborative models of care. Interdisciplinary teams and collaborative care approaches to health and wellness are common in primary, community health, and specialty care contexts (e.g., Woltmann et al., 2012). In these contexts, for certain medical problems (e.g., chronic pain, gastrointestinal distress), if patients show signs of symptom persistence despite compliance with prescribed otherwise effective treatments (or if there are compliance problems), psychologists should conduct a thorough assessment to determine the presence of exposure to PTEs and dysregulated stress responses and/or emotional inhibition. These problems can be targeted with stress management, mindfulness, and expression-promoting interventions. Unfortunately, at present there are no best practice recommendations for multidisciplinary care among subpopulations of patients who have been exposed to PTEs.

Author Biographies

Candice Presseau, PhD, is a postdoctoral clinical research fellow at the *Massachusetts Veterans Epidemiology Research and Information Center* (MAVERIC) at the VA Boston Healthcare System. Her area of interest and expertise is the phenomenology and treatment of PTSD and co-occurring symptoms and disorders.

Danielle S. Berke, PhD, is a postdoctoral clinical research fellow at the *Massachusetts Veterans Epidemiology Research and Information Center* (MAVERIC) at the VA Boston Healthcare System. Areas of interest and expertise include the phenomenology of interpersonal trauma, the treatment of trauma-related distress, and violence prevention.

Julie D. Yeterian, PhD, is a postdoctoral clinical research fellow at the *Massachusetts Veterans Epidemiology Research and Information Center* (MAVERIC) at the VA Boston Healthcare System. She specializes in the assessment and treatment of PTSD and substance use disorders.

Brett Litz, PhD, is a clinical psychologist and professor in the Departments of Psychiatry and Psychology at Boston University. He is also the director of the Mental Health Core of the *Massachusetts Veterans Epidemiological Research and Information Center* at the VA Boston Healthcare System.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Amstadter, A. B., Aggen, S. H., Knudsen, G. P., Reichborn-Kjennerud, T., & Kendler, K. S. (2013). Potentially traumatic event exposure, posttraumatic stress disorder, and axis I and II comorbidity in

- a population-based study of Norwegian young adults. *Social Psychiatry and Psychiatric Epidemiology*, 48(2), 215–223. doi:10.1007/s00127-012-0537-2
- Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology*, 74(5), 1252–1265. doi:10.1037/0022-3514.74.5.1252
- Benjet, C., Bromet, E., Karam, E. G., Kessler, R. C., McLaughlin, K. A., Ruscio, A. M., ... Alonso, J. (2016). The epidemiology of traumatic event exposure worldwide: Results from the World Mental Health Survey Consortium. *Psychological Medicine*, 46(2), 327–343. doi:10.1017/S0033291715001981
- Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist*, 59(1), 20–28. doi:10.1037/0003-066X.59.1.20
- Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion*, 3(1), 48–67. doi:10.1037/1528-3542.3.1.48
- Campbell, R., Sefl, T., & Ahrens, C. E. (2003). The physical health consequences of rape: Assessing survivors' somatic symptoms in a racially diverse population. *Women's Studies Quarterly*, 31(1/2), 90–104.
- Charuvastra, A., & Cloitre, M. (2008). Social bonds and posttraumatic stress disorder. *Annual Review of Psychology*, 59, 301–328. doi:10.1146/annurev.psych.58.110405.085650
- D'Andrea, W., Sharma, R., Zelechowski, A. D., & Spinazzola, J. (2011). Physical health problems after single trauma exposure when stress takes root in the body. *Journal of the American Psychiatric Nurses Association*, 17(6), 378–392. doi:10.1177/1078390311425187
- Denollet, J., Rombouts, H., Gillebert, T. C., Brutsaert, D. L., Sys, S. U., & Stroobant, N. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *The Lancet*, 347(8999), 417–421. doi:10.1016/S0140-6736(96)90007-0
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245–258. doi:10.1016/S0749-3797(98)00017-8
- Flinn, M. V., & England, B. G. (1995). Childhood stress and family environment. *Current Anthropology*, 36(5), 854–866. doi:10.1086/204444
- Friedman, M. J. (2015). *Posttraumatic and acute stress disorders*. New York, NY: Springer.
- Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology*, 39(3), 281–291. doi:10.1017/S0048577201393198
- Hayes, S. C., Wilson, K. G., Gifford, E. V., Follette, V. M., & Strosahl, K. (1996). Experiential avoidance and behavioral disorders: A functional dimensional approach to diagnosis and treatment. *Journal of Consulting and Clinical Psychology*, 64(6), 1152–1168. doi:10.1037/0022-006X.64.6.1152
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 617–627. doi:10.1001/archpsyc.62.6.617
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, 52(12), 1048–1060. doi:10.1001/archpsyc.1995.03950240066012
- Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman, M. J. (2013). National estimates of exposure to traumatic events and PTSD prevalence using DSM-IV and DSM-5 criteria. *Journal of Traumatic Stress*, 26(5), 537–547. doi:10.1002/jts.21848
- Krause, N., Shaw, B. A., & Cairney, J. (2004). A descriptive epidemiology of lifetime trauma and the physical health status of older adults. *Psychology and Aging*, 19(4), 637–648. doi:10.1037/0882-7974.19.4.637
- Litz, B. T. (2014). Resilience in the aftermath of war trauma: A critical review and commentary. *Interface Focus*, 4, 1–10. doi:10.1098/rsfs.2014.0008
- McEwen, B. S. (2003). Mood disorders and allostatic load. *Biological Psychiatry*, 54, 200–207. doi:10.1016/S0006-3223(03)00177-X

- McLaughlin, K. A., Koenen, K. C., Hill, E. D., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2013). Trauma exposure and posttraumatic stress disorder in a national sample of adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(8), 815–830. doi:10.1016/j.jaac.2013.05.011
- Mills, K. L., Teesson, M., Ross, J., & Peters, L. (2006). Trauma, PTSD, and substance use disorders: Findings from the Australian National Survey of Mental Health and Well-Being. *The American Journal of Psychiatry*, 163(4), 651–658. doi:10.1176/appi.ajp.163.4.652
- Pacella, M. L., Hruska, B., & Delahanty, D. L. (2013). The physical health consequences of PTSD and PTSD symptoms: A meta-analytic review. *Journal of Anxiety Disorders*, 27(1), 33–46. doi:10.1016/j.janxdis.2012.08.004
- Perry, B. D., Pollard, R. A., Blaichley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and “use-dependent” development of the brain: How “states” become “traits”. *Infant Mental Health Journal*, 16(4), 271–291.
- Plumb, J. C., Orsillo, S. M., & Luterek, J. A. (2004). A preliminary test of the role of experiential avoidance in post-event functioning. *Journal of Behavior Therapy and Experimental Psychiatry*, 35(3), 245–257. doi:10.1016/j.jbtep.2004.04.011
- Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological Medicine*, 41(01), 71–83. doi:10.1017/S0033291710000401
- Rytwinski, N. K., Scur, M. D., Feeny, N. C., & Youngstrom, E. A. (2013). The co-occurrence of major depressive disorder among individuals with posttraumatic stress disorder: A meta-analysis. *Journal of Traumatic Stress*, 26(3), 299–309. doi:10.1002/jts.21814
- Schnurr, P. P., & Green, B. L. (2004). Understanding relationships among trauma, post-traumatic stress disorder, and health outcomes. *Advances in Mind-Body Medicine*, 20(1), 18–29.
- Schnurr, P. P., Spiro, A., III, & Paris, A. H. (2000). Physician-diagnosed medical disorders in relation to PTSD symptoms in older male military veterans. *Health Psychology*, 19(1), 91–97. doi:10.1037/0278-6133.19.1.91
- Scott, K. M., Koenen, K. C., Aguilar-Gaxiola, S., Alonso, J., Angermeyer, M. C., Benjet, C., ... Iwata, N. (2013). Associations between lifetime traumatic events and subsequent chronic physical conditions: A cross-national, cross-sectional study. *PLoS One*, 8(11), e80573. doi:10.1371/journal.pone.0080573
- Shipherd, J. C., & Beck, J. G. (2005). The role of thought suppression in posttraumatic stress disorder. *Behavior Therapy*, 36(3), 277–287. doi:10.1016/S0005-7894(05)80076-0
- Spitzer, C., Barnow, S., Völzke, H., John, U., Freyberger, H. J., & Grabe, H. J. (2009). Trauma, post-traumatic stress disorder, and physical illness: Findings from the general population. *Psychosomatic Medicine*, 71(9), 1012–1017. doi:10.1097/PSY.0b013e3181bc76b5
- Woltmann, E., Grogan-Kaylor, A., Perron, B., Georges, H., Kilbourne, A. M., & Bauer, M. S. (2012). Comparative effectiveness of collaborative chronic care models for mental health conditions across primary, specialty, and behavioral health care settings: Systematic review and meta-analysis. *American Journal of Psychiatry*, 169(8), 790–804. doi:10.1176/appi.ajp.2012.11111616

Suggested Reading

- D’Andrea, W., Sharma, R., Zelechowski, A. D., & Spinazzola, J. (2011). Physical health problems after single trauma exposure when stress takes root in the body. *Journal of the American Psychiatric Nurses Association*, 17(6), 378–392. doi:10.1177/1078390311425187
- Perry, B. D., Pollard, R. A., Blaichley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and “use-dependent” development of the brain: How “states” become “traits”. *Infant Mental Health Journal*, 16(4), 271–291.
- Schnurr, P. P., & Green, B. L. (2004). Understanding relationships among trauma, post-traumatic stress disorder, and health outcomes. *Advances in Mind-Body Medicine*, 20(1), 18–29.

Schizophrenia

Thanh Le, Kyle R. Mitchell, Elana Schwartz,
Jessica McGovern, and Alex S. Cohen

*Department of Psychology, Louisiana State University College of Science,
Baton Rouge, LA, USA*

Overview

Schizophrenia is a severe brain disease that is one of the most costly sources of chronic dysfunction in the world (World Health Organization [WHO], 2001). Its operational definition rests on the clinical presentation of delusions, hallucinations, disorganized behavior and speech, and negative symptoms (e.g., anhedonia, asociality, blunted affect, and alogia) (American Psychiatric Association, 2013), though virtually every function associated with the central nervous system is affected in some regard. It has a lifetime prevalence of approximately 0.5–1%, though schizophrenia spectrum disorders and subclinical traits likely affect a much broader segment of the population (MacDonald & Schulz, 2009). Despite decades of empirical attention, its cause has remained a mystery, and a cure is unlikely to manifest in the near future. Nonetheless, significant gains have been made in understanding the functional consequences of the disorder and in symptom amelioration. At present, a regimen of case management, antipsychotic medication, and various psychosocial interventions has helped reign in symptoms to a fairly manageable level for many individuals with the disease (Dixon et al., 2010). What remains is a constellation of cognitive, emotional, and motivational symptoms that impairs individual's ability to effectively function within mainstream social and vocational domains. In order to improve quality of life, improve functioning, and reduce social burden associated with schizophrenia, there is considerable effort aimed at understanding, assessing, and treating these “residual” symptoms within the disorder. This is no easy endeavor, as these efforts are pushing the very limits of translational, basic, and clinical sciences. With that in mind, the present chapter provides an overview of recent research in this domain with a particular focus on methodological, multidisciplinary, and technological advances that will hopefully improve the lives of patients with schizophrenia and their caregivers in the near future.

Cognitive Deficits

Individuals with schizophrenia experience relatively severe cognitive impairments, on the order of a full standard deviation across a wide range of basic (e.g., attention, memory), social, and metacognitive abilities. These impairments have been identified in the premorbid and prodromal stages of the disorder, among patients experiencing their first episode of psychosis, and throughout adulthood (Addington, Saeddi, & Addington, 2006; Cornblatt, Green, Walker, & Mittal, 2009). These impairments are generally unresponsive to current psychiatric medication. Importantly, cognitive impairments are one of the most robust predictors/concomitants of functional outcome both concurrently and prospectively. More specifically, deficits in basic cognitive abilities are reliably and highly associated with lower quality of life, poorer social skills, higher unemployment rates, difficulty with independent living, less time spent in the community, and reduced success following rehabilitation programs (Green, Kern, Braff, & Mintz, 2000). Furthermore, studies examining the effects of composite cognitive measures have found that cognitive impairment may account for 20–60% of the variance in real-world outcomes even after accounting for psychotic and other psychiatric symptoms (Green et al., 2000). These findings coupled with a review of the extensive literature have led some investigators to propose that cognitive impairments are the most important factor for understanding long-term disability associated with the disorder (Cornblatt et al., 2009).

While it is clear that schizophrenia is associated with severe cognitive deficits, the true nature of the cognitive dysfunction remains unclear. Traditionally, test batteries partition global cognition into separate domains such as memory, attention, executive functioning, and more. Partly due to this fact coupled with hopes of informing tailored interventions, many studies have provided evidence that discrete cognitive domains are largely responsible for social, occupational, and other dysfunctions (Green et al., 2000). However, other investigators challenge this view of specificity. For example, prior studies have shown that traditional domains of cognitive function are highly interrelated, indicating that the selective deficits observed exist against a backdrop of generalized cognitive deficits (Dickinson, Ragland, Gold, & Gur, 2008). This specific versus general issue raises fundamental questions about the neural underpinnings of cognitive impairments and has direct implications for assessment practice and intervention design. Additionally, investigators are focused on identifying mechanisms and mediators that may explain *how* basic cognition is related to functional outcome and identify further treatment targets. Social cognition, broadly defined as the ability of individuals to correctly identify and adaptively make sense of others' thoughts, emotions, intentions, and social cues, has emerged as a likely mediator as it requires basic cognitive skills and has obvious links to social behaviors. The rapid growth in the study of social cognition has led to important findings: social cognition has stronger relationships with social functioning than cognition (Fett et al., 2011), and some aspects of social cognition (e.g., social perception and social knowledge) fully mediate the relationship between cognition and social problem solving and work skills (Addington et al., 2006). More recently, metacognition has also been examined as a potential mediator between basic cognition and everyday outcomes. Metacognition can be defined as a spectrum of mental activities that involve thinking about thinking and, more specifically, describes the ability of an individual to recognize and hold specific thoughts and feelings of complex representations of self and other over time (Lysaker et al., 2011). Similar to social cognition, metacognition has demonstrated relationships to occupational and social functioning (Lysaker et al., 2011), and aspects of metacognition (e.g., mastery) have been found to fully mediate the influence of cognition on social contact and empathy (Lysaker et al., 2010).

Given the ubiquitous influences of cognitive impairments in schizophrenia, current efforts to understand the exact nature of these substantial and persistent deficits have led to unprecedented cooperation between multiple federal agencies, private pharmaceutical companies, and academia. For example, various stakeholders, including researchers, the National Institute of Mental Health (NIMH), the Federal Drug Agency (FDA), and industry, started two initiatives—the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) and the Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia (CNTRICS)—that resulted in the development of consensus cognitive test batteries assessing domains of cognition central to schizophrenia. More specifically, the MATRICS initiative (Nuechterlein et al., 2008) focused on identifying tasks with established psychometric properties to include in a standardized test battery that would be utilized in clinical trials aimed at ameliorating cognitive dysfunction in schizophrenia. In contrast, the CNTRICS initiative (Carter & Barch, 2007) focuses on leveraging cognitive neuroscience-based methods and technologies (e.g., neuroimaging, electrophysiology, and animal paradigms) to isolate cognitive functions that may be conflated in more traditional batteries, such as the MATRICS, where a single cognitive task may tap multiple cognitive processes (e.g., visual scanning, attention, problem solving, etc.). Researchers have also investigated the heritability and genetic component of common cognitive deficits, or endophenotypes, shared by individuals with schizophrenia and their healthy relatives. Studies such as the Consortium on the Genetics of Schizophrenia (COGS), a multisite NIMH-sponsored collaboration, are making groundbreaking discoveries on the genetic architecture on these endophenotypes that can foster a better understanding of pathophysiology of schizophrenia, improve early identification of the illness, and guide the development of preventive interventions (Swerdlow, Gur, & Braff, 2015). Lastly, cognitive remediation (CR) (discussed below), which involves the systematic use of methods aimed at improving cognitive functioning through the practice of cognitive exercises on either computer-based or paper-and-pencil tests, have been found to improve cognitive outcomes, with small to moderate effect (Revell, Neill, Harte, Khan, & Drake, 2015).

Emotion Abnormalities

Individuals with schizophrenia experience a range of emotion abnormalities, including abnormalities in emotion regulation, emotion recognition, and emotion experience and expression. Meta-analytic studies examining emotion perception indicate that individuals with schizophrenia evidence deficits on the order of one standard deviation below controls (Kohler, Walker, Martin, Healey, & Moberg, 2010). Individuals with schizophrenia also show abnormalities in emotion regulation, or the ability to influence the timing and intensity, as well as how they experience and express these emotions (van der Meer, van't Wout, & Aleman, 2009). These individuals report higher trait negative and lower positive affect than controls (Kring & Elis, 2013). Whereas healthy controls typically employ healthy emotion regulation skills like suppression or reappraisal of negative emotions, individuals with schizophrenia use fewer healthy skills and use more unhealthy skills like avoidance of negative emotion (van der Meer et al., 2009). Individuals with schizophrenia also exhibit deficits in measures of verbal, facial, and acoustic emotional expression (Kring & Elis, 2013), but the exact nature of these deficits is unclear at the present time.

Of the myriad of emotional abnormalities, anhedonia, or a reduction in capacity to experience pleasure, is a poorly understood but crucial emotion deficit in schizophrenia. Anhedonia is evident in a number of neuropsychiatric disorders. In schizophrenia, anhedonia is part of a

subset of symptoms consistent with reductions in normal functioning or negative symptoms. These symptoms are a negative prognostic indicator, and in particular anhedonia is associated with deficits in social and occupational functioning (Kring & Elis, 2013). Despite the range of functional impairments associated with anhedonia, researchers have only recently begun to address these symptoms. Emerging research indicates that anhedonia in schizophrenia is not merely an extension of the symptoms seen in other neuropsychiatric disorders. In fact, recent research indicates that anhedonia is a complex and unique set of deficits that until recently have been difficult to explain, leading researchers to coin the term the “emotion paradox.” This pattern of deficits consists of deficits in trait hedonic response, but intact in-the-moment hedonic responses (Cohen & Minor, 2010).

A number of cognitive, affective, and neurobiological mechanisms have been proposed to understand anhedonia in schizophrenia. These mechanisms include deficits in autobiographical memory as well as abnormal activation of semantic memory networks that lead to inaccuracies in recall of past pleasurable experiences (Cohen, Najolia, Brown, & Minor, 2011). Another potential cognitive mechanism posits that abnormalities in working memory contribute to dysfunctional beliefs about or representations of pleasure (Strauss, 2013). Deficits in inhibition of negative emotion are a potential mechanism, which suggests that deficits in emotion regulation contribute to self-reported anhedonia (Cohen & Minor, 2010). A number of brain regions are of interest; however, current literature focuses primarily on reward processing circuitry and implicates areas including the ventral striatum (Dowd & Barch, 2010). The results of these studies posit that deficits in reward processing are of primary concern in schizophrenia. Prior research has been informative in clarifying areas of deficit in schizophrenia, but greater fidelity is necessary in understanding the mechanisms associated with this symptom. Informed by our current understanding, future research integrating basic affective neuroscience, behaviorally based research, and self-report and interview data are likely to be instructive in advancing our understanding of anhedonia in schizophrenia.

Motivation and Effort Deficits

Avolition, a deficit in initiation and persistence in goal-directed activities, is a particularly deleterious symptom of schizophrenia. Of note, avolition in schizophrenia is sometimes used interchangeably with decreased drive, amotivation, or apathy (Foussias & Remington, 2010). This symptom may manifest as setting or engaging in less effortful goals, such as more often choosing to watch TV as opposed to goals that require more forethought and planning such as creating a meal for several people. At its worst, individuals high in avolition may become so withdrawn as to appear inert or catatonic.

Avolition and the other negative symptoms respond poorly to existing medications and other treatments (Kirkpatrick, Fenton, Carpenter, & Marder, 2006). Presence and severity of avolition predict worse functioning in terms of daily living skills independent of depressive, cognitive, positive, and other negative symptoms (Foussias & Remington, 2010). Avolition is not merely a withdrawal reaction to onset of psychosis; in fact, it is one of the most commonly described prodromal (“prepsychotic”) features of schizophrenia and is prevalent through onset of the first psychotic episode and through chronic phases of illness (Faerden et al., 2009; Kirkpatrick et al., 2006). Importantly, this deficit in pursuing and maintaining goal-directed activities occurs despite these individuals reporting in the moment that they enjoy the activities at similar levels to healthy controls when they do engage in them (Cohen & Minor, 2010). Given the prevalent, persistent, and pernicious nature of this symptom, it is critical to better

understand the underlying mechanisms that contribute to this deficit and to have reliable, valid, and practical measures of this deficit.

It has become increasingly clear over the last decade that motivation and effort is a complicated and multifaceted construct. One promising objective measure of avolition comes from a strong body of translational evidence directly linking disrupted dopamine functioning in areas sensitive to reward information (e.g., striatum) to reduced willingness to exert effort to obtain larger rewards (see Fervaha, Foussias, Agid, & Remington, 2013). Whereas rodents are typically willing to exert greater effort (e.g., climb over a barrier) to obtain a more desirable reward (e.g., more or tastier food pellets), under a variety of manipulations that decrease dopamine in these key reward areas, rodents tend to prefer the easier choice with a smaller reward (e.g., no barrier for fewer or less “tasty” food pellets; Fervaha et al., 2013). These behavioral patterns mirror findings in humans wherein individuals with schizophrenia tend to prefer more perfunctory tasks over more laborious tasks even when the payoff may be greater. Several effort-based decision-making paradigms have been developed for use in humans, including the Energy Expenditure for Rewards Task (EEfRT) (Treadway et al., 2012) and Balloon Effort Task (Gold et al., 2013). In these tasks, individuals are asked to make several choices between varying low-effort/low-reward (e.g., \$1 for 20 button presses) and a high-effort/high-reward (e.g., \$7 for 100 button presses) options where the high-effort reward value (e.g., \$3, \$7) and probability (e.g., 50, 100%) of reward receipt are varied. This type of paradigm has been shown to activate the same reward pathways as those in the animal models (Treadway et al., 2012). Most importantly, mounting evidence suggests that a similar pattern of effort-based performance deficits found in animal models is evident in schizophrenia; that is, individuals with schizophrenia tend to opt for the low-effort/low-reward choice more often than healthy controls but only under conditions where high-effort rewards were highest and where probability of reward receipt was highest (i.e., under conditions where it would seem to be most “worth it” to exert the extra effort; Barch, Treadway, & Schoen, 2014; Gold et al., 2013). This pattern has been interpreted to mean that individuals with schizophrenia have deficits in their ability to translate reward-relevant information about costs and benefits of allocating effort into goal-directed behavior. This pattern has been associated with negative symptoms or apathy and with worse community and work functioning (Barch et al., 2014; Gold et al., 2013).

Implications for Assessment: Present and Future

Historically, investigators have collected data on dynamic psychological processes through single-occasion interview-based measures or self-report-based measures in a laboratory or clinical setting. However, such measures have a number of drawbacks that challenge both their reliability and validity. For example, individuals with schizophrenia show moderate to severe cognitive deficits across a number of domains directly relevant to autobiographical recall and introspection, including but not limited to impairments in delayed memory, attention, and executive functioning (Green et al., 2000). Additionally, retrospective self-reports are vulnerable to biases such that both psychiatric and healthy populations are more likely to report personally relevant, recently occurring, or mood congruent events (Trull & Ebner-Priemer, 2009). Ecological momentary assessment (EMA) is a powerful and innovative assessment strategy that avoids these common measurement shortcomings and instead captures in-the-moment assessment of personal experiences that are known to change over time (Trull & Ebner-Priemer, 2009). Modern EMA techniques employ electronic mobile devices (e.g., smartphone or personal

data assistants) to deliver a preprogrammed signal that prompts individuals to complete self-report questionnaires that focus on current or very recent states or behaviors. EMA data are more ecologically valid than those collected from traditional retrospective assessment strategies because they repeatedly assess individuals in their normal daily environment and reduce the effect of recall bias that is normally associated with delayed self-reporting.

Several studies have shown that many individuals with severe psychiatric and functional impairment can and will use EMA techniques successfully given the opportunity and appropriate training (Granholm, Ben-Zeev, Fulford, & Swendsen, 2013; Oorschot, Kwapil, Delespaul, & Myin-Germeys, 2009). EMA methodologies have also made important contributions to further our understanding of the emotions, clinical symptoms, and behaviors in individuals with schizophrenia. For example, Gard, Kring, Gard, Horan, and Green (2007) employed an EMA study to help resolve the “emotional paradox” of anhedonia in schizophrenia and found that while patients exhibited deficits in anticipating pleasure from future events, they also exhibited intact consummatory (or in-the-moment) pleasure for current activities. The authors suggest that although patients often self-report appropriate responses to emotional stimuli while experiencing them in the moment, they tend to report less pleasure when describing noncurrent emotions often elicited from trait questionnaires using a retrospective or prospective format. Furthermore, EMA also enables for the examination of dynamic prospective relationships between variables within individuals, thus providing information about directional effects that can inform and enhance interventions for schizophrenia (Granholm et al., 2013). EMA is just beginning to be applied to clinical assessment (Cohen & Elvevåg, 2014).

Implications for Treatment: Present and Future

Current traditional psychosocial treatments aimed at improving cognitive impairments, emotion, and motivation symptoms for individuals with schizophrenia have had limited success thus far. With respect to psychosocial treatment, a recent review and meta-analysis (Revell et al., 2015) of CR administered for approximately 12 weeks (range 8–105) for a total of about 40 hr (range 12–156) found a small positive effect of CR on global cognition. Additionally, traditional antipsychotic medication has not proved to be as beneficial for negative symptoms and cognitive deficits; thus, recent research has examined effects of other drugs such as oxytocin and galantamine. Oya, Matsuda, Matsunaga, Kishi and Iwata (2016) conducted a meta-analysis of seven oxytocin augmentation therapy (OAT) randomized controlled trials in individuals with schizophrenia and found that OAT was superior to a placebo in only a few general scores on a measure of positive and negative symptoms. Limited preliminary research on galantamine has demonstrated positive effects on delayed memory, attention, and processing speed, as well as alogia and social cognition (Koola et al., 2014). Current psychosocial standards of treatment require many resources (e.g., overall cost and time), and the paucity of information for new drug approaches highlights the need for interventions that utilize novel treatment delivery systems and are feasible, flexible, and cost efficient.

Small effect sizes aside, another issue with treating residual symptoms of schizophrenia, particularly psychosocial treatments, involves the cost. Recently, Kazdin and Blase (2011) advocated for a shift in psychotherapy research and practice to aid in the reduction of the mental illness burden by finding new ways to deliver mental health services to those needing but not receiving treatment. For example, interventions that can be administered by a nonprofessional or peer mental health worker may serve as a viable alternative given the cost

associated with a doctoral or master's level clinician provide individual treatment. A recent meta-analysis of randomized control trials (Fuhr et al., 2014) examined the effects of peer-delivered interventions compared with treatment as usual (TAU) or to treatment delivered by a licensed healthcare professional among individuals with a severe mental illness. The researchers found small effects of peer-delivered interventions on quality of life and hope, which suggest that peer-led interventions may be equivalent to professionally delivered interventions at improving clinical symptoms. Alternatively, there have been recent efforts to disseminate therapeutic interventions through mobile devices (e.g., smartphones, tablets) to individuals with schizophrenia. Granholm, Ben-Zeev, Link, Bradshaw, and Holden (2012) designed a mobile text message intervention to target medication adherence, socialization, and auditory hallucinations and was administered three times per day, 6 days a week for 12 weeks. This study reported improvements in all of the treatment domains for many of the consumers as well as an 83–86% valid response rate. In another recent study, Ben-Zeev et al. (2014) utilized a smartphone intervention designed to provide illness management support to individuals with schizophrenia. Participants received three prompts per day for 1 month that provided tailored interventions. In this study, participants utilized the smartphone on 86% of the days they had the device, and reductions in positive, global, and depressive symptomatology were evident. More research is needed to understand the clinical efficacy of these peer-led and mobile interventions, though preliminary results suggest that peer-delivered and mobile applications appear to be a feasible and promising option in conjunction with or as an alternative for traditional therapeutic approaches.

Conclusions

By almost any criterion, schizophrenia is among the most debilitating of psychiatric disorders and has profound influence on the lives of people with the illness. While symptoms such as hallucinations and delusions are modestly tractable, “residual” symptoms (e.g., cognitive, emotion, and motivation) endure beyond acute episodes and are prognostic of nearly every facet of daily functioning. Our goal in this chapter has been to summarize our understanding of three complex residual symptoms of schizophrenia. First, we documented that basic, social, and metacognitive impairments have a highly deleterious effect on social and functional outcome. Second, we illustrated that anhedonia in schizophrenia does not reflect a reduced capacity to experience pleasure in the moment, but rather reflect deficits in anticipating and retrospectively reporting degree of pleasure. Third, we presented compelling evidence that disrupted dopamine functioning and effort valuation may underlie avolition. Furthermore, translating theories and methodologies from cognitive and affective neuroscience, genetics, and emerging technologies (e.g., mobile devices) have enabled investigators to more accurately pinpoint the mechanisms underlying these symptoms. The routine use of interview and self-report measures, translational laboratory measures, and EMA may be the only way to ensure comprehensive coverage of domains that needed to be targeted during treatment.

To conclude, the search for effective schizophrenia treatment continues. Schizophrenia research is continuously evolving and incorporating scientific, methodological, and technological advances. Increased collaboration among various stakeholders and vast resources allocated in an effort to understand and treat the illness has also been observed. Despite the challenge of curing schizophrenia, there is cause for optimism so long as innovative researchers and clinicians continue to leverage contemporary translational research and emerging technologies in the search for the next-generation treatment.

Author Biographies

Thanh Le graduated from the University of California, San Diego, in 2011 and is a graduate student who works with Dr. Alex Cohen at Louisiana State University. His interests include utilizing behavioral and mobile assessments to understand the cognitive and affective mechanisms underlying social functioning impairments in serious mental illness.

Kyle R. Mitchell graduated from the University of Texas at Austin in 2012 and is a graduate student who works with Dr. Alex Cohen at Louisiana State University. His interests are affective abnormalities, their relationship to neurocognitive systems in individuals with schizophrenia spectrum disorders, and the application of biobehavioral systems to understanding this relationship.

Elana Schwartz earned a bachelor's degree in psychology at the University of Maryland, College Park, and completed a master's degree in clinical psychology at Towson University under Dr. Kerri Goodwin. Ms. Schwartz is currently working on a PhD in clinical psychology at Louisiana State University mentored by Dr. Alex Cohen.

Jessica McGovern, MA, is a graduate student in the PhD clinical psychology program at LSU. Her broad interests are in the interactions between emotion, cognition, and symptomatology in schizophrenia spectrum disorders as they relate to understanding the underlying neural mechanisms.

Alex S. Cohen, PhD, is the director of the Affective Science and Psychopathology Laboratory at Louisiana State University. He is a licensed clinical psychologist and is an active researcher on understanding residual symptoms of schizophrenia.

References

- Addington, J., Saeedi, H., & Addington, D. (2006). Influence of social perception and social knowledge on cognitive and social functioning in early psychosis. *British Journal of Psychiatry*, 189(4), 373–378.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.
- Barch, D. M., Treadway, M. T., & Schoen, N. (2014). Effort, anhedonia, and function in schizophrenia: Reduced effort allocation predicts amotivation and functional impairment. *Journal of Abnormal Psychology*, 123(2), 387–397.
- Ben-Zeev, D., Brenner, C. J., Begale, M., Duffecy, J., Mohr, D. C., & Mueser, K. T. (2014). Feasibility, acceptability, and preliminary efficacy of a smartphone intervention for schizophrenia. *Schizophrenia Bulletin*, 40(6), 1244–1253.
- Carter, C. S., & Barch, D. M. (2007). Cognitive neuroscience-based approaches to measuring and improving treatment effects on cognition in schizophrenia: The CNTRICS Initiative. *Schizophrenia Bulletin*, 33(5), 1131–1137.
- Cohen, A. S., & Elvevåg, B. (2014). Automated computerized analysis of speech in psychiatric disorders. *Current Opinion in Psychiatry*, 27(3), 203–209.
- Cohen, A. S., & Minor, K. S. (2010). Emotional experience in patients with schizophrenia revisited: Meta-analysis of laboratory studies. *Schizophrenia Bulletin*, 36(1), 143–150.
- Cohen, A. S., Najolia, G. M., Brown, L. A., & Minor, K. S. (2011). The state-trait disjunction of anhedonia in schizophrenia: Potential affective, cognitive and social-based mechanisms. *Clinical Psychology Review*, 31(3), 440–448.
- Cornblatt, B. A., Green, M. F., Walker, E. F., & Mittal, V. A. (2009). Schizophrenia: Etiology and neurocognition. In P. H. Blaney & T. Millon (Eds.), *Oxford textbook of psychopathology* (pp. 298–332). New York, NY: Oxford University Press.

- Dickinson, D., Ragland, J. D., Gold, J. M., & Gur, R. C. (2008). General and specific cognitive deficits in schizophrenia: Goliath defeats David? *Biological Psychiatry*, 64(9), 823–827.
- Dixon, L. B., Dickerson, F., Bellack, A. S., Bennett, M., Dickinson, D., Goldberg, R. W., ... Kreyenbuhl, J. (2010). The 2009 schizophrenia PORT psychosocial treatment recommendations and summary statements. *Schizophrenia Bulletin*, 36(1), 48–70.
- Dowd, E. C., & Barch, D. M. (2010). Anhedonia and emotional experience in schizophrenia: Neural and behavioral indicators. *Biological Psychiatry*, 67(10), 902–911.
- Faerden, A., Friis, S., Agartz, I., Barrett, E. A., Nesvåg, R., Finset, A., & Melle, I. (2009). Apathy and functioning in first episode psychosis. *Psychiatric Services*, 60(11), 1495–1503.
- Fervaha, G., Foussias, G., Agid, O., & Remington, G. (2013). Neural substrates underlying effort computation in schizophrenia. *Neuroscience and Biobehavioral Reviews*, 37(10 Pt 2), 2649–2665.
- Fett, A.-K. J., Viechtbauer, W., Dominguez, M.-G., Penn, D. L., van Os, J., & Krabbendam, L. (2011). The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, 35(3), 573–588.
- Foussias, G., & Remington, G. (2010). Negative symptoms in schizophrenia: Avolition and Occam's razor. *Schizophrenia Bulletin*, 36(2), 359–369.
- Fuhr, D. C., Salisbury, T. T., De Silva, M. J., Atif, N., van Ginneken, N., Rahman, A., & Patel, V. (2014). Effectiveness of peer-delivered interventions for severe mental illness and depression on clinical and psychosocial outcomes: A systematic review and meta-analysis. *Social Psychiatry and Psychiatric Epidemiology*, 49(11), 1691–1702.
- Gard, D. E., Kring, A. M., Gard, M. G., Horan, W. P., & Green, M. F. (2007). Anhedonia in schizophrenia: Distinctions between anticipatory and consummatory pleasure. *Schizophrenia Research*, 93, 253–260.
- Gold, J. M., Strauss, G. P., Waltz, J. A., Robinson, B. M., Brown, J. K., & Frank, M. J. (2013). Negative symptoms of schizophrenia are associated with abnormal effort-cost computations. *Biological Psychiatry*, 74(2), 130–136.
- Granholm, E., Ben-Zeev, D., Fulford, D., & Swendsen, J. (2013). Ecological Momentary Assessment of social functioning in schizophrenia: Impact of performance appraisals and affect on social interactions. *Schizophrenia Research*, 145(1-3), 120–124.
- Granholm, E., Ben-Zeev, D., Link, P. C., Bradshaw, K. R., & Holden, J. L. (2012). Mobile assessment and treatment of schizophrenia (MATS): A pilot trial of an interactive text-messaging intervention for medication adherence, socialization, and auditory hallucinations. *Schizophrenia Bulletin*, 38(3), 414–425.
- Green, M. F., Kern, R. S., Braff, D. L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: Are we measuring the “right stuff”? *Schizophrenia Bulletin*, 26(1), 119–136.
- Kazdin, A. E., & Blase, S. L. (2011). Rebooting psychotherapy research and practice to reduce the burden of mental illness. *Perspectives on Psychological Science*, 6(1), 21–37.
- Kirkpatrick, B., Fenton, W. S., Carpenter, W. T., Jr., & Marder, S. R. (2006). The NIMH-MATRICES consensus statement on negative symptoms. *Schizophrenia Bulletin*, 32(2), 214–219.
- Kohler, C. G., Walker, J. B., Martin, E. A., Healey, K. M., & Moberg, P. J. (2010). Facial emotion perception in schizophrenia: A meta-analytic review. *Schizophrenia Bulletin*, 36(5), 1009–1019.
- Koola, M. M., Buchanan, R. W., Pillai, A., Aitchison, K. J., Weinberger, D. R., Aaronson, S. T., & Dickerson, F. B. (2014). Potential role of the combination of Galantamine and Memantine to improve cognition in schizophrenia. *Schizophrenia Research*, 157(1-3), 84–89.
- Kring, A. M., & Elis, O. (2013). Emotion deficits in people with schizophrenia. *Annual Review of Clinical Psychology*, 9(1), 409–433.
- Lysaker, P. H., McCormick, B. P., Snethen, G., Buck, K. D., Hamm, J. a., Grant, M., ... Dimaggio, G. (2011). Metacognition and social function in schizophrenia: Associations of mastery with functional skills competence. *Schizophrenia Research*, 131(1-3), 214–218.
- Lysaker, P. H., Shea, A. M., Buck, K. D., Dimaggio, G., Nicolò, G., Procacci, M., ... Rand, K. L. (2010). Metacognition as a mediator of the effects of impairments in neurocognition on social function in schizophrenia spectrum disorders. *Acta Psychiatrica Scandinavica*, 122, 405–413.

- MacDonald, A. W., & Schulz, S. C. (2009). What we know: Findings that every theory of schizophrenia should explain. *Schizophrenia Bulletin*, 35(3), 493–508.
- Nuechterlein, K. H., Green, M. F., Kern, R. S., Baade, L. E., Barch, D. M., Cohen, J. D., ... Marder, S. R. (2008). The MATRICS consensus cognitive battery, part 1: Test selection, reliability, and validity. *American Journal of Psychiatry*, 165(2), 203–213.
- Oorschot, M., Kwapił, T., Delespaul, P., & Myin-Germeys, I. (2009). Momentary assessment research in psychosis. *Psychological Assessment*, 21(4), 498–505.
- Oya, K., Matsuda, Y., Matsunaga, S., Kishi, T., & Iwata, N. (2016). Efficacy and safety of oxytocin augmentation therapy for schizophrenia: An updated systematic review and meta-analysis of randomized, placebo-controlled trials. *European Archives of Psychiatry and Clinical Neuroscience*, 266(5), 439–450.
- Revell, E. R., Neill, J. C., Harte, M., Khan, Z., & Drake, R. J. (2015). A systematic review and meta-analysis of cognitive remediation in early Schizophrenia. *Schizophrenia Research*, 168(1-2), 213–222.
- Strauss, G. P. (2013). The emotion paradox of anhedonia in schizophrenia: Or is it? *Schizophrenia Bulletin*, 39(2), 247–250. doi:10.1093/schbul/sbs192
- Swerdlow, N. R., Gur, R. E., & Braff, D. L. (2015). Consortium on the Genetics of Schizophrenia (COGS) assessment of endophenotypes for schizophrenia: An introduction to this Special Issue of schizophrenia research. *Schizophrenia Research*, 163(1-3), 9–16.
- Treadway, M. T., Buckholtz, J. W., Cowan, R. L., Woodward, N. D., Rui, L., Ansari, M. S., ... Zald, D. H. (2012). Dopaminergic mechanisms of individual differences in human effort-based decision-making. *The Journal of Neuroscience*, 32(18), 6169–6176.
- Trull, T. J., & Ebner-Priemer, U. W. (2009). Using experience sampling methods/ecological momentary assessment (ESM/EMA) in clinical assessment and clinical research: Introduction to the special section. *Psychological Assessment*, 21(4), 457–462.
- van der Meer, L., van't Wout, M., & Aleman, A. (2009). Emotion regulation strategies in patients with schizophrenia. *Psychiatry Research*, 170(2-3), 108–113.
- World Health Organization. (2001). *The World Health Report 2001: Mental health: New understanding, new hope*. Geneva, Switzerland: Author.

Suggested Reading

- Cohen, A. S., & Elvevåg, B. (2014). Automated computerized analysis of speech in psychiatric disorders. *Current Opinion in Psychiatry*, 27(3), 203–209.
- Green, M. F., Kern, R. S., Braff, D. L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: Are we measuring the “right stuff”? *Schizophrenia Bulletin*, 26(1), 119–136.
- MacDonald, A. W., & Schulz, S. C. (2009). What we know: Findings that every theory of schizophrenia should explain. *Schizophrenia Bulletin*, 35(3), 493–508.

Obesity

Michael G. Perri, Melissa H. Laitner, and Aviva H. Ariel

*Department of Clinical and Health Psychology, College of Public Health and Health Professions,
University of Florida, Gainesville, FL, USA*

Scope, Seriousness, and Etiology of Obesity

Prevalence of Obesity

Obesity is characterized by increased body weight caused by an excess accumulation of fat. In adults, it is defined operationally as a body mass index (BMI) (weight in kilograms divided by height in meters squared) of 30 or greater. Individuals with BMIs of 25 or greater but less than 30 are described as “overweight.” During the course of the twentieth century and continuing into the first decade of the current century, the United States witnessed a remarkable rise in the prevalence of obesity. The obesity rate rose from an estimated prevalence of less than 5% in 1900 to more than 36% in 2011–2014, with most of the increase occurring from 1970 to 2010 (Helmchen & Henderson, 2004; Komlos & Brabec, 2011; Ogden, Carroll, Kit, & Flegal, 2014; Ogden, Carroll, Fryar, & Flegal, 2015).

Over the past 40 years, a sharp increase in the prevalence of obesity has also been observed worldwide. Closely associated with economic development, higher rates of obesity have been identified not just in the affluent nations of North America and Europe but also in a broad array of low- and middle-income countries across the globe, particularly in China and India. The World Health Organization (WHO) (2014) estimates that more than 600 million people (approximately 13% of the world’s adult population) are obese and that most people in the world now live in countries where overnutrition and obesity are responsible for more deaths than malnutrition and underweight.

In the United States, data from the National Health and Nutrition Examination Survey (NHANES) indicate that approximately 37% of adults are obese (Ogden et al., 2015) and an additional 34% are overweight (Ogden et al., 2014). Among adults 20 years and older, the prevalence of obesity has risen steadily since the 1970s. Currently, 34.3% of men and 38.3% of women are obese (Ogden et al., 2015). An examination of prevalence in 2011–2014 (Ogden et al., 2015) by race, ethnicity, and gender revealed the following pattern.

The highest rates were observed in non-Hispanic Black adults (56% for women; 37.5% for men) and Hispanic adults (45.7% for women; 39.0% for men) followed by non-Hispanic Whites (35.5% for women; 33.6% for men) and lowest in non-Hispanic Asians (11.9% for women; 11.2% for men).

In children and adolescents ages 2–19 years, obesity is defined as a BMI at or above the 95th percentile of the Centers for Disease Control and Prevention (CDC) sex-specific BMI-for-age growth charts from 2000 (Kuczmarski et al., 2000). Across all ages of young people, the prevalence of obesity in 2011–2014 (Ogden et al., 2015) was 17.1% among girls and 16.9% among boys. Data from 2011 to 2012 (Ogden et al., 2014) indicate an additional 14.4% of girls and 15.3% of boys were overweight (defined as BMI-for-age >85th percentile and <95th percentile of the CDC growth charts). In 2011–2014 (Ogden et al., 2015), the highest rates were seen in Hispanic youth (22.4% for boys; 21.4% for girls) and non-Hispanic Black children and adolescents (20.7% for girls; 18.4% for boys), followed by non-Hispanic White youth (15.1% for girls; 14.3% for boys), and lowest in Asian children and adolescents (11.8% for boys; 5.3% for girls).

Consequences of Obesity

Evidence documenting the deleterious impact of excess weight on health and longevity continues to accumulate. Obesity is directly associated with an array of health complications, including the spectrum of cardiovascular diseases and associated risk factors including ischemic heart disease, angina, myocardial infarction, heart failure, sudden death, stroke, hypertension, dyslipidemia, insulin resistance, and type 2 diabetes, as well as asthma, osteoarthritis, and certain cancers (breast, colorectal, endometrial, kidney, pancreatic, liver, and gallbladder; Jensen et al., 2014).

BMI is positively associated with premature death, disability, and reduced quality of life. Estimates suggest the number of obesity-related deaths per year in the United States may be as high as 400,000 (Mokdad et al., 2003). Obesity exacts a toll not just in terms of morbidity and mortality; it also has substantial economic and psychosocial effects. Obesity accounts for 20.6% of the nation's health expenditures, an amount in excess of \$200 billion dollars per year (Cawley & Meyerhoefer, 2012). Medical costs for obesity increase with degree of obesity such that people with a BMI >40 accrue costs that are two- to threefold greater than normal weight individuals (Arterburn, Maciejewski, & Tsevat, 2005). The social and psychological effects of obesity are profound and include social discrimination, personal distress, decreased emotional well-being, and overall lower health quality of life (Puhl & Heuer, 2010).

Genetic and Environmental Contributors

The determinants of obesity include genetic and environmental factors and their interaction. Several genetic profiles have been identified that suggest body weight is linked to inherited biological factors that regulate energy and expenditure (Albuquerque, Stice, Rodríguez-López, Manco, & Nóbrega, 2015). However, few specific genes have been identified that put an individual at increased genetic risk for obesity, and the expression of genetic influences on body weight appears to be mediated by the interaction between inherited genetic profile and environmental exposure (Rosenquist et al., 2015).

During the latter half of the twentieth century, a variety of important environmental and cultural changes with the potential to influence the expression of genetic predisposition to weight gain occurred. These included (a) major modifications in industrial mechanization that

reduced energy expenditure across a broad array of occupations; (b) the adoption of an automobile-centered way of life and changes to the built environment that discouraged walking; (c) the rise of fast-food restaurants and the ready availability of low-cost, energy-dense foods provided in larger serving sizes than home-cooked meals; (d) the increased proportion of women in the workforce and the greater number of meals eaten away from home; and (e) the introduction and widespread adoption of screen-based entertainment and information exchange technologies (e.g., televisions, personal computers, and the Internet). The confluence of these changes created an “obesogenic environment”—an environment that promoted high energy intake and low energy expenditure. Among genetically susceptible individuals in the population, this combination may have produced increased body weights, thereby contributing to the rise in the prevalence of obesity (Swinburn, Egger, & Raza, 1999). Many of these same factors that contributed to the rise in the prevalence of obesity in the United States are now at play in low- and middle-income countries throughout the world, resulting in the growing global prevalence of obesity (WHO, 2014).

Treatment Options

In the United States, an array of options is available for the management of obesity. These include self-help approaches, commercial weight-loss programs, behavioral or “lifestyle” treatments, pharmacotherapy, and bariatric surgery.

Self-Help Approaches

The majority of people attempting to lose weight do so on their own. However, while anecdotal reports of success are common, empirical studies show on average the degree of weight loss achieved via self-help is quite modest. For example, a recent meta-analysis of 23 studies of self-help approaches to weight management (Hartmann-Boyce, Jebb, Fletcher, & Aveyard, 2015) showed a mean loss of 1.8 kg at 6 months and no changes at 1 year.

Recent technological advances have made Internet and mobile health (mHealth) programs for weight management widely available to help people lose weight on their own. Indeed, numerous devices and applications are available to assist people in tracking and modifying their physical activity, dietary intake, and body weight. However, very few high-quality studies exist to provide empirical support regarding the efficacy of Internet and mHealth interventions for self-management of obesity. Recent reviews suggest that interactive mHealth technologies can enhance the effectiveness of in-person weight-loss intervention, boosting mean weight losses by 1 kg, but the effects of Internet and mHealth programs as stand-alone interventions appear to be minimal (Kodama et al., 2012; Okorodudu, Bosworth, & Corsino, 2015).

Despite the limited effectiveness of self-help programs in general, some individuals achieve and maintain substantial weight reductions. For example, the National Weight Control Registry (NWCR) (Wing & Phelan, 2005) tracks a cohort of obese individuals who have lost 30 pounds or more and maintained that loss for at least 1 year. Understanding factors associated with success among NWCR participants may offer insights into effective self-help strategies for weight management.

Most NWCR members reported using a combination of strategies including decreased energy intake (e.g., reducing fats and sugars, portion control, and counting calories) and increased physical activity (e.g., walking, swimming, and strength training) to achieve their initial weight reductions. To maintain their losses, individuals in the NWCR report participating

in approximately 1 hr/day of moderate-intensity exercise, consuming fewer than 1,400 calories per day, eating the same foods on weekdays and weekends, consuming a healthy breakfast, exerting high levels of control over their eating, and self-monitoring through weekly weighing and/or daily tracking of food intake. The NWCR participants appear to be a relatively unique group of people, and it is unclear how representative they are of the obese population. Nonetheless, the approaches they employ for managing their weight may be useful guides for others dealing with obesity.

Commercial Weight-Loss Programs

Although many overweight and obese individuals seek treatment through commercial weight-loss programs, research regarding the effectiveness of such programs remains relatively limited. In a recent systematic review of 45 studies including 39 randomized controlled trials, Gudzone et al. (2015) found Jenny Craig® and Weight Watchers® had 12-month mean body weight reductions that were 4.9 and 2.6%, respectively, greater than controls. Nutrisystem® showed 3.8% greater body weight loss at 3 months compared with controls, but there was limited information available regarding its longer term effectiveness. Very-low-calorie programs (<800kcal/day) including Health Management Resources®, Medifast®, and OPTIFAST® had initial mean body weight reductions that were 4.0% or more than what was accomplished with dietary counseling, but beyond 6 months this advantage was reduced. Atkins® low-carbohydrate diet showed 0.1–2.9% greater mean weight losses at 12 months compared with counseling, and insufficient information was available regarding the effects of SlimFast®. Thus, there is empirical evidence to suggest that commercial programs can elicit greater weight losses than control conditions, particularly during the first 3–6 months of treatment.

Behavioral “Lifestyle” Interventions

Since the early 1970s, behavioral or “lifestyle” interventions have employed principles derived from learning theory, particularly operant and social cognitive theories, to elicit and maintain changes in eating and activity patterns and thereby produce weight loss. Participants are taught an array of behavior modification strategies to decrease their energy intake and increase their energy expenditure. Goal setting for daily caloric intake (e.g., 1,200kcal/day) and physical activity (e.g., 30 min/day of walking), coupled with daily self-monitoring of foods consumed and minutes of exercise completed, comprise the foundation of behavioral interventions (Butryn, Webb, & Wadden, 2011). Other behavioral modification strategies employed include stimulus control (decreasing environmental triggers for eating), self-reinforcement (rewarding oneself for progress), cognitive reframing (replacing negative thoughts with positive ones), assertiveness training (learning how to decline offers of food), and problem solving (systematic brainstorming to deal with barriers to progress; Wadden, Webb, Moran, & Bailer, 2012). Following an initial phase focused on inducing weight loss, an extended care phase, consisting of 6–12 months of less frequent contacts delivered via face-to-face or telephone, is commonly employed to help participants maintain the behavioral changes and weight losses achieved during initial treatment (Perri et al., 2008).

Obese adults who participate in a behavioral lifestyle intervention lose an average of 8–10% of their initial body weight over the first 4–6 months of treatment with a gradual regaining of weight after treatment ends (Perri et al., 2014; Wadden et al., 2012). Extended care “maintenance” programs appear helpful in slowing or delaying weight regain (Perri et al., 2008).

The landmark Diabetes Prevention Program (DPP) (DPP Research Group, 2002) demonstrated the effects of lifestyle treatment on the prevention of disease. Overweight and obese adults ($n = 3,234$) at risk for diabetes achieved an initial weight loss of 7% and maintained approximately half that amount over 3 years. The lifestyle participants showed a 58% reduction in the incidence of diabetes compared with a placebo control condition, whereas a medication group that received the antidiabetes medication metformin had a 34% decrease. Ten-year follow-up data from the DPP study indicated that the cumulative incidence of diabetes remained lowest for participants in lifestyle treatment compared with those who received metformin or placebo (DPP Research Group, 2009). During the 10-year follow-up period, participants in the lifestyle program on average returned to their baseline weights. Nonetheless, the 10-year incidence of diabetes for the lifestyle participants was 16 and 34% lower than for those in the metformin and placebo conditions, respectively. The DPP researchers concluded that the years spent at a lower weight reduced the overall risk for diabetes among the lifestyle participants (DPP Research Group, 2009).

Further documentation regarding the beneficial health effects of behavioral interventions for weight loss comes from the Look AHEAD study (Look AHEAD Research Group, 2013), the largest ($n = 5,145$) and longest (maximum follow-up = 13.5 years) randomized controlled trial of behavioral treatment for weight loss. The Look AHEAD lifestyle program produced a mean body weight reduction of 8.6% at the end of the first year followed by a gradual regaining of weight over the next 4 years and the maintenance of losses of approximately 4–5% in subsequent years (Look AHEAD Research Group, 2014). At year 8, the participants sustained on average more than half of their initial losses, and the majority successfully attained body weight reductions of 5% or more. Although the Look AHEAD study was not successful in its aim of reducing cardiovascular mortality and morbidity (perhaps due to the greater medication use by participants in the comparison condition), the study was successful in reducing cardiovascular risk factors, insulin use, sleep apnea, and depression and in improving participants' quality of life, physical functioning, and mobility (Look AHEAD Research Group, 2010, 2013).

Collectively, the findings regarding behavioral interventions for obesity indicate that they can produce weight changes of sufficient magnitude to improve health with no risk of serious side effects. Limitations of behavioral treatment include a regaining of lost weight and the limited availability of state-of-the-art interventions in most communities.

Pharmacotherapy

The 2013 Obesity Management Guidelines developed by the American Heart Association, the American College of Cardiology, and the Obesity Society (Jensen et al., 2014) suggest that individuals with a BMI > 30 (or a BMI > 27 with an obesity-related comorbidity) may be appropriate candidates for pharmacotherapy *as an adjunct to a comprehensive lifestyle intervention*. Currently, there are five weight-loss medications approved by the Food and Drug Administration (FDA) for long-term use. Each medication, its mechanism of action, and weight changes commonly associated with its use in clinical trials are described briefly below.

Orlistat (Xenical®)

Orlistat inhibits lipase, the enzyme in the gastrointestinal tract responsible for fat digestion, and reduces the absorption of fats by approximately 30%. Pooled data from a meta-analysis (Rucker, Padwal, Li, Curioni, & Lau, 2007) showed a mean weight reduction with orlistat of approximately 8.6 kg compared with 5.5 kg for dietary counseling plus placebo. This finding

suggests that the incremental weight-loss benefit of orlistat is approximately 3.1 kg. Common side effects associated with orlistat include gastrointestinal distress, including fecal urgency and fecal incontinence.

Naltrexone/Bupropion (Contrave®)

Naltrexone/bupropion is a combination of two FDA-approved drugs, naltrexone and bupropion. Naltrexone is an opioid antagonist used to treat alcohol and opioid dependence. Bupropion is a norepinephrine–dopamine reuptake inhibitor approved as an antidepressant and also used for smoking cessation. Results from a clinical trial that directly examined the effects of behavioral treatment plus placebo versus behavioral treatment plus naltrexone/bupropion showed that among all randomized participants there were 56-week mean weight losses of 4.9 and 7.8 kg, respectively, indicating an incremental benefit of naltrexone/bupropion of approximately 2.9 kg (Wadden et al., 2011). Nausea, constipation, headache, and vomiting are common side effects of the use of naltrexone/bupropion.

Lorcaserin (Belviq®)

Lorcaserin selectively stimulates the 5-HT_{2C} receptor for serotonin and thereby suppresses appetite and increases satiety. In randomized trials (e.g., Smith et al., 2010), lorcaserin has produced mean weight reductions of 6.0–6.6% compared with losses of approximately 3% for diet plus placebo. Hence, the incremental weight-loss benefit of lorcaserin appears to be 3.0–3.6%. Common side effects include headache, dizziness, fatigue, nausea, dry mouth, and constipation. While chemically similar to fenfluramine (a weight-loss drug discontinued due to its association with heart valve disease), the use of lorcaserin has not been associated with an increase in heart valve problems.

Phentermine/Topiramate (Qsymia®)

This compound is a combination of the stimulant phentermine, which is approved for short-term use in weight management, and the antiseizure/antimigraine drug topiramate. Phentermine is a centrally acting appetite suppressant that works in a manner similar to amphetamine. Topiramate appears to assist in weight loss by inhibiting orexigenic glutamate signaling and increasing energy utilization. A randomized controlled trial (Gadde et al., 2011) of the standard dose of phentermine/topiramate showed an incremental benefit of a 6.6% weight loss versus placebo. Dizziness, nausea, and fatigue are common side effects associated with the use of phentermine/topiramate.

Liraglutide (Saxenda®)

Liraglutide, the newest drug to be approved by the FDA for weight loss, is a glucagon-like peptide-1 (GLP-1) receptor agonist that mimics an intestinal hormone that signals satiety. A lower dose of liraglutide (marketed as Victoza®) is used as a treatment for type 2 diabetes. Randomized trial data presented to the FDA suggest that 1 year of treatment with liraglutide produces body weight reductions of 4.0–5.4% above the weight losses accomplished with diet plus placebo (FDA, 2014). Common side effects include nausea, hypoglycemia, and diarrhea, and there is some concern that the use of liraglutide may be associated with acute pancreatitis.

In summary, an array of medications are now available that may enhance the effectiveness of lifestyle treatments for obesity. Each of these pharmacological agents increases weight loss, and each is associated with notable side effects. Moreover, the withdrawal of previous weight-loss

drugs from the market serves as an object lesson that serious adverse effects may become apparent only when a drug is used in larger populations for longer periods of time than in the FDA approval process.

Bariatric Surgery

Surgery may be an appropriate option for adults with a BMI > 40 (or a BMI > 35 with an obesity-related comorbid condition) who have not responded sufficiently to behavioral treatments with or without pharmacotherapy (Jensen et al., 2014). The four types of operations that are commonly offered in the United States are described below.

Adjustable gastric band

In the “gastric banding” procedure, the surgeon places a small band around the top of the stomach to create a pouch that limits the quantity of food that can be ingested, which helps the patient feel full after eating a small amount. The feeling of satiety is governed by the size of the opening between the pouch and the remainder of the stomach. The gastric band contains a circular balloon that allows the surgeon to increase or decrease the size of the opening between the upper and lower portions of the stomach according to the needs of the patient. Gastric banding generally produces slow and steady weight losses that are larger than what is commonly achieved with behavioral interventions but smaller than other bariatric surgery procedures. Mean body weight reductions of 14–16% are commonly observed 1–3 years after surgery (Colquitt, Pickett, Loveman, & Frampton, 2014). While gastric banding has fewer complications than other procedures, it has the highest rate of reoperation (>30%), often related to slippage or malfunctioning of the band.

Roux-en-Y gastric bypass

Gastric bypass, long considered the “gold standard” of bariatric surgeries, involves a complex set of procedures that entail the restriction of food intake coupled with changes in the absorption of nutrients. The surgeon first creates a small pouch at the top of the stomach. Next, the small intestine is connected directly to the pouch so that after food leaves the pouch, it bypasses the remainder of the stomach, the duodenum, and a portion of the upper intestine. Transporting food directly from the pouch into the small intestine changes how the digestive tract absorbs food, resulting in reduced absorption of nutrients and calories consumed. Gastric bypass is more complicated to perform than gastric banding, requires a longer hospital stay, and has a greater risk of near-term surgical complications, but it produces much larger weight losses than gastric banding. Patients commonly lose 30–35% of their body weight within 1 year, and this degree of weight reduction is typically maintained well at follow-ups of 3–10 years (Colquitt et al., 2014). The metabolic and weight-loss effects of gastric bypass typically include partial if not full remission of type 2 diabetes. The disadvantages of gastric bypass include malabsorption of vitamins and minerals (often requiring supplementation) as well as adverse reactions to the consumption of sugary or starchy foods that can cause rapid heart rate, dizziness, abdominal pain, and vomiting.

Vertical sleeve gastrectomy

This irreversible procedure (known as the “gastric sleeve”) is performed by removing approximately 80% of the stomach. What remains is a tube-like pouch that holds a considerably smaller volume than the normal stomach, thereby decreasing the amount of food that can be

consumed. The gastric sleeve induces large, rapid weight loss comparable in magnitude ($>30\%$ of body weight) at 1 year to the gastric bypass (Colquitt et al., 2014). The gastric sleeve also produces favorable changes in the gut hormones that suppress hunger, increase satiety, and improve blood sugar control. Overall, the gastric sleeve appears to be as effective as gastric bypass with respect to weight loss and its impact on type 2 diabetes. Moreover, the rates of complications associated with the gastric sleeve, while higher than gastric banding, are lower than gastric bypass.

Biliopancreatic diversion with a duodenal switch

This surgery, commonly referred to as the “duodenal switch,” is a complex procedure that entails (a) removal of a large portion of the stomach (to increase satiety), (b) rerouting food away from much of the small intestine (to limit the absorption of nutrients), and (c) modifying how bile and other digestive juices influence digestion (to decrease further the absorption of calories). Unlike the other procedures described previously, over time the duodenal switch allows patients eventually to resume eating “normal” amounts of food while experiencing long-term weight losses that are larger than those produced by gastric banding, gastric bypass, and the gastric sleeve procedures (Colquitt et al., 2014). The “duodenal switch” results in beneficial changes to gut hormones (which also occur in the bypass and sleeve procedures) and appears to be the most effective surgical treatment for diabetes. The duodenal switch has higher complication rates and greater risk for mortality than alternative procedures—factors that must be carefully considered by prospective patients.

Bariatric surgery offers individuals with severe obesity the opportunity to achieve very large weight losses that are generally well maintained over the long run and are associated with dramatic improvements in many of the comorbidities associated with severe obesity. The major disadvantages associated with bariatric surgery include the potential for perioperative and postoperative complications that could extend hospitalizations, require additional surgeries, and, in rare cases, result in death. The significant changes in eating patterns associated with most bariatric procedures pose important challenges to many patients. Behavioral counseling is often recommended before and after surgery to help patients prepare for and deal with the significant lifestyle changes dictated by bariatric surgery.

Clinical Guidelines

The American College of Cardiology, the American Heart Association, and the Obesity Society have developed a comprehensive set of guidelines for the treatment of obesity in adults (Jensen et al., 2014). The guidelines, written as advice to providers of medical care, recommend that primary care providers measure height, weight, and waist circumference in overweight and obese adults, at least annually. Counseling to lose 3–5% of body weight is recommended for patients with a BMI >25 who are at increased risk for cardiovascular disease (i.e., those with elevated blood pressure, prediabetes, or hyperlipidemia). A comprehensive, high-intensity (i.e., >14 sessions over 6 months) lifestyle program led by trained interventionists is recommended for adults with a BMI >30 . Individuals who are unable to achieve or maintain moderate weight losses after participating in a comprehensive lifestyle intervention may be appropriate candidates for pharmacotherapy (for adults with BMI ≥ 30 or ≥ 27 with a comorbid condition) or bariatric surgery (for adults with BMI ≥ 40 or ≥ 35 with comorbid condition). In recommending treatment, clinicians are encouraged to consider an individual’s age, severity of obesity, comorbid conditions, behavioral health history, and treatment preference.

Conclusions

The current global epidemic of obesity has focused attention on the need to better understand, manage, and control this serious threat to public health. An array of cultural and environmental changes associated with economic success and increasing affluence have promoted higher food consumption and decreased energy expenditure, thereby potentiating a genetic predisposition to obesity in much of the population. In the United States, the rise in the prevalence of obesity has resulted in increasingly larger portions of healthcare expenditures devoted to the treatment of obesity and its comorbidities.

At the individual level, most people with obesity prefer to manage their weight on their own. Some subgroups, such as the NWCR participants, show dramatic success in the self-management of obesity, but generally there is scant evidence to support the effectiveness of self-help approaches for obesity management. Some commercial weight-loss programs offer resources likely to result in tangible but modest weight reductions. Among professionally delivered interventions, behavioral lifestyle interventions reliably produce weight changes of sufficient magnitude to improve health, and an array of medications are now available that may enhance the effectiveness of lifestyle treatments. For individuals with severe obesity, bariatric surgery offers the opportunity to achieve very large weight losses accompanied by significant improvements in many of the comorbidities associated with severe obesity.

Despite progress that has been made in the treatment of obesity, numerous challenges remain. For large segments of the population, access to obesity treatment is limited by governmental regulations and lack of coverage by health insurance policies. Furthermore, little research has addressed the important issues of dissemination and implementation of obesity treatment in community settings. More research is needed to deal with the problem of poor weight-loss maintenance and how an improved understanding of the interplay between biological and behavioral factors that contribute to obesity might be utilized to develop innovative and more effective treatments. Finally, greater attention needs to be given to the prevention of obesity and how governmental policies might modify the environmental factors responsible for the global rise in the prevalence of obesity.

Author Biographies

Michael G. Perri, PhD, is the Robert G. Frank Endowed Professor of Clinical and Health Psychology and dean of the University of Florida College of Public Health and Health Professions. His research focuses on the development, evaluation, and dissemination of cost-effective programs for the management of obesity. Findings from the 20+ randomized trials conducted by his team have had a significant impact on theory, research, and clinical care related to the behavioral treatment of obesity.

Melissa H. Laitner, MS, is a PhD candidate in the Department of Clinical and Health Psychology at the University of Florida where she is concurrently completing a master's degree in public health. Her research interests include behavioral treatment for obesity, health disparities, and health policy. Her dissertation research focuses on examining potential disparities between rural and urban communities in regard to obesity, diabetes, and prediabetes.

Aviva H. Ariel, MS, is a PhD candidate in the Department of Clinical and Health Psychology at the University of Florida where she is concurrently completing a master's degree in public health with a concentration in social and behavioral sciences. Her research interests include

holistic health promotion, body image disturbance, and behavioral interventions for weight management and disordered eating.

References

- Albuquerque, D., Stice, E., Rodríguez-López, R., Manco, L., & Nóbrega, C. (2015). Current review of genetics of human obesity: From molecular mechanisms to an evolutionary perspective. *Molecular Genetics and Genomics*, 290, 1191–1221.
- Arterburn, D. E., Maciejewski, M. L., & Tsevat, J. (2005). Impact of morbid obesity on medical expenditures in adults. *International Journal of Obesity*, 29, 334–339.
- Butryn, M. L., Webb, V., & Wadden, T. A. (2011). Behavioral treatment of obesity. *The Psychiatric Clinics of North America*, 34, 849–859.
- Cawley, J., & Meyerhoefer, C. (2012). The medical care costs of obesity: An instrumental variables approach. *Journal of Health Economics*, 31, 219–230.
- Colquitt, J. L., Pickett, K., Loveman, E., & Frampton, G. K. (2014). Surgery for weight loss in adults. *Cochrane Database of Systematic Reviews*, 8. Retrieved from <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD003641.pub4/epdf>
- Diabetes Prevention Program Research Group. (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine*, 346, 393–403.
- Diabetes Prevention Program Research Group. (2009). 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *The Lancet*, 374, 1677–1686.
- Federal Drug Administration. (2014, May 11). *FDA briefing information: Meeting of the Endocrinologic and Metabolic Drugs Advisory Committee*. Washington, DC: Author.
- Gadde, K. M., Allison, D. B., Ryan, D. H., Peterson, C. A., Troupin, B., Schwieters, M. L., & Day, W. W. (2011). Effects of low-dose, controlled-release, phentermine plus topiramate combination on weight and associated comorbidities in overweight and obese adults (CONQUER): A randomised, placebo-controlled, phase 3 trial. *The Lancet*, 377, 1341–1352.
- Gudzune, K. A., Doshi, R. S., Mehta, A. K., Chaudhry, Z. W., Jacobs, D. K., Vakil, R. M., ... Clark, J. M. (2015). Efficacy of commercial weight-loss programs: An updated systematic review. *Annals of Internal Medicine*, 162, 501–512.
- Hartmann-Boyce, J., Jebb, S. A., Fletcher, B. R., & Aveyard, P. (2015). Self-help for weight loss in overweight and obese adults: Systematic review and meta-analysis. *American Journal of Public Health*, 105, e43–e57.
- Helmchen, L. A., & Henderson, R. M. (2004). Changes in the distribution of body mass index of white US men, 1890–2000. *Annals of Human Biology*, 31, 174–181.
- Jensen, M. D., Ryan, D. H., Apovian, C. M., Ard, J. D., Comuzzie, A. G., Donato, K. A., ... Yanovski, S. Z. (2014). 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Journal of the American College of Cardiology*, 63, 2985–3023.
- Kodama, S., Saito, K., Tanaka, S., Horikawa, C., Fujiwara, K., Hirasawa, R., ... Sone, H. (2012). Effect of web-based lifestyle modification on weight control: A meta-analysis. *International Journal of Obesity*, 36, 675–685.
- Komlos, J., & Brabec, M. (2011). The trend of BMI values of US adults by deciles, birth cohorts 1882–1986 stratified by gender. *Economics and Human Biology*, 9, 234–250.
- Kuczmarski, R. J., Ogden, C. L., Grummer-Strawn, L. M., Flegal, K. M., Guo, S. S., Wei, R., ... Johnson, C. L. (2000). CDC growth charts: United States. *National Center for Health Statistics*. Retrieved from <http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/fullreport.htm>
- Look AHEAD Research Group. (2010). Long term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes: Four year results of the Look AHEAD trial. *Archives of Internal Medicine*, 170, 1566–1575.

- Look AHEAD Research Group. (2013). Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. *New England Journal of Medicine*, 369, 145–154.
- Look AHEAD Research Group. (2014). Eight-year weight losses with an intensive lifestyle intervention: The Look AHEAD study. *Obesity*, 22, 5–13.
- Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. S., & Marks, J. S. (2003). Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *Journal of the American Medical Association*, 289, 76–79.
- Ogden, C. L., Carroll, M. D., Fryar, C. D., & Flegal, K. M. (2015). Prevalence of obesity among adults and youth: United States, 2011–2014. *NCHS Data Brief*, 219, 1–8. Retrieved from Centers for Disease Control and Prevention website: <http://www.cdc.gov/nchs/data/databriefs/db219.htm>
- Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011–2012. *Journal of the American Medical Association*, 311, 806–814.
- Okorodudu, D. E., Bosworth, H. B., & Corsino, L. (2015). Innovative interventions to promote behavioral change in overweight or obese individuals: A review of the literature. *Annals of Medicine*, 47, 179–185.
- Perri, M. G., Limacher, M. C., Durning, P. E., Janicke, D. M., Lutes, L. D., Bobroff, L. B., ... Martin, A. D. (2008). Extended-care programs for weight management in rural communities: The treatment of obesity in underserved rural settings (TOURS) randomized trial. *Archives of Internal Medicine*, 168, 2347–2354.
- Perri, M. G., Limacher, M. C., von Castel-Roberts, K., Daniels, M. J., Durning, P. E., Janicke, D. M., ... Martin, A. D. (2014). Comparative effectiveness of three doses of weight-loss counseling: Two-year findings from the Rural LITE trial. *Obesity*, 22, 2293–2300.
- Puhl, R. M., & Heuer, C. A. (2010). Obesity stigma: Important considerations for public health. *American Journal of Public Health*, 100, 1019–1028.
- Rosenquist, J. N., Lehrer, S. F., O'Malley, A. J., Zaslavsky, A. M., Smoller, J. W., & Christakis, N. A. (2015). Cohort of birth modifies the association between FTO genotype and BMI. *Proceedings of the National Academy of Science*, 112, 354–359.
- Rucker, D., Padwal, R., Li, S. K., Curioni, C., & Lau, D. C. W. (2007). Long term pharmacotherapy for obesity and overweight: Updated meta-analysis. *British Medical Journal*, 335, 1194–1199.
- Smith, S. R., Weissman, N. J., Anderson, C. M., Sanchez, M., Chuang, E., Stubbe, S., ... Shanahan, W. R. (2010). Multicenter, placebo-controlled trial of lorcaserin for weight management. *New England Journal of Medicine*, 363, 245–256.
- Swinburn, B., Egger, G., & Raza, F. (1999). Dissecting obesogenic environments: The development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Preventive Medicine*, 29, 563–570.
- Wadden, T. A., Foreyt, J. P., Foster, G. D., Hill, J. O., Klein, S., O'Neil, P. M., ... Dunayevich, E. (2011). Weight loss with naltrexone SR/bupropion SR combination therapy as an adjunct to behavior modification: The COR-BMOD Trial. *Obesity*, 19, 110–120.
- Wadden, T. A., Webb, V. L., Moran, C. H., & Bailer, B. A. (2012). Lifestyle modification for obesity: New developments in diet, physical activity, and behavior therapy. *Circulation*, 125, 1157–1170.
- Wing, R. R., & Phelan, S. (2005). Long-term weight loss maintenance. *The American Journal of Clinical Nutrition*, 82, 222S–225S.
- World Health Organization. (2014). *Global status report on noncommunicable diseases 2014*. Retrieved from http://apps.who.int/iris/bitstream/10665/148114/1/9789241564854_eng.pdf?ua=1.

Suggested Reading

- Bray, G. A., & Bouchard, C. (Eds.) (2014). *Handbook of obesity* (4th ed.). Boca Raton, FL: CRC Press.
- Bray, G. A., & Wadden, T. A. (2015). Improving long-term weight loss maintenance: Can we do it? *Obesity*, 23, 2–3.

- Jensen, M. D., Ryan, D. H., Apovian, C. M., Ard, J. D., Comuzzie, A. G., Donato, K. A., ... Yanovski, S. Z. (2014). 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Journal of the American College of Cardiology*, 63, 2985–3023.
- U.S. Department of Health and Human Services. (2014). *Healthy people 2020: Nutrition and weight status*. Retrieved from <http://healthypeople.gov/2020/topicsobjectives2020/overview.aspx?topicid=29>

Pain and Health Psychology

Robert J. Gatchel¹ and Don McGeary²

¹*Clinical Health Psychology Research, Department of Psychology, College of Science,
The University of Texas at Arlington, Arlington, TX, USA*

²*Department of Psychiatry, Department of Family and Community Medicine,
The University of Texas Health Science Center San Antonio, San Antonio, TX, USA*

Overview

The first non-edited and comprehensive textbook in health psychology was published by Gatchel and Baum (1983). At that time, the field of health psychology was simply an array of basic and sometimes fragmented different areas (e.g., clinical psychology, cognitive psychology, experimental psychology, neuropsychology, physiological psychology, social psychology, etc.). These areas, though, began to touch upon important biobehavioral and medical content topics new to the field, such as adherence to medical regimens, promoting healthy lifestyles, rehabilitation approaches to disability, and the prevention and treatment of chronic pain, just to name a few. At about this time, when the term health psychology was still a relatively new one, a series of five edited volumes were published under the title, *Handbook of Psychology and Health* (Baum & Singer, 1982, 1987; Baum, Taylor, & Singer, 1984; Gatchel, Baum, & Singer, 1982; Krantz, Baum, & Singer, 1983). The books in this series focused on specific topic areas including pain. As subsequently noted by Baum, Revenson, and Singer (2001) in their handbook, this earlier series was published over several years just as health psychology became firmly established in its own right. There have been numerous other such handbooks published since then. Moreover, the quite diverse areas of how health psychology and medical illnesses are interrelated were perfectly aligned with the very important and influential Institute of Medicine's (IOM) report on *Living Well and Chronic Illness: A Call for Public Health Action* (Institute of Medicine, Committee on Living Well with Chronic Disease: Public Action to Reduce Disability, Improve Functioning and Quality of Life, 2012). This major IOM report was written as "... a guide for immediate and precise action to reduce the burden of all forms of chronic illnesses through the development and implementation of cross-cutting and coordinated strategies to help Americans live well..." (A-1). One such illness category was

chronic pain. Indeed, one of the most frequently cited reasons for patients in the United States to seek medical care is due to pain. Recent estimates reveal that 25.3 million American adults (11.2%) suffer from daily (chronic) pain and 23.4 million (10.3%) report “a lot of pain” (Nahin, 2015). Moreover, according to earlier statistics from the National Academy of Sciences (Gaskin & Richard, 2011), the societal costs of chronic pain and related disability in the United States alone is \$560–\$635 billion each year. Unfortunately, many chronic pain conditions cannot yet be successfully treated. More clinical research is still needed to develop better treatment methods. Thus, social, economic, and biobehavioral research have become increasingly important in medicine and in the general related area of health psychology.

The above subsequently led to another very influential IOM report, “Relieving Pain in America” (Institute of Medicine of the National Academy of Science, 2011), which highlighted the urgent need for the development of better methods for pain management because the ever-increasing costs associated with current treatment approaches cannot be sustained. This important focus has been further reinforced by National Institutes of Health’s (NIH’s) recent “National Pain Strategy: A Comprehensive Population Health-Level Strategy for Pain” (<https://www.federalregister.gov/articles/2015/04/02/2015-07626/solicitation-of-written-comments-on-draft-national-pain-strategy>). The mission of the strategy is to “Enhance pain research efforts and promote collaboration across the government, with the ultimate goals of advancing fundamental understanding of pain and improving pain-related treatment strategies.” It was also charged to develop “a comprehensive population level health strategy for pain prevention, treatment, management, education, reimbursement, and research that includes specific goals, actions, time frames, and resources” as recommended in the IOM report on “Relieving Pain in America.” The proposed strategy also emphasizes the use of the biopsychosocial model of pain, which is the underpinning of interdisciplinary pain assessment and treatment.

The result of the new area of health psychology led to the replacement of the outdated *biomedical reductionism*, or a “dualistic” perspective that the mind and body function separately and independently, to a more heuristic *biopsychosocial approach* to medicine, initially introduced by Engel (1977) and that further emerged in the 1980s. In the context of pain, the biopsychosocial model views it as the result of a dynamic interaction among biological, psychological, and social factors that perpetuate and may even worsen the clinical presentation. Gatchel and others (Gatchel, 2004, 2005; Gatchel, McGeary, McGeary, & Lippe, 2014; Gatchel & Okifuji, 2006; Gatchel, Peng, Peters, Fuchs, & Turk, 2007; Gatchel & Turk, 1999; Turk & Monarch, 2002), for example, have further advanced this health psychology/biopsychosocial perspective of pain.

The Biopsychosocial Model of Pain

As earlier reviewed by Gatchel and Okifuji (2006), traditional interventions for chronic pain had predominantly involved monotherapies, such as surgery, injections, and a wide array of pharmacotherapeutic approaches. However, as Turk and Gatchel (2002) began to highlight, a more comprehensive interdisciplinary approach was needed to address both the physical and psychosocial factors involved in chronic pain. A major outgrowth, in turn, was the development of the biopsychosocial model. As briefly discussed earlier, this model views pain as the result of a dynamic interaction among biological, psychological, and social factors that perpetuate and may even worsen the clinical presentation. Each patient experiences pain uniquely, and a range of psychological and socioeconomic factors can interact

with physical pathology to modulate that patient's report of symptoms and subsequent pain-related disability. This model has been very influential in the area of pain, especially with the resultant development of treatment- and cost-effective interdisciplinary pain management programs (Gatchel et al., 2007; Gatchel & Okifuji, 2006), to be reviewed later. Such programs, based upon the biopsychosocial model, have been found to be the most heuristic approach to understanding and assessing chronic pain (Gatchel et al., 2007). Indeed, the influential IOM report (Institute of Medicine of the National Academy of Science, 2011, p. 35) states that "Today, most researchers and clinicians who specialize in pain issues use the '**biopsychosocial model**' [denoting the combination of biological, psychological and social/family/cultural contexts of pain to understand and treat chronic pain (Gatchel et al., 2007)]."

The biopsychosocial model focuses on both disease and illness, with illness being viewed as the complex interaction among biological, psychological, and social factors (Gatchel, 2005). As succinctly summarized by Turk and Monarch (2002), *disease* is usually viewed as a pathophysiological event that results in a disruption of some anatomical/physiological structure or organ system functioning. *Illness*, on the other hand, is usually viewed as a more *subjective* experience that a specific disease is present. *Illness*, therefore, basically refers to how a "sick" individual and possibly members of his/her family accommodate and react to the resultant symptoms of possible disability and limited activities of daily living. As Turk and Monarch (2002) further point out, the distinction between disease and illness can basically be viewed as similar to the difference one can make between *nociception* and *pain*. Essentially, *nociception* refers to the stimulation of nerves that transmit signals to the brain about potential tissue damage. In contrast, *pain* is the subjective perception that results from the sensory input to the brain. Moreover, such input is usually "filtered" through a number of layers (e.g., past learning experiences, psychosocial influences, genetic predispositions, etc.). Therefore, because the biopsychosocial model of chronic pain views each individual as experiencing pain uniquely, it is important to evaluate the different dimensions of this interactive process (Gatchel, 2004). Other comprehensive reviews of the biopsychosocial model of pain can also be found elsewhere (Gatchel, 2004; Gatchel et al., 2007, 2014; Turk & Monarch, 2002).

Interdisciplinary Pain Management

The biopsychosocial model of chronic pain was one of the first models designed to accommodate the complexity of the chronic pain experience. As the model began to permeate academic and clinical medicine, so, too, did multicomponent models of pain management care. To date, the interdisciplinary pain management approach stands as one of the best and most efficacious examples of comprehensive pain management (Gatchel et al., 2014). Before exploring interdisciplinary care in greater detail, one must first be able to draw a distinction between *interdisciplinary* and *multidisciplinary* care. In an excellent review of interdisciplinary pain management models, Turk et al. (2010) carefully explained how interdisciplinary and multidisciplinary models differ. They appropriately highlight that, although the two models may both include components that match the dimensions of the biopsychosocial pain conceptualization, a truly interdisciplinary pain management program is more integrated and all treatment components are administered "under one roof" at the same pain management facility. In contrast, multidisciplinary care may be delivered by different providers at different facilities, thus decreasing integrative care and constant communication among providers

“under one roof.” Integration of pain management services is essential and can manifest in multiple ways, although most agree that coordination of care through daily team-based communication “under one roof” and common treatment goals is the key to effective integrative pain management (Gatchel et al., 2014). Interdisciplinary pain management teams will often include pain medicine physicians, nursing staff, clinical health psychology, and physical/occupational therapy providers who synthesize their care through effective leadership and commonly developed goals to create a comprehensive treatment program, with the resultant effectiveness greater than the sum of its parts (Okifuji, 2003).

A number of published studies describe the effectiveness of interdisciplinary pain management models in multiple pain populations (Gagnon, Stanos, van der Ende, Rader, & Harden, 2013; Gatchel et al., 2014). Indeed, a PubMed search for “interdisciplinary pain management” returned 50 published studies between 1995 and 2015, most of which demonstrate significant benefit of interdisciplinary pain management programs for pain of various etiologies. Despite a wealth of supportive research outcomes, interdisciplinary pain management programs are widely unavailable to many pain patients. Numerous barriers prohibit both the implementation of comprehensive pain management programs and the long-term follow-up needed to ensure that gains are maintained. First, some have noted that third-party payer reimbursement policies have played an important role in limiting the availability of more comprehensive pain management options (Gagnon et al., 2013). This is quite surprising in light of the staggering cost of chronic pain over time and the substantial socioeconomic gains that are possible through interdisciplinary care (Lippe & Polatin, 2014). Others have noted that interdisciplinary programs may not be utilized as well as they could be because many pain treatment providers prioritize simple palliative pain management (e.g., medications) over interdisciplinary options. Breivik, Eisenberg, O’Brien, and OPENMinds (2013) have noted that palliative pain relief options may be preferred because chronic pain patients and their most oft-seen medical providers (e.g., non-specialty physicians) are either not aware of more effective treatment options or are not familiar with the data supporting interdisciplinary pain management. There is some hope that increased US congressional attention to the pain management crisis, and federal policy calling more interdisciplinary healthcare options, will alleviate these concerns in the future (Gatchel et al., 2014).

In order to truly improve the availability of interdisciplinary pain programs, there needs to be some agreement on the necessary components. In a “Letter to the Editor” in the journal, *Pain*, Kaiser, Deckert, Kopkow, Schmitt, and Sabatowski (2014) pointed out that there is no broadly accepted definition of multidisciplinary or interdisciplinary pain programs (including the level of emphasis required for psychosocial intervention). This has resulted in heterogeneity in interdisciplinary pain programs under study, making it difficult to examine the state of the research meta-analytically with much refinement. Some have stressed a need to establish common outcomes across pain programs that can be used to uniformly examine the important components of pain management (i.e., key ingredients) for translation (Deckert et al., 2015). In fact, the NIH have developed the *Patient-Reported Outcomes Measurement Information System* (PROMIS) (<http://www.nihpromis.org/>) in order to encourage all pain practitioners to use the set of outcome measures. Regardless, most who study interdisciplinary pain management seem to agree that psychosocial intervention is vital if a pain management program wants to holistically address the biopsychosocial model (Gatchel et al., 2014). Numerous studies have uncovered psychosocial constructs of pain (e.g., pain catastrophizing, pain acceptance, fear avoidance, etc.) that contribute to the pain management process (Vowles, McCracken, & Eccleston, 2007), and subsequent studies have found that these concepts may mediate the relationship between pain experience and mood (Weiss et al., 2013). In light of these findings,

interdisciplinary pain programs must appreciate the value added by the clinical health psychologist in order for the treatment team to be successful.

Functional Restoration Programs

Despite the heterogeneity of interdisciplinary pain programs, there are a few models under study with more definite composition and a more definite role of clinical health psychology. Of these, functional restoration (FR) programs represent some of the most well researched, with a strong emphasis on return to function and the pursuit of long-term socioeconomic goals (e.g., reduced medical visits for pain management, increased employment, increased retention of employment). The mechanics of FR pain and disability management typically include an emphasis on both physical capacity and psychosocial functioning (Mayer, McGeary, & Gatchel, 2004). Health psychology is a prominent component of FR. In fact, the recommended FR treatment team should always include a psychologist, along with a pain management physician, nurse, and physical or occupational therapist. These providers must work in an interdisciplinary fashion to quantify function across all relevant rehabilitation domains (e.g., functional capacity, mood, pain coping) and to develop an integrated treatment program that targets these domains synergistically (Gatchel & Okifuji, 2006). Treatment begins with a comprehensive assessment battery (i.e., quantify function), and the resulting data are used to identify treatment targets. As an interdisciplinary program, FR's goals are determined collaboratively across all providers and are agreed upon by patients who are given regular feedback about their assessment and shown evidence of physical *and* psychosocial progress over the course of the intensive FR program. Again, the clinical health psychologist plays a key role in the FR treatment process. For example, the psychologist helps mitigate emotional disruption (e.g., depression) that may affect physical function and/or engagement in rehabilitation with other FR team members (Heiskanen, Roine, & Kalso, 2012) and helps the patient learn how to effectively track goals and communicate with other healthcare providers to ensure that their clinical needs are met and that they are progressing toward their goals. They also teach the patient effective pain management strategies that can be used to overcome self-limiting functional activity, which helps maximize the benefits of physical rehabilitation (Gatchel et al., 2014).

Over the past 30 years, numerous studies have confirmed the efficacy and effectiveness of FR pain management models, often showing significant gains beyond those demonstrated in one-dimensional programs of physical/physiotherapy or biomedical care (Mayer et al., 1987; Steiner et al., 2013). As studies of FR have progressed, research surrounding these interdisciplinary programs has started to expand, with increasing emphasis on the effectiveness of FR in managing orthopedic/musculoskeletal pain and common comorbidities (including sleep, non-orthopedic pain conditions, and psychiatric disorders) that typically present with chronic pain. For example, Hartzell et al. (2014) found that patients with chronic musculoskeletal pain and comorbid fibromyalgia show a significant improvement in fibromyalgia symptoms after completing an FR program (e.g., 41% of completers no longer met criteria for fibromyalgia after completing treatment). Asih, Neblett, Mayer, and Gatchel (2014) found a significant improvement in insomnia among FR program completers, although the presence of insomnia was a predictor of program completion, and more severe insomnia symptoms persisted beyond treatment. As noted above, interdisciplinary pain management programs (including FR) can contribute to significant reductions in some emotional distress conditions (e.g., depression), but there are fewer data supporting the application of FR to more complex psychosocial

presentations like posttraumatic stress disorder (PTSD) (McGeary, Moore, Vriend, Peterson, & Gatchel, 2011). This is particularly relevant because of the increasing attention paid to PTSD and pain-related comorbidity among military members and veterans who served as part of the post-9/11 war effort.

Chronic pain and PTSD have been found to co-occur quite frequently in some military populations. Specifically, Lew et al. (2009) report that over 58% of veterans assessed for trauma and pain in a VA Polytrauma Network Site presented with comorbid pain and PTSD and the presence of PTSD in the context of pain can have significant and deleterious effects on response to pain management intervention (McGeary et al., 2011). Little has been done to examine treatments for pain and PTSD comorbidity, but there is solid evidence suggesting that FR programs are generally effective for military service members with chronic pain (Gatchel et al., 2009), and some psychosocial interventions work quite well for military PTSD (McLean & Foa, 2014). Thus, it stands to reason that a comprehensive interdisciplinary pain management program (like FR) could incorporate effective psychosocial treatment components for PTSD to develop an efficacious treatment option for this significant problem. Large-scale studies of interdisciplinary pain and PTSD management are underway, but some smaller studies have already started to show the benefits of combined treatments for this comorbidity (Plagge, Lu, Lovejoy, Karl, & Dobscha, 2013).

Conclusions

The field of health psychology has grown significantly over the past several decades, and few subspecialty fields have benefitted more from this growth than chronic pain. As the field of healthcare providers who work with chronic pain increasingly adopts and expands the biopsychosocial model, greater integration of treatment will likely follow. Indeed, recent changes in legislation and the growing body of evidence supporting integrated treatment for pain bode well for the growth of interdisciplinary models. Research on psychosocial variables in the pain experience and pain management has rapidly expanded over the past 30 years, and health psychology is now widely acknowledged as a vital component of interdisciplinary pain management teams. This growth is likely to continue as pain management intervention and research start to apply the biopsychosocial model to even more complex chronic pain presentations (e.g., comorbid pain and PTSD).

Author Biographies

Dr. Robert J. Gatchel, PhD, ABPP, is the Nancy P. & John G. Penson endowed professor of clinical health psychology at the University of Texas at Arlington. He is internationally known for his research on biopsychosocial pain management, and his scientific contributions have resulted in numerous awards including an NIH Senior Scientist Award and the Gold Medal Award for Life Achievement in the Application of Psychology from the American Psychological Association.

Dr. Don McGeary, PhD, ABPP, is an associate professor in the Department of Psychiatry and a ReACH scholar at the University of Texas Health Science Center at San Antonio. He is the principle investigator of several federally funded studies of chronic pain and psychiatric comorbidities with a particular emphasis on managing pain-related polymorbidities in military service members and veterans.

References

- Asih, S., Neblett, R., Mayer, T. G., & Gatchel, R. J. (2014). Does patient-reported insomnia improve in response to interdisciplinary functional restoration for chronic disabling occupational musculoskeletal disorders? *Spine*, 39(17), 1384–1392.
- Baum, A., Revenson, T. A., & Singer, J. A. (2001). *Handbook of health psychology*. Mahway, NJ: Lawrence Erlbaum Associates.
- Baum, A., & Singer, J. E. (Eds.) (1982). *Issues in child health and adolescent health* (Vol. 2). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Baum, A., & Singer, J. E. (Eds.) (1987). *Stress* (Vol. 5). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Baum, A., Taylor, S. E., & Singer, J. E. (Eds.) (1984). *Psychological aspects of health* (Vol. 4). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Breivik, H., Eisenberg, E., O'Brien, T., & OPENMinds. (2013). The individual and societal burden of chronic pain in Europe: The case for strategic prioritisation and action to improve knowledge and availability of appropriate care. *BMC Public Health*, 13, 1229.
- Deckert, S., Kaiser, U., Kopkow, C., Trautmann, F., Sabatowski, R., & Schmitt, J. (2015). A systematic review of the outcomes reported in multimodal pain therapy for chronic pain. *European Journal of Pain*, 20(1), 51–63.
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196(4286), 129–136.
- Gagnon, C. M., Stanos, S. P., van der Ende, G., Rader, L. R., & Harden, R. N. (2013). Treatment outcomes for workers compensation patients in a US-based interdisciplinary pain management program. *Pain Practice*, 13(4), 282–288.
- Gaskin, D. J., & Richard, P. (2011). The economic costs of pain in the United States. *Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research*. Retrieved from <http://www.ncbi.nlm.nih.gov/books/NBK92521/>
- Gatchel, R. J. (2004). Comorbidity of chronic mental and physical health disorders: The biopsychosocial perspective. *American Psychologist*, 59, 792–805.
- Gatchel, R. J. (2005). *Clinical essentials of pain management*. Washington, DC: American Psychological Association.
- Gatchel, R. J., & Baum, A. (1983). *An introduction to health psychology*. Reading, MA: Addison-Wesley.
- Gatchel, R. J., Baum, A., & Singer, J. E. (Eds.) (1982). *Clinical psychology and behavioral medicine: Overlapping disciplines* (Vol. 1). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Gatchel, R. J., McGeary, D. D., McGeary, C. A., & Lippe, B. (2014). Interdisciplinary chronic pain management: Past, present and the future. *American Psychologist, Special Issue on "Psychology and Chronic Pain"*, 69(2), 119–130.
- Gatchel, R. J., McGeary, D. D., Peterson, A. L., Moore, M. E., LeRoy, K., Isler, W. C., ... Edell, T. (2009). Preliminary findings of a randomized controlled trial of an interdisciplinary functional restoration program. *Military Medicine*, 174, 270–277.
- Gatchel, R. J., & Okifuji, A. (2006). Evidence-based scientific data documenting the treatment and cost-effectiveness of comprehensive pain programs for chronic nonmalignant pain. *The Journal of Pain*, 7(11), 779–793.
- Gatchel, R. J., Peng, Y., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, 133, 581–624.
- Gatchel, R. J., & Turk, D. C. (Eds.) (1999). *Psychological factors in pain: Critical perspectives*. New York, NY: Guilford.
- Hartzell, M. M., Neblett, R., Perez, Y., Brede, E., Mayer, T. G., & Gatchel, R. J. (2014). Do comorbid fibromyalgia diagnoses change after a functional restoration program in patients with chronic disabling occupational musculoskeletal disorders? *Spine*, 39(17), 1393–1400.
- Heiskanen, T., Roine, R. P., & Kalso, E. (2012). Multidisciplinary pain treatment—Which patients do benefit? *Scandinavian Journal of Pain*, 3(4), 201–207.

- Institute of Medicine, Committee on Living Well with Chronic Disease: Public Action to Reduce Disability, Improve Functioning and Quality of Life. (2012). *Living well with chronic illness: A call for public health action*. Washington, DC: The National Academies Press. Retrieved from http://www.nap.edu/catalog.php?record_id=13272
- Institute of Medicine of the National Academy of Science. (2011). *Relieving pain in America: A blueprint for transforming prevention, care, education, and research* (pp. 5–5). Washington, DC: Institute of Medicine.
- Kaiser, U., Deckert, S., Kopkow, C., Schmitt, J., & Sabatowski, R. (2014). Letter to the editor: Dose or content? Effectiveness of pain rehabilitation programs for patients with chronic low back pain: A systematic review. *Pain, 155*(9), 1903–1904.
- Krantz, D. S., Baum, A., & Singer, J. E. (Eds.) (1983). *Cardiovascular disorders and behavior* (Vol. 3). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Lew, H. L., Otis, J. D., Tun, C., Kerns, R. D., Clark, M. E., & Cifu, D. X. (2009). Prevalence of chronic pain, posttraumatic stress disorder, and persistent postconcussive symptoms in OIF/OEF veterans: Polytrauma clinical triad 2026 Research to improve the lives of veterans: Approaches to traumatic brain injury; screening, treatment, management, and rehabilitation in Arlington, Virginia, April 30 to May 2, 2008. *Journal of Rehabilitation Research and Development, 46*(6), 697–702.
- Lippe, B., & Polatin, P. (2014). The interdisciplinary treatment approach for chronic pain management: The key components for success. In R. J. Gatchel & I. Z. Schultz (Eds.), *Handbook of musculoskeletal pain and disability disorders in the workplace*. New York, NY: Springer.
- Mayer, T. G., Gatchel, R. J., Mayer, H., Kishino, N. D., Keeley, J., & Mooney, V. (1987). A prospective two-year study of functional restoration in industrial low back injury: An objective assessment procedure. *JAMA, 258*(13), 1763–1767.
- Mayer, T. G., McGeary, D., & Gatchel, R. J. (2004). Chronic pain management through functional restoration for spinal disorders. In J. F. Frymoyer & S. Wiesel (Eds.), *Adult and pediatric spine* (3rd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- McGeary, D., Moore, M., Vriend, C. A., Peterson, A. L., & Gatchel, R. J. (2011). The evaluation and treatment of comorbid pain and PTSD in a military setting: An overview. *Journal of Clinical Psychology in Military Settings, 18*, 155–163.
- McLean, C. P., & Foa, E. B. (2014). The use of prolonged exposure therapy to help patients with post-traumatic stress disorder. *Clinical Practice, 11*(2), 233–241.
- Nahin, R. L. (2015). Estimates of pain prevalence and severity in adults: United States, 2012. *The Journal of Pain, 16*(8), 769–780. <http://dx.doi.org/10.1016/j.jpain.2015.05.002>
- Okifuji, A. (2003). Interdisciplinary pain management with pain patients: Evidence for its effectiveness. *Seminars in Pain Management, 1*(2), 110–119.
- Plagge, J. M., Lu, M. W., Lovejoy, T. I., Karl, A. I., & Dobscha, S. K. (2013). Treatment of comorbid pain and PTSD in returning veterans: A collaborative approach utilizing behavioral activation. *Pain Medicine, 14*(8), 1164–1172.
- Steiner, A. S., Sartori, M., Leal, S., Kupper, D., Gallice, J. P., Rentsch, D., ... Genevay, S. (2013). Added value of an intensive multidisciplinary functional rehabilitation programme for chronic low back pain patients. *Swiss Medical Weekly, 143*, w13763.
- Turk, D. C., & Gatchel, R. J. (Eds.) (2002). *Psychological approaches to pain management: A practitioner's handbook* (2nd ed.). New York, NY: Guilford.
- Turk, D. C., & Monarch, E. S. (2002). Biopsychosocial approaches on chronic pain. In R. J. Gatchel & D. C. Turk (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (pp. 3–29). New York, NY: Guilford Press.
- Turk, D. C., Stanos, S. P., Palermo, T. M., Paice, J. A., Jamison, R. N., Gordon, D. B., & Clark, M. E. (2010). *Interdisciplinary pain management*. Glenview, IL: American Pain Society.
- Vowles, K. E., McCracken, L. M., & Eccleston, C. (2007). Processes of change in treatment for chronic pain: The contributions of pain, acceptance, and catastrophizing. *European Journal of Pain, 11*(7), 779–787.

Weiss, K. E., Hahn, A., Wallace, D. P., Biggs, B., Bruce, B. K., & Harrison, T. E. (2013). Acceptance of pain: Associations with depression, catastrophizing, and functional disability among children and adolescents in an interdisciplinary chronic pain rehabilitation program. *Journal of Pediatric Psychology*, 38(7), 756–765.

Suggested Reading

Okifuji, A. (2003). Interdisciplinary pain management with pain patients: Evidence for its effectiveness. *Seminars in Pain Medicine*, 1(2), 110–119.

Turk, D. C., & Gatchel, R. J. (2018). *Psychological approaches to pain management: A practitioner's handbook*. New York, NY: Guilford Press.

Perinatal Depression

Rachel Vanderkruik, Elizabeth Lemon, Laura River,
and Sona Dimidjian

Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA

The transition to motherhood can be both a risky and rewarding time for women physically, interpersonally, and psychologically. Nearly 20% of mothers worldwide are affected by depression during pregnancy or the postpartum (i.e., the “perinatal” time period) (Gavin et al., 2005). Perinatal depression represents a significant public health issue for women and families. Here, we will review the conceptualization and classification, correlates and consequences, risk and contributing factors, and detection and management of depression during pregnancy and the postpartum.

Conceptualization and Classification

There is considerable variability in the severity and duration of symptoms of depression that may be experienced during the perinatal period. For example, postpartum blues, or “baby blues,” are fleeting feelings of depression and anxiety that many new mothers experience within the first 10 days after giving birth. In contrast, the term “depression” is often used in clinical contexts to refer to an episode of major depression. The diagnostic criteria for major depression are the same during pregnancy and the postpartum as in other times of life; thus, it is important to underscore that terms such as “perinatal depression” do not indicate a distinct clinical disorder but rather the occurrence of depression during this specific life phase. Major depression is indicated by the presence of at least 2 weeks of depressed mood or loss of interest or pleasure in activities, for most of the day, nearly every day, accompanied by at least four additional symptoms (i.e., sleep disruption, change in appetite, psychomotor agitation or retardation, fatigue, feelings of worthlessness or guilt, problems with concentration or decision making, or thoughts of death or suicide). Further, these symptoms must cause functional impairment.

Depression during the perinatal period is captured in the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition, as a specifier of major depressive disorder. To receive a diagnosis of major depressive disorder with “peripartum onset,” a woman must meet all of the criteria for a major depressive episode, and the onset of depressive symptoms must have occurred during pregnancy or within 4 weeks after delivery. There is inconsistency within the field, however, about this time restriction, with many defining postpartum depression as onset up to 12 months following delivery.

A recent review of 16 longitudinal studies found that the average rate of antenatal depression is 17% and the average rate of postpartum depression is 13% (Underwood, Waldie, D’Souza, Peterson, & Morton, 2016). The point prevalence of antenatal depression is 11% in the first trimester, and 9% in the rest of pregnancy, using DSM criteria for diagnosis (Gavin et al., 2005). Postpartum depression has a period prevalence of up to 19.2% of women within 3 months of delivery; the point prevalence of postpartum depression has been recorded as highest in the third month postpartum (13%), followed by a point prevalence of about 10% during the fourth through seventh months postpartum, and 7% thereafter, also using DSM criteria (Gavin et al., 2005).

There is significant comorbidity between postpartum depression and anxiety. Women who have a history of postpartum depression, or who are experiencing current symptoms of postpartum depression, are also at risk for the onset of mania/hypomania (bipolar disorder), although this has been understudied. While rare, symptoms of psychosis may co-occur with perinatal depression, particularly in the postpartum, potentially increasing the severity of the disorder.

Correlates and Consequences of Depression During Pregnancy and the Postpartum

Perinatal depression is associated with increased risk of negative correlates and consequences for the overall health, well-being, and developmental trajectories of both mother and offspring, as well as a broader impact on society.

Mothers

Depression during pregnancy has been associated with maternal physical health problems, compromised immune function, and preeclampsia (Field, 2011). Postpartum depression is linked to increased risk of factors that interfere with parenting, including persistent negative affect, increased self-focus, and negative perceptions of oneself and others (O’Hara & McCabe, 2013). Many women who experience depression in pregnancy also experience postpartum depression. Perinatal depression also poses risk for future episodes of depression (O’Hara & McCabe, 2013).

Offspring

Depression during pregnancy is associated with an increased risk of preterm delivery, low birth weight, reduced breastfeeding initiation rate, heightened infant distress and sleep disturbance, and emotional and behavioral problems in childhood (Field, 2011). Depression during the postpartum has been associated with heightened infant distress and sleep problems, as well as compromised infant self-regulation, stress, and arousal, and poor cognitive

functioning and emotional and behavioral disorders in infancy and childhood (e.g., O'Hara & McCabe, 2013).

Society

Beyond the risks to individual women and their families, depression presents challenges from public health and economic perspectives. Lifetime costs of perinatal depression have been estimated at 23,151 British pounds for mothers (US\$30,805.56 as of time of writing) and 52,577 British pounds for children (US\$70,808.07), based on costs of healthcare, as well as reduced productivity and quality of life (Bauer, Knapp, & Parsonage, 2016).

Risk and Protective Factors

Demographic Factors

We lack a clear and consistent picture of the demographic factors associated with depression during pregnancy and postpartum. Younger maternal age has been associated with increased depressive symptoms during pregnancy and postpartum, as has older maternal age during pregnancy (e.g., Biaggi, Conroy, Pawlby, & Pariante, 2016); however, this association has not been consistently reported in all of the studies included in these reviews. Similarly, belonging to a minority race/ethnicity may be associated with increased rates of depression during pregnancy (Biaggi et al., 2016), but the association was not consistent across all studies included in this review. Few studies have examined the association of race/ethnicity with depression in the postpartum period, although it does appear that there are racial/ethnic disparities in rates of postpartum depression (Liu & Tronick, 2013).

Socioeconomic status (SES) is likely an important factor. There is some evidence that low SES has a small effect on the development of postpartum depression, yet varying criteria used to define "low income" contribute to inconsistent evidence of this relationship. Several reviews of demographic risk factors for antenatal depression have identified unemployment, low income, and low educational attainment to be associated with depression during pregnancy; however these associations were not found in all studies reviewed (Biaggi et al., 2016; Lancaster et al., 2010). Having Medicaid or other public insurance versus private insurance is more consistently associated with increased likelihood of antenatal depressive symptoms (Lancaster et al., 2010). Additionally, there is some evidence that women who live in rural areas may face heightened risk for postpartum depression, perhaps because they have less access to care.

Biological and Health Factors

There has been growing interest in identifying the pathophysiology of depression during pregnancy and postpartum. Much of the work to date has examined the roles of stress and reproductive hormones as risk and protective factors. The evidence for the role of cortisol in the development of perinatal depression is mixed; however, most studies support an association between elevated cortisol and brief mood fluctuations during pregnancy and postpartum "blues"; in contrast, blunted cortisol is likely related to postpartum depression, particularly when it is more chronic (Yim, Tanner Stapleton, Guardino, Hahn-Holbrook, & Schetter, 2015). Some data suggest that oxytocin may be an important player in the pathophysiology of postpartum depression, with evidence of an inverse relationship between oxytocin and

depression severity (Yim et al., 2015). Several studies have found heightened sensitivity to estrogen fluctuations in perinatal depression (Yim et al., 2015). Moreover, genetic factors appear to influence depression differently depending on the point in time in the perinatal period that the symptoms emerge (Yim et al., 2015). Understanding the contribution of hormonal factors to the timing and course of depression during pregnancy and the postpartum will be an important direction for future research.

Recent research has begun to explore how the development timing of perinatal depression influences child outcomes, as well as how mechanisms of maternal depression may shape the somatic and behavioral health of the child. Such attention to the sensitive periods of when a woman may be at greatest risk for depression, hormonal factors, or exposure effects of perinatal depression on the offspring has implications for design and administration of interventions. Emerging research is using magnetic resonance imaging to examine the neurobiological profile of postpartum depression, which appears to be similar to major depression in the general population, and is also examining the relationship between inflammation and perinatal depression as a promising direction for future research.

Finally, several health-related behaviors may also influence risk for perinatal depression. Physical activity during and after pregnancy may be associated with a reduced risk for postnatal depression, yet more research is needed to identify an optimal dose of physical activity for such risk reduction. The evidence relevant to breastfeeding as a potential risk factor has been equivocal: some data suggest that not breastfeeding is associated with postpartum depression; however, others suggest that this association may be due to other risk factors such as income, stress, and social support. Additionally, postpartum depressive symptoms have been positively associated with unhealthful behaviors such as smoking and poor maternal diet. There is limited evidence to suggest an association between postpartum depression and deficiencies in biotin, homocysteine, zinc, and vitamin B₁₂; more robust evidence supports an association between low vitamin D levels during pregnancy and increased risk for postpartum depression, and there is insufficient evidence to support a link between postpartum depression and levels of maternal iron, selenium, or omega-3 fatty acids (DHA and EPA) (e.g., Serati, Redaelli, Buoli, & Altamura, 2016). Finally, sleep disturbance during the postpartum period may be associated with postpartum depression, although the direction of the relationship is unclear.

Psychosocial Factors

There is strong evidence supporting an association between stressful life events (e.g., losing a job, moving) and elevated symptoms of depression during pregnancy (Lancaster et al., 2010). Support for an association between experiencing stressful life events during the perinatal period and depression in the postpartum has been mixed, with some studies finding a weak association (Yim et al., 2015) and others finding that the experience of multiple stressful life events from the start of pregnancy to shortly after childbirth is associated with depressive symptom severity and increased risk for postpartum depression (Lancaster et al., 2010). A more consistent association has been identified between chronic stressors and postpartum depression. Parenting stress, and in particular caring for difficult infants (e.g., because of temperament, crying, and colic), is consistently associated with elevated levels of depression in the postpartum period, as are general life stress, perceived stress, and feelings of overwhelm (Yim et al., 2015). However, daily stressors are not consistently associated with depression during pregnancy (Lancaster et al., 2010).

Social support has been identified as a key factor in protecting against depression among pregnant and postpartum women. Women with poor social support may be at greater risk for

antenatal depressive symptoms or postpartum depression (Lancaster et al., 2010). Lack of intimate partner support may predict depression during pregnancy, particularly in the context of intimate partner violence, whereas support from an intimate partner and overall relationship satisfaction are associated with lower levels of perinatal depression (e.g., Pilkington, Milne, Cairns, Lewis, & Whelan, 2015). Specific partner factors including emotional closeness, low levels of conflict, effective communication, and sufficient emotional and instrumental support are associated with lower depression during pregnancy and the postpartum (Pilkington et al., 2015). Non-cohabitation with an intimate partner was associated with increased risk of depressive symptoms during pregnancy (Lancaster et al., 2010), but not all findings included in this review were consistent. There also appears to be a relationship between an unwanted or unplanned pregnancy and postpartum depression, and the prevalence of depression among women with unintended pregnancies may be as much as two times higher (e.g., Abajobir, Maravilla, Alati, & Najman, 2016).

Clinical History and Comorbidity

A family history of depression and having experienced a previous episode of depression, especially a past perinatal depressive episode, are predictors of perinatal depression. Experiencing anxiety before or during pregnancy increases the risk of developing depression both during pregnancy and postpartum. Experiencing depression during pregnancy also substantially increases the risk of developing postpartum depression (Biaggi et al., 2016; O'Hara & McCabe, 2013; Underwood et al., 2016). A history of childhood sexual abuse is associated with perinatal depression, although the association is more consistently seen during pregnancy than postpartum (Wosu, Gelaye, & Williams, 2015). Substance abuse during pregnancy and the postpartum is also associated with increased rates of depression, although the direction of the relationship is unclear (e.g., Forray, Gotman, Kershaw, & Yonkers, 2014). Body image and eating concerns also may increase risk for perinatal depression. A recent review reported a consistent but weak association between body image dissatisfaction and the onset of perinatal depression (Silveira, Ertel, Dole, & Chasan-Taber, 2015). A history of eating disorders, particularly bulimia nervosa and binge eating disorder, increases risk for perinatal depression.

Detection and Management

Screening and Engagement

Recently, there has been increasing attention to the importance of screening for depression during pregnancy and the postpartum period. American Congress of Obstetricians and Gynecologists (ACOG)'s Committee on Obstetric Practice recommends that physicians screen women for depression and anxiety symptoms at least once during the perinatal period with a validated tool. ACOG also stated that the screening should include appropriate follow-up procedures, including the provision of therapy or a referral to receive treatment. Similarly, the US Preventive Services Task Force (USPSTF) recommended that all adults should be screened for depression, including at-risk populations of pregnant and postpartum women, again highlighting a call for adequate systems to ensure appropriate follow-up and treatment (Siu et al., 2016). It has been recommended that practitioners take advantage of women's routine antenatal and infant checkups to screen for elevated depressive symptom severity.

Recently, a systematic review was conducted for the USPSTF surrounding the effects of depression screening and treatment for pregnant and postpartum women (O'Connor, Rossom, Henninger, Groom, & Burda, 2016). Based on six trials, the authors reported an 18–59% relative reduction in the risk of depression at follow-up after participation in programs involving depression screening, with or without additional treatment components, compared to usual care. These data suggest that screening pregnant and postpartum women for depression may reduce depressive symptoms.

There are several instruments that have been validated for use during pregnancy and the postpartum period to screen for depression. A commonly used instrument is the Edinburgh Postnatal Depression Scale (EPDS), which consists of 10 self-reported items, takes less than 5 min to complete, has been translated into 12 languages, requires a low reading level, and is easy to score. The Patient Health Questionnaire 9 (PHQ-9) is another brief screening tool often used for perinatal depression.

Research has explored common barriers to treatment-seeking behaviors for perinatal women. Women are more likely to prefer non-pharmacologic (i.e., psychotherapeutic) treatments for depression, rather than antidepressant medication (Battle, Salisbury, Schofield, & Ortiz-Hernandez, 2013). Additionally, research has found that depressed perinatal women often experience a high level of decisional conflict, or uncertainty, as to how to treat their depression. Such decisional conflict is associated with a lower likelihood to engage in treatment (Battle et al., 2013). There have been recent efforts to explore models of screening, support for treatment decision making, and therapeutic care that increase the likelihood that women who may benefit from mental health support will be screened and receive treatment (e.g., medical home models, integration into primary care, engaging patients in treatment decision making, including mental health providers in OB-GYN clinics).

Prevention and Treatment

Services are available that can help to prevent perinatal depression. There is evidence that interventions based in cognitive behavioral therapy (CBT), mindfulness-based cognitive therapy (MBCT), and interpersonal therapy (IPT) are effective in reducing the risk of depression during the perinatal period. Some women with an elevated risk for perinatal depression (i.e., women with a history of depression) may benefit from prophylactic treatment with a selective serotonin reuptake inhibitor (SSRI) antidepressant to prevent perinatal depression.

Both pharmacological and non-pharmacological interventions are effective for treating depressed pregnant and postpartum women. Recent guidelines published by the American Psychiatric Association (APA) and ACOG provide instructions for mental healthcare providers regarding appropriate treatment with pharmacotherapy as well as considerations for safety with respect to the use of pharmacotherapy during pregnancy (Yonkers et al., 2009). A growing body of research describes the efficacy and safety of antidepressant medication for depression in pregnancy; however significant gaps still remain. There may be small increases in the risk of preeclampsia, postpartum hemorrhage, or miscarriage, as well as adverse infant outcomes (e.g., preterm birth, pulmonary hypertension, small for gestational age), associated with second-generation antidepressant use during pregnancy (O'Connor et al., 2016).

Recent guidelines also indicate that some forms of psychotherapy should be considered as a treatment, either in place of, or to augment, pharmacotherapy. CBT, IPT, and behavioral activation (BA) therapy demonstrate promise in ameliorating perinatal depressive symptoms. In their review, O'Connor et al. (2016) pooled results for the benefit of CBT for pregnant and postpartum women with screen-detected depression and found an increase in the likelihood of

remission compared with usual care. A variety of promising delivery methods for treating perinatal depression exist, including Internet-based therapy and peer-delivered psychological interventions in addition to delivery by mental health professionals.

Some work has examined complementary and alternative medicine (CAM) interventions for perinatal depression. Numerous CAM treatments have been studied, including omega-3 fatty acid, folate, and St. John's wort supplements, exercise, and bright light therapy, as either monotherapies or adjunctive treatment for women during the perinatal period (for a review see Deligiannidis & Freeman, 2014). Further study is needed to examine the efficacy and safety of these CAM treatments for perinatal depression.

Summary

It is not uncommon for a woman to experience depression during pregnancy or the postpartum, particularly in the presence of risk factors such as a history of depression, low social support, or other stressors in her life. The consequences of untreated perinatal depression are substantial for both mothers and offspring, as well as for society as a whole. Healthcare providers should consider the identified risk and protective factors for women during the perinatal time period to assess the level of a woman's risk for experiencing perinatal depression and current severity of symptoms or disorder. There has been a strong focus on efforts to screen perinatal women for depression during the perinatal period, yet additional work remains to ensure that women receive the follow-up support and care that may benefit them after they are screened. There are effective interventions available through a variety of delivery methods for both the prevention and treatment of perinatal depression. Consideration for perinatal mental health should be a top priority in order to promote women's health, as well as the healthy development of children born to women with depression.

Author Biographies

Rachel Vanderkruijk is a doctoral student in the clinical psychology program at the University of Colorado Boulder. She received a BA in biology from Bowdoin College and an MSc from Harvard T. H. Chan School of Public Health. Her research interests focus on increasing access to mental health services for women during the perinatal period and disseminating evidenced-based interventions for mental and behavioral health issues.

Elizabeth Lemon is a doctoral student in the clinical psychology program at the University of Colorado Boulder. She received her BA in psychology from Humboldt State University, California, and her MA in psychology from Boston University. Elizabeth's research interests focus on mood and anxiety disorders during pregnancy and the postpartum period, as well as developing new ways to access and deliver clinical care to decrease barriers to mental health services.

Laura River is currently a doctoral student in the child clinical psychology program at the University of Denver. She received her BA in psychology from Pomona College. Her research interests center on parent-child and romantic attachment during the transition to parenthood, as well as interventions promoting parent and infant well-being.

Dr. Sona Dimidjian is an associate professor in the Department of Psychology and Neuroscience at the University of Colorado Boulder. Her research focuses on cultivating enduring well-being for women and children. She is a leading expert in the treatment and

prevention of depression, focusing on the mental health of women during pregnancy and postpartum. She received her BA in psychology from the University of Chicago and her PhD in clinical psychology from the University of Washington.

References

- Abajobir, A. A., Maravilla, J. C., Alati, R., & Najman, J. M. (2016). A systematic review and meta-analysis of the association between unintended pregnancy and perinatal depression. *Journal of Affective Disorders*, 192, 56–63.
- Battle, C. L., Salisbury, A. L., Schofield, C. A., & Ortiz-Hernandez, S. (2013). Perinatal antidepressant use: Understanding women's preferences and concerns. *Journal of Psychiatric Practice*, 19(6), 443–453.
- Bauer, A., Knapp, M., & Parsonage, M. (2016). Lifetime costs of perinatal anxiety and depression. *Journal of Affective Disorders*, 192, 83–90.
- Biaggi, A., Conroy, S., Pawlby, S., & Pariante, C. M. (2016). Identifying the women at risk of antenatal anxiety and depression: A systematic review. *Journal of Affective Disorders*, 191, 62–77.
- Deligiannidis, K. M., & Freeman, M. P. (2014). Complementary and alternative medicine therapies for perinatal depression. *Best Practice & Research. Clinical Obstetrics & Gynaecology*, 28(1), 85–95.
- Field, T. (2011). Prenatal depression effects on early development: A review. *Infant Behavior & Development*, 34(1), 1–14.
- Forray, A., Gotman, N., Kershaw, T., & Yonkers, K. A. (2014). Perinatal smoking and depression in women with concurrent substance use. *Addictive Behaviors*, 39(4), 749–756.
- Gavin, N. I., Gaynes, B. N., Lohr, K. N., Meltzer-Brody, S., Gartlehner, G., & Swinson, T. (2005). Perinatal depression: A systematic review of prevalence and incidence. *Obstetrics & Gynecology*, 106(5), 1071–1083.
- Lancaster, C. A., Gold, K. J., Flynn, H. A., Yoo, H., Marcus, S. M., & Davis, M. M. (2010). Risk factors for depressive symptoms during pregnancy: A systematic review. *American Journal of Obstetrics and Gynecology*, 202(1), 5–14.
- Liu, C. H., & Tronick, E. (2013). Rates and predictors of postpartum depression by race and ethnicity: Results from the 2004 to 2007 New York City PRAMS survey (Pregnancy Risk Assessment Monitoring System). *Maternal and Child Health Journal*, 17(9), 1599–1610.
- O'Connor, E., Rossom, R. C., Henninger, M., Groom, H. C., & Burda, B. U. (2016). Primary care screening for and treatment of depression in pregnant and postpartum women: Evidence report and systematic review for the US Preventive Services Task Force. *JAMA*, 315(4), 388–406.
- O'Hara, M. W., & McCabe, J. E. (2013). Postpartum depression: Current status and future directions. *Annual Review of Clinical Psychology*, 9, 379–407.
- Pilkington, P. D., Milne, L. C., Cairns, K. E., Lewis, J., & Whelan, T. A. (2015). Modifiable partner factors associated with perinatal depression and anxiety: A systematic review and meta-analysis. *Journal of Affective Disorders*, 178, 165–180.
- Serati, M., Redaelli, M., Buoli, M., & Altamura, A. C. (2016). Perinatal major depression biomarkers: A systematic review. *Journal of Affective Disorders*, 193, 391–404.
- Silveira, M. L., Ertel, K. A., Dole, N., & Chasan-Taber, L. (2015). The role of body image in prenatal and postpartum depression: A critical review of the literature. *Archives of Women's Mental Health*, 18(3), 409–421.
- Siu, A. L., Bibbins-Domingo, K., Grossman, D. C., Baumann, L. C., Davidson, K. W., Ebell, M., ... Pignone, M. P. (2016). Screening for depression in adults: US Preventive Services Task Force recommendation statement. *JAMA*, 315(4), 830–837.
- Underwood, L., Waldie, K., D'Souza, S., Peterson, E. R., & Morton, S. (2016). A review of longitudinal studies on antenatal and postnatal depression. *Archives of Women's Mental Health*, 19(5), 711–720.

- Wosu, A. C., Gelaye, B., & Williams, M. A. (2015). History of childhood sexual abuse and risk of prenatal and postpartum depression or depressive symptoms: An epidemiologic review. *Archives of Women's Mental Health*, 18(5), 659–671.
- Yim, I. S., Tanner Stapleton, L. R., Guardino, C. M., Hahn-Holbrook, J., & Schetter, C. D. (2015). Biological and psychosocial predictors of postpartum depression: Systematic review and call for integration. *Annual Review of Clinical Psychology*, 11, 99–137.
- Yonkers, K. A., Wisner, K. L., Stewart, D. E., Oberlander, T. F., Dell, D. L., Stotland, N., ... Lockwood, C. (2009). The management of depression during pregnancy: A report from the American Psychiatric Association and the American College of Obstetricians and Gynecologists. *General Hospital Psychiatry*, 31(5), 403–413.

Suggested Reading

For more information about treatment options for perinatal depression, see the following:

- Cohen, L. S., Wang, B., Nonacs, R., Viguera, A. C., Lemon, E. L., & Freeman, M. P. (2010). Treatment of mood disorders during pregnancy and postpartum. *The Psychiatric Clinics of North America*, 33(2), 273–293.
- Pearlstein, T. (2008). Perinatal depression: treatment options and dilemmas. *Journal of Psychiatry & Neuroscience*, 33(4), 302–318.

Suicide in the Context of Health Psychology

Maryke Van Zyl-Harrison, Tanya Hunt, Rebekah Jazdzewski, Yimi Omofuma, Paola Mendoza-Rivera, and Bruce Bongar

Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA

Health psychology is a multifaceted subfield of psychology that aims to understand the human experience by combining two traditional theoretical approaches: natural science and human science (Marks, Murray, & Evans, 2005). The natural science approach focuses on studying human behavior through experimental studies, while the human science approach focuses on exploring the underlying meanings of human behavior (Marks et al., 2005). Through the combination of these two approaches, health psychology is able to study how biology, social conditions, and psychological stress intertwine and shape human behavior (Kaplan, 2009).

Health psychology currently covers a broad range of practice within the discipline of psychology. Throughout the last several decades, there have been numerous attempts to describe the activity, within the context, of what is now called health psychology. Some of the terms previously used have included behavioral medicine, psychosomatic medicine, and medical psychology. Behavioral medicine is the interdisciplinary field related to the development and combination of behavioral and biomedical science, knowledge, and procedures involving prevention, diagnosis, treatment, and rehabilitation of health and illness (Schwartz & Weiss, 1978). This definition was purposefully intended to not subscribe to any one specific theoretical orientation or discipline. Those involved in this field includes various health professionals like health psychologists, nurses, nutritionists, physicians, and members of other fields who decide to engage in the practice, teaching, development of policy, or research related to the combination of behavioral and biomedical disciplines regarding health and disease. Psychosomatic medicine refers to the interdisciplinary practice of examining the interaction between social, psychological, and behavioral components of physiological processes. Medical psychology refers to both the practice of psychology within a medical school facility and the

study of psychological factors associated with all the components of diagnosing and treating physical health illness and disease. The relationship between physical and psychological symptoms can be addressed on individual, group, and systematic levels.

The American Psychological Association (APA) formally recognized health psychology as a specialty over 20 years ago (Belar & Deardorff, 2009). Health psychology entails the use of scientific knowledge and research regarding psychological factors concerning the interaction of thoughts, emotions, personality, and biopsychosocial factors adaptation to illness and disease, disability, rehabilitation, and health promotion involving individuals, groups, and the healthcare system.

Health Psychology Theory of Suicide

The purview of health psychology includes the examination of environment, cultural factors, mental distress, health habits, impulsivity, substance use, coping skills and resources, and chronic stress levels as divisions of mechanisms that may lead to risky or unhealthy behaviors (Taylor, Repetti, & Seeman, 1997). Health psychology evaluation of the underlying factors of risky behavior can assist in identifying factors that can lead to suicidal behaviors. However, reducing suicide behaviors includes not only reducing problematic behaviors but also increasing positive behaviors. It must be mentioned that cultural factors can also impact the acceptance of suicidal behaviors. Therefore, understanding the effect of culture, healthy environments, support systems, resilience, and coping skill can lead to the formulation of protective factors and safety plans that may prevent or reduce suicidal behaviors.

The role of psychology in health is quite prominent as the literature suggests that most health models hypothesize that psychosocial stressors negatively impact the physical and mental health of individuals (Watson & Pennebaker, 1989). For example, a review by Leeb, Lewis, and Zolotor (2011) found that severe psychological distress during childhood was positively correlated with future negative physical health outcomes (e.g., chronic pain, respiratory illness, cancer), psychological disorders (e.g., depression, anxiety, PTSD), and increased suicide risk. Considering that suicide is regarded as the tenth leading cause of death in the United States (CDC, 2015), the field of health psychology has heightened its efforts to the understanding and reduction of factors that lead to an increased risk of suicide. Particularly, two health psychology theories, the biopsychosocial perspective and the lifespan perspective, may be used to understand the topic of suicide from a health psychology perspective.

Biopsychosocial Perspective

The biopsychosocial perspective is a scientific model that considers the development of illness and recovery due to the interaction of biological factors, such as genetics; psychological processes, like mood and personality characteristics; and social factors, including culture and ecology (Engel, 1980). Evaluating all the elements that impact a person both positively and negatively allows for a more comprehensive understanding of the development and maintenance of illness, recovery, and well-being. The biopsychosocial perspective combines all these previously independent factors of health and disease and recognizes that the non-physiological elements of a person can change the person's physiological reaction, resulting in vulnerability to the development of disease. In addition, these nonphysiological elements can impact the course of the illness (Jackson, Antonucci, & Brown, 2003). This perspective

is of particular value when considering the increasing intersectionality of populations. Hoffman (2000) discussed how the biopsychosocial perspective can potentially explain some trends concerning the issue of suicide. According to Engel's (1997) biopsychosocial model, physical health is affected by biological, cultural, and psychological variables (Marks et al., 2005). This perspective (which is one of the main influences of health psychology) may help to understand why suicide rates are increasing among patients with chronic illness, as well as why there appears to be an increased awareness about how psychosocial factors play a role in suicide (Hoffman, 2000). Hoffman (2000) suggested that, even though humans seem to be living longer, they are experiencing more chronic illness and a poorer quality of life. Therefore, this decreased quality of life may have led to an increased motivation to commit suicide and to a change in how societies view suicide, which has led to policy changes regarding the legitimacy of physician-assisted suicide (e.g., Oregon's *Death with Dignity Act*) (Hoffman, 2000).

Lifespan Perspective

The lifespan perspective relates to the study of the consistency and transformation of development that occurs throughout the course of life. This perspective stipulates that no particular age period can be considered the primary attribute to the regulation of the developmental process. Essentially the lifespan perspective maintains that during all stages of life, the developmental processes are always at work and can occur at any time during a person's life from birth to death. The system of changes that occur during development can differ in terms of time, direction, and order (Baltes, 1987). A theoretical aspect of the lifespan perspective is the gain/loss argument, which is a product of the belief that function declines as humans age. However, it has been proposed that any change within the adaptive capacity is a form of development, regardless if the change is positive or negative, suggesting that all growth and change is a combination of desirable and undesirable effects (Baltes, 1987). Another theoretical foundation of the lifespan perspective is plasticity, meaning an individual's development is based on testing the environmental limits and experiences, suggesting that humans are molded by the limitations of their cultural environments (Baltes, 1987). The lifespan perspective can also be taken to explain suicide trends (Hoffman, 2000). According to this perspective, people are more likely to be subjected to illness and isolation as they become older; thus, research shows that suicide might be seen as a more acceptable solution to chronic illness and poor quality of life among elderly individuals who have a harder time dealing with the vicissitudes associated with aging (Hoffman, 2000). However, more research is required to address how lifespan issues may affect the views and perceived consequences of suicide.

Suicide in the Context of Primary Care Medicine

Since suicide tends to be viewed as a mental health issue, clinicians in primary care medicine are often less prepared to respond to suicidal thoughts or behaviors. There are clear gaps in the training of primary care clinicians when it comes to the assessment and treatment of suicide. It is important to consider possible psychological causes of physical complaints, particularly those that are challenging to explain using medical reasoning. Additionally, physical illness, particularly chronic illness, may lead to a sense of hopelessness and suicidal ideation. Ultimately, a patient-centered integrative care approach compliments an enhanced awareness of culturally based expressions of psychological distress.

Training

Keeping in mind the trends indicating an increase in suicide rates among chronically ill individuals (Hoffman, 2000), one could imagine that the issue of suicide is likely to be prevalent in primary care practices. Indeed, a study by Luoma, Martin, and Pearson (2002) found that, while only one in five victims of suicide reached out to mental health providers within 1 month of committing suicide, approximately 45% of suicide victims sought primary care providers within 1 month of committing suicide. The tendency to turn to primary care physicians for mental healthcare could present a potential problem if primary care physicians are not properly trained to identify or treat suicidal individuals. According to Sudak et al. (2007), even though the Accreditation Council for Graduate Medical Education requires training programs to provide students with suicide-related didactic content, less than 50% of the training directors that participated in their study demonstrated confidence or satisfaction with the adequacy of their programs' suicide prevention training. Another potential problem that stems from the limited suicide prevention training received by primary care physicians is that they are not mandated to obtain suicide-related continuing education, yet they have the capacity to prescribe psychotropic medications to depressed patients, which may in turn exacerbate their suicidal ideations (Stuber & Quinnett, 2013). Literature shows that antidepressants like selective serotonin reuptake inhibitors (SSRI's) are linked to increased suicide attempts, particularly among children and young adults (Fergusson et al., 2005; Ghaziuddin, Merchant, Dopp, & King, 2014). These findings are particularly important since suicide is the third leading cause of death in the United States for individuals between the ages of 15 and 24 (Swanson, McGinty, Fazel, & Mays, 2015, p. 370).

Common Presentation

Since primary care is the first line of defense against suicide, it is important to consider the warning signs and common presentations of risk that primary care physicians may come in contact with. Suicidal ideation may be presented in ways that are not merely psychological, but present as somatic symptoms. Individuals endorsing suicidal thoughts often present comorbid mood and substance use disorders (MacLean, Kinley, Jacobi, Bolton, & Sareen, 2011). Older adults who are suicidal tend to seek out their primary care physicians with complaints of somatic symptoms related to physical illness or functional impairment, rather than depressive symptoms (Conwell et al., 2000). Similarly, Asian American patients tend to present psychological distress as somatic symptoms like headaches or stomach pain. Individuals from different cultures may express suicidality in different ways. It is important for clinicians in primary care to be aware of these atypical presentations of psychological distress, particularly suicide, in order to accurately assess suicidal ideation.

Furthermore, an awareness of the differences in suicide rates as well as the commonly used means of suicide among individuals who share a cultural identity could enable the early detection of suicide risk in primary care. It is important to note that suicide is the eighth leading cause of death among American Indian/Alaskan Natives and the tenth leading cause of death among Caucasians in the United States. Elderly Asian/Pacific Islander women over the age of 65 have higher suicide rates than any other ethnic group in the same age range (Chu, Hsieh, & Tokars, 2011). African American adolescent boys and Latina adolescent girls have been shown to have higher suicide rates, suicidal ideation, and attempts than their same-aged Caucasian peers, and ideation, attempts, and completions among LGBTQ individuals have been consistently higher when compared with nonsexual minority populations (Silenzio, Pena, Duberstein, Cerel, & Knox, 2007).

Mind–Body

Physical pain can not only present negative physical sensations but also emotional distress. Medical and psychological problems can sometimes mask one another. While some patients may report medically unexplained physical symptoms (MUPS) (Weiland et al., 2015), often-times categorized under somatic symptom disorders in the Diagnostic and Statistical Manual of Mental Disorders (DSM), others can experience medical illnesses that first present as psychological symptoms. Some patients may present with mental symptoms, such as depression and anxiety, which are caused by, or exacerbated by, physical diseases such as pancreatic carcinoma, diabetes, heart disease, arthritis, kidney disease, HIV/AIDS, lupus, multiple sclerosis (MS), and hypothyroidism. As psychologists, it is important to conduct thorough interviews at the start of treatment, which includes gathering a medical history, making a referral for a physical evaluation to rule out medical conditions that may be impacting their symptoms, and being aware of changes in patient presentation in terms of appearance, emotional state, physical state, and symptomatology. Conversely, some patients may present in primary care with MUPS. Research indicates that up to 50% of patients who present in general or specialist care are MUPS patients (Weiland et al., 2015). Many of these patients report not feeling understood or empathized with and can develop symptoms of depression and anxiety as a result (Weiland et al., 2015). Furthermore, medical specialists may subject these patients to different tests, for fear of missing a medical cause, and may find these patients difficult to handle (Weiland et al., 2015). Patient-centered communication can help improve patient outcome and overall experience. As part of a multidisciplinary team, psychologists can help develop training programs for specialists in other disciplines on how to assess patients who have MUPS (Weiland et al., 2015).

Impact of Suicide on Health

Suicidal ideation affects individual health, while acts of suicide affect community health. Suicidal ideation is correlated with an increase of substance use. Past suicidal ideation or suicidal attempts increase the likelihood for opioid misuse (Ashrafioun, Bishop, Conner, & Pigeon, 2017). Suicidal attempts are also strongly associated with later alcohol, nicotine, and cannabis dependence (Agrawal et al., 2017). General physical health is also affected by suicidal ideation. Perceived health decreases as suicidal ideation increases (Kim, Hong, Yook, & Kang, 2017). Exposure to a peer suicide in one's community increases the likelihood of another suicide in the community, a phenomenon called suicide contagion (Randall, Nickel, & Colman, 2015). Suicidal contagion is especially impactful for adolescents and in indigenous communities (Goodwin-Smith, Hicks, Hawke, Alver, & Raftery, 2013; Randall et al., 2015). The continuous exposure to death by suicide crumbles social well-being within the community and brings added distress to already vulnerable populations (Goodwin-Smith et al., 2013).

Health Psychology Preventing Suicide

Many general health diagnoses, such as cancer, cardiac disease, chronic pain, diabetes, functional bowel disorder, and erectile dysfunction, have been linked to an increase in stress levels, physical and psychological pain, as well as an increase in risk of suicide among patients. Common mental illnesses, such as depression, panic attacks, bipolar disorder, and posttraumatic stress disorder (PTSD), are often associated with a risk of suicide. Furthermore, there is a growing body of research highlighting the direct or indirect correlation between unhealthy

behavior and diseases. With clinical psychology being a field focused on the study of behavior, and psychologist being trained and equipped to manage suicide crisis, it therefore stands to reason that incorporating mental healthcare in the treatment of individuals diagnosed with general health problems, at an early stage, can help reduce the risk of death by suicide.

Individuals diagnosed with terminal or chronic illnesses such as cancer, cardiac diseases, or chronic pain often report experiencing feelings of hopelessness (Ahn et al., 2015; Suarez et al., 2015), which is correlated with an increased risk of suicidal behavior (Hirsch, Visser, Chang, & Jeglic, 2012). Evidence-based psychological treatment approaches, such as cognitive behavioral therapy (CBT) and dialectical behavior therapy (DBT), have strong research backing as treatments that help reduce suicidal cognitions, behaviors, and feelings of hopelessness (Comtois & Linehan, 2006; Mewton & Andrews, 2016). Having psychologists as part of treatment teams for patients with terminal or chronic illnesses can help reduce the risk of suicidal behavior, as psychologists can provide the vital support required to treat suicidal ideation.

Identification of Risk

Assessment of suicide risk in patients in primary care occurs similarly to risk assessment in the mental health field. Assessment tools such as the Beck Depression Inventory and Hospital Anxiety and Depression Scale (Aiello-Laws, 2010) can be used. However, some assessment tools used by psychologists can be cumbersome and lengthy and may not be appropriate for use in the primary care setting. Highlighted as a core knowledge and skill, it is important for clinicians to be competent in selecting appropriate evidence-based screening measures for primary care settings. Psychologists can assist members of the treatment care team, such as nurses, who have more interactions with the patients, in incorporating brief measures that can be included in routine appointments. These measures may assist the treatment team in monitoring for illnesses like depression, anxiety, or insomnia, which can co-occur with medical diagnoses, as well as risk-related symptoms like hopelessness. Some medication may lead to impulsive or suicidal behavior or have side effects that increase depressive symptoms. As highlighted above, it is imperative that clinicians be aware of the different side effects of the medications that their patients are taking and that they monitor the patient's response to their medications frequently throughout treatment.

Integration

Integrated care can be defined as unified care experienced by patients, when a team of behavioral health and primary care providers, in collaboration with the patient and their family, using an organized and cost-effective method, work together for the purpose of providing patient-centered care. The healthcare system is steadily moving toward a more integrated approach to treatment, incorporating biological, psychological, and social factors in patient care. The Joint Principles of the Patient-Centered Medical Home (PCMH), published in 2007, highlighted the need for personal, coordinated, and comprehensive healthcare by a team of clinicians, addressing all of a patient's needs, further emphasizing the importance of integrated behavioral healthcare. One of the key aspects addressed by integration is unification of records. Integration allows for the availability of shared medical records, collaborative decision making, and shared responsibility of patient care. Barriers to communication between healthcare professionals are reduced, allowing for improved

treatment of the patient as a whole, incorporating different aspects of the patient's life in their treatment as is applicable.

Traditionally, the role of psychologists was to assess, diagnose, and treat psychopathology. However, over previous decades, psychology in healthcare has expanded to be multifaceted, from understanding the development and maintenance of illness and disease to determining the development and impact of individuals' and communities' health behaviors (Sarafino & Smith, 2014). Health psychology has provided psychological interventions aimed to address and understand health behavior, provide a basis for clinical care, promote research, and inform program and public policy implementation. The role of psychology within the field of health originates from the premise that individuals' psychological processes are interrelated to biological processes. The understanding associated with what and how all behaviors, including health-related ones, are influenced is invaluable to the medical care system. This idea has led to the development and implementation of psychologically driven interventions to promote health. This multisystem approach combining psychological and physical aspects has proven to be successful in improving functioning and well-being.

Though significant progress has been made, there remains a high level of stigma surrounding mental health. Integrated care reduces the focus on mental health and increases the emphasis on the overall well-being of the patient, thereby reducing the stigma surrounding mental health treatment. Furthermore, the patient-centered approach helps to empower patients by increasing their involvement in their own care and fostering a sense of control over their treatment. The population of the United States is increasingly growing more diverse and older. Therefore, the field of health psychology must continue to refine and adapt conceptual models, research methods, and resources for application that are influenced by these variations.

Changes over Time

Suicide prevention in healthcare has expanded from targeting individuals in crisis to a preventative approach that involves the community in outreach and prevention efforts. Stakeholders in the community are now being trained to identify suicidal community members. Social relationships are being utilized in suicide safety plans to facilitate resilience. Media campaigns not only target at-risk individuals but also are beginning to offer information about how to be an advocate for a suicidal peer. These new developments are utilizing the community's unique risk and resilience factors to create suicide prevention programs.

WHO Global Goals

The Millennium Development Goals (MDGs) were created in 2000 by a collective of 189 countries. The objective was for countries and partners to work together in order to globally reduce poverty, gender inequality, child mortality, and diseases such as HIV/AIDS and to improve maternal health, environmental sustainability, and global partnerships, with targets set for 2015. Many of these goals relate to health, whether directly or indirectly. One of the goals outlined by the MDGs and the World Health Organization (WHO) is the reduction of suicide. In an effort to sustain and expand upon the MDG's of 2000, the UN presented a list of 17 sustainable development goals (SDGs), to be met by 2030. Specifically, these SDG's resolve to reduce premature mortality related to noncommunicable diseases by one third to promote mental health and well-being. The objective is to achieve a 10% reduction of suicide

by 2020 (World Health Organization, 2013). To reach this goal for suicide, smaller goals to increase mental health were set by the WHO. With this objective in mind, countries are encouraged to use expanded mental health treatments in order to prevent suicide. The inclusion of noncommunicable diseases and the explicit mention of mental health serve as an indication that mental health and well-being are of increasing importance on the agenda of global development.

Future Directions

The inclusion of mental health, particularly suicide, in the WHO recommendations toward improved global health, indicates how significant the issue of suicide is from a health perspective. As a result, several policy changes have been implemented by national and global organizations in an attempt to enhance suicide prevention efforts worldwide. The WHO and the US Joint Commission on Accreditation of Healthcare Organizations (JCAHOs) have employed specific recommendations that aimed to improve suicide risk assessment and treatment. Some of these recommendations included conducting suicide risk assessments on any individual over 10 years old that presented with either a medical or psychological condition, as well as being responsible for deglamorizing suicide in the media (Dua et al., 2011).

WHO Recommendations

Over time, recommendations from the WHO for suicide prevention have evolved from an individualistic approach to one that begins with the community and then reaches inward. In 2000, the WHO focused their recommendations on demographic risk factors. These recommendations were focused on those with a high risk of suicide. The recommendations consisted of detecting such individuals and managing individual risk by asking targeted questions and referring them to a psychiatrist or hospital. The WHO expanded suicide prevention in 2001 with the recommendation of increased research, training for mental health workers, and public awareness campaigns. An ecological orientation became the framework in 2003 for suicide prevention. This involves intertwining organizations such as schools, companies, and regions to create health networks (Kickbusch, 2003).

The current recommendations consist of surveillance, prevention, and evaluation. The WHO recommends surveillance of patterns of suicide in communities and suicidal behavior in individuals. Additionally, it suggests that community patterns such as suicidal contagion should be observed and recorded. The common behaviors of suicidal individuals should also be documented in order to increase knowledge of risk factors. Knowing how communities and individuals react in regard to suicide creates opportunities for more accurate prevention strategies.

The WHO has three levels of prevention strategies: universal, selective, and indicated. Universal prevention strategies reach the entire population. This type of strategy is effective for reaching low-risk individuals and educates non-suicidal individuals in order to create a supportive network. A mental health stigma awareness campaign is an example of a universal prevention strategy. Selective interventions target specific vulnerable populations, such as those who are unemployed, adolescents, and people with mental illnesses. Selective interventions focus on training for stakeholders in communities that are vulnerable to suicide. One such intervention would be training high school teachers how to identify and appropriately support a suicidal student. The general training of teachers is a selective intervention, but this

specific action of the teacher is an indicated intervention. Indicated interventions directly target individuals that have a high risk for suicide. Integrating a public health approach into the prevention plan is recommended by the WHO. This shows a commitment to suicide prevention from the government. Restricting suicide means and increasing access to mental health services are common interventions that governments have implemented successfully to reduce suicide.

Finally, the aforementioned prevention strategies need to be evaluated in order to ensure effectiveness. The outcome being measured should be clearly outlined before the intervention is administered. Important evaluation metrics include the rate of suicide, suicide attempts, and suicidal ideation. The population being evaluated should be considered when choosing an outcome to measure. Suicidal ideation should be used instead of suicide in a small population since suicidal ideation is more common and enough data is available to conduct statistically significant evaluations.

Joint Commission Recommendations

The Joint Commission developed their own set of recommendations to reduce suicide mortality nationally in the United States, which addressed the provision of suicide screenings, assessing for safety needs, and providing resources like crisis hotline numbers for discharged patients (Czyz et al., 2016). Recently, the Joint Commission's recommendations were updated to include "safety planning, discharge, treatment, and follow-up care" (p. 175), among other things (Czyz et al., 2016)

The Joint Commission's recommendations for preventing suicide are targeted for health-care professionals. Their approach begins with assessing all patients with an evidence-based screening tool and observing high-risk individuals through their history of suicide. Patients in a suicidal crisis should not be left alone. The emergency department or psychiatric unit should be immediately called, and possible suicidal means, such as bandages, elastic tubing, and plastic bags, should be kept away. Lower-risk individuals should be given referrals to outpatient mental health providers, introduced to the suicide hotline, have lethal means restricted, and develop a safety plan. Even after discharge, suicidal prevention should continue. Safety plans are most effective when friends and family are involved. Therapists also have a direct role after discharge. The Joint Commission recommends therapies that directly target suicidal ideation like Cognitive Therapy for Suicide Prevention and Collaborative Assessment and Management of Suicide.

The Joint Commission had specific recommendations for suicide in the hospital setting. These were based differences between psychiatric hospitals and medical hospitals. Medically ill patients who committed suicide were older and more likely to be employed and married compared with the psychiatric patients. Since medically ill patients are more likely to commit suicide in the hospital, the Joint Commission recommends increased suicide assessment for patients in the medical hospitals. A systemic collection of data on suicides and suicide attempts in hospital settings should also be created to fill the gap of information for this issue.

As a recommendation for assessing suicide in primary care, be aware that individuals who are part of ethnic and sexual minority subgroups may be at higher risk for suicide, particularly those with multiple minority identities. Be cognizant that culture affects how suicidal ideation is expressed, which methods for suicidal behavior are chosen and which stressors are more salient. Keep in mind that a mental illness diagnosis is not an adequate indication of risk for suicide and that it may be necessary to consider culturally specific stressors and the social environment within the patient's cultural context.

Recommendations for Future Research

Successful integration of psychologists in primary care requires an understanding and acceptance of patient-centered care by healthcare providers. More research focusing on how to optimize the systems to ensure that every aspect of a patient's life is duly addressed is important. This includes research into and the development of policies that ensure that the practice, training, and financing of behavioral health services are included in benefit or insurance plans for patients.

Suicidal behavior can occur at different stages in an individual's life and in conjunction with different medical illnesses as well. Research has shown that individuals are more likely to visit their primary care providers prior to seeking out professionals for mental health-related problems (Luoma et al., 2002). It is therefore crucial for primary care providers to be equipped to assess for mental illness and risk-taking behavior in patients. Future research should look into evaluating treatment models in primary care settings, particularly focusing on risk assessment and crisis intervention for patients.

Integrative Care

Although public health organizations like the WHO and the US JCAHO have advanced how healthcare professionals manage patients exhibiting suicidal behavior, there remains plenty of room for improvement. To continue achieving progress, the best option appears to be for mental health providers and healthcare providers to take an integrative approach and collaborate in the management of their shared patients. Though healthcare practitioners are the experts on physical health, they do not always have the necessary training or the time to conduct psychological assessments and provide psychotherapy to their patients (Nash, McKay, Vogel, & Masters, 2012). Therefore, psychologists are pivotal members in this contemporary concept of multidisciplinary teams. In integrated primary care, effective psychologists must possess the knowledge and skills to serve as consultants, teachers, and supervisors for primary care providers, as well as properly assess patients, provide psychotherapy, remain up-to-date in evidence-based practices, evaluate progress, and serve as case managers.

Author Biographies

Maryke Van Zyl-Harrison, MA, is a doctoral candidate in clinical psychology with an emphasis in diversity and community mental health at Palo Alto University. Maryke earned her bachelor's degree from Stellenbosch University in South Africa and her masters in marriage and family therapy from Pepperdine University in Los Angeles. She has worked to address HIV/AIDS, substance abuse, and suicide risk among underserved communities. Maryke works with children with serious medical diagnoses and adults with chronic pain. Most recently, she serves as coordinator of a special interest group on risk, resilience, and reasons for living within the International Association for Suicide Prevention.

Tanya Hunt is currently working on earning her PhD in clinical psychology with an emphasis in diversity and community mental health at Palo Alto University. Her areas of concentration also include culture, trauma, and health psychology. She earned her BBA from the University of Kentucky. Tanya has over 20 years' experience working with marginalized populations. She has provided services in employment and substance abuse counseling, as well as adjudicating claims based on mental and physical health. Tanya is currently providing mental

health services including addressing health disparities to a diverse population within an integrated healthcare setting.

Rebekah Jazdzewski, BA, is currently working on her doctorate in clinical psychology with an emphasis in adult trauma at Palo Alto University. Rebekah earned her bachelor's degree from Point Loma Nazarene University where she studied coping mechanisms in underserved populations. Currently, she is working with Native American tribes to study suicide prevention and resilience. Rebekah also serves as a student therapist at the Gronowski Center, working as a therapist in community mental health.

Yimi Omofuma, MA, is currently pursuing her doctorate in clinical psychology with an emphasis in trauma at Palo Alto University. Yimi earned her bachelor's degree from Southern Illinois University, Carbondale, Illinois and her masters in forensic psychology from Fairleigh Dickinson University in New Jersey. She currently works at an Early Intervention Clinic, providing emergency psychology for recently traumatized individuals. Furthermore, Yimi serves on the board of the Imani Foundation, a nonprofit organization currently dedicated to working with women and children who are survivors of abuse in Nigeria.

Paola M. Mendoza-Rivera, BA, is currently pursuing her PhD in clinical psychology with an emphasis in forensic psychology at Palo Alto University. Paola achieved her bachelor's degree in general psychology as well as a second bachelor's degree in criminal justice from the University of New Haven in West Haven, Connecticut. Currently, she is working on building her resume and developing her practical clinical skills at Palo Alto University's Gronowski Center. Paola is serving as a student therapist in the Gronowski Center's Clinica Latina, a bilingual specialty clinic that specializes in treating underserved Latinx populations.

Bruce Bongar, PhD, ABPP, FAPM, CPsychol, CSci, is currently the Calvin Distinguished Professor of Psychology at Palo Alto University and has previously served as consulting professor of psychiatry and behavioral sciences at Stanford Medical School. Dr. Bongar received his PhD from the University of Southern California and maintained a private practice for 25 years. Dr. Bongar is past president of the section on Clinical Crises and Emergencies of the Division of Clinical Psychology of the American Psychological Association. Dr. Bongar has also been a winner of the Edwin Shneidman Award from the American Association of Suicidology for outstanding early career contributions to suicide research and the Louis I. Dublin Award for lifetime achievement in research on suicidology.

References

- Agrawal, A., Tillman, R., Grucza, R. A., Nelson, E. C., McCutcheon, V. V., Few, L., ... Hesselbrock, V. M. (2017). Reciprocal relationships between substance use and disorders and suicidal ideation and suicide attempts in the Collaborative Study of the Genetics of Alcoholism. *Journal of Affective Disorders*, 213, 96–104.
- Ahn, M. H., Park, S., Lee, H. B., Ramsey, C. M., Na, R., Kim, S. O., ... Hong, J. P. (2015). Suicide in cancer patients within the first year of diagnosis. *Psycho-Oncology*, 24(5), 601–607.
- Aiello-Laws, L. B. (2010). Assessing the risk for suicide in patients with cancer. *Clinical Journal of Oncology Nursing*, 14(6), 687.
- Ashrafioun, L., Bishop, T. M., Conner, K. R., & Pigeon, W. R. (2017). Frequency of prescription opioid misuse and suicidal ideation, planning, and attempts. *Journal of Psychiatric Research*, 92, 1–7.
- Baltes, P. B. (1987). Theoretical propositions of life-span developmental psychology: On the dynamics between growth and decline. *Developmental Psychology*, 23(5), 611.
- Belar, C. D., & Deardorff, W. W. (2009). Introduction to clinical health psychology. In C. D. Belar & W. W. Deardorff (Eds.), *Clinical health psychology in medical settings: A practitioner's guidebook* (pp. 3–16). Washington, DC: American Psychological Association. doi:10.1037/11852-001

- Centers for Disease Control. (2015). U.S. Government Reports. Atlanta, Georgia, USA.
- Chu, J. P., Hsieh, K.-Y., & Tokars, D. A. (2011). Help-seeking tendencies in Asian Americans with suicidal ideation and attempts. *Asian American Journal of Psychology*, 2(1), 25.
- Comtois, K. A., & Linehan, M. M. (2006). Psychosocial treatments of suicidal behaviors: A practice-friendly review. *Journal of Clinical Psychology*, 62(2), 161–170. MEDLINE with Full Text, EBSCOhost (accessed June 29, 2017).
- Conwell, Y., Lyness, J. M., Duberstein, P., Cox, C., Seidlitz, L., DiGiorgio, A., & Caine, E. D. (2000). Completed suicide among older patients in primary care practices: A controlled study. *Journal of the American Geriatrics Society*, 48(1), 23–29.
- Czyz, E. K., Horwitz, A. G., Arango, A., Cole-Lewis, Y., Berona, J., & King, C. A. (2016). Coping with suicidal urges among youth seen in a psychiatric emergency department. *Psychiatry Research*, 241, 175–181.
- Dua, T., Barbui, C., Clark, N., Fleischmann, A., Poznyak, V., van Ommeren, M., ... Freeman, M. (2011). Evidence-based guidelines for mental, neurological, and substance use disorders in low- and middle-income countries: Summary of WHO recommendations. *PLoS Medicine*, 8(11), e1001122.
- Engel, G. L. (1980). The clinical application of the biopsychosocial model. *The American Journal of Psychiatry*, 137(5), 535–544.
- Engel, G. L. (1997). From biomedical to biopsychosocial: Being scientific in the human domain. *Psychosomatics*, 38(6), 521–528.
- Fergusson, D., Doucette, S., Glass, K. C., Shapiro, S., Healy, D., Hebert, P., & Hutton, B. (2005). Association between suicide attempts and selective serotonin reuptake inhibitors: Systematic review of randomised controlled trials. *BMJ*, 330(7488), 396.
- Ghaziuddin, N., Merchant, C., Dopp, R., & King, C. (2014). A naturalistic study of suicidal adolescents treated with an SSRI: Suicidal ideation and behavior during 3-month post-hospitalization period. *Asian Journal of Psychiatry*, 11, 13–19.
- Goodwin-Smith, I., Hicks, N., Hawke, M., Alver, G., & Raftery, P. (2013). Living beyond Aboriginal suicide: Developing a culturally appropriate and accessible suicide postvention service for Aboriginal communities in South Australia. *Advances in Mental Health*, 11(3), 238–245. doi:10.5172/jamh.2013.11.3.238
- Hirsch, J. K., Visser, P. L., Chang, E. C., & Jeglic, E. L. (2012). Race and ethnic differences in hope and hopelessness as moderators of the association between depressive symptoms and suicidal behavior. *Journal of American College Health*, 60(2), 115–125. *Psychology and Behavioral Sciences Collection*, EBSCOhost (accessed June 29, 2017).
- Hoffman, M. A. (2000). Suicide and hastened death: A biopsychosocial perspective. *The Counseling Psychologist*, 28(4), 561–572.
- Jackson, J. S., Antonucci, T. C., & Brown, E. (2003). A cultural lens on biopsychosocial models of aging. *Advances in Cell Aging and Gerontology*, 15, 221–241.
- Kaplan, R. M. (2009). Health psychology: Where are we and where do we go from here? *Mens Sana Monographs*, 7(1), 3.
- Kickbusch, I. (2003). The contribution of the World Health Organization to a new public health and health promotion. *American Journal of Public Health*, 93(3), 383–388.
- Kim, M.-S., Hong, Y.-C., Yook, J.-H., & Kang, M.-Y. (2017). Effects of perceived job insecurity on depression, suicide ideation, and decline in self-rated health in Korea: A population-based panel study. *International Archives of Occupational and Environmental Health*, 90(7), 663–671.
- Leeb, R. T., Lewis, T., & Zolotor, A. J. (2011). A review of physical and mental health consequences of child abuse and neglect and implications for practice. *American Journal of Lifestyle Medicine*, 5(5), 454–468.
- Luoma, J. B., Martin, C. E., & Pearson, J. L. (2002). Contact with mental health and primary care providers before suicide: A review of the evidence. *American Journal of Psychiatry*, 159(6), 909–916.
- MacLean, J., Kinley, D. J., Jacobi, F., Bolton, J. M., & Sareen, J. (2011). The relationship between physical conditions and suicidal behavior among those with mood disorders. *Journal of Affective Disorders*, 130(1), 245–250.

- Marks, D. F., Murray, M., & Evans, B. (2005). *Health psychology: Theory, research and practice*. Sage.
- Mewton, L., & Andrews, G. (2016). Cognitive behavioral therapy for suicidal behaviors: Improving patient outcomes. *Psychology Research and Behavior Management*, 9, 21. PsycINFO, EBSCOhost (accessed June 29, 2017)
- Nash, J. M., McKay, K. M., Vogel, M. E., & Masters, K. S. (2012). Functional roles and foundational characteristics of psychologists in integrated primary care. *Journal of Clinical Psychology in Medical Settings*, 19(1), 93–104.
- Randall, J. R., Nickel, N. C., & Colman, I. (2015). Contagion from peer suicidal behavior in a representative sample of American adolescents. *Journal of Affective Disorders*, 186, 219–225. doi:10.1016/j.jad.2015.07.001
- Sarafino, E. P., & Smith, T. W. (2014). *Health psychology: Biopsychosocial interactions*. Wiley.
- Schwartz, G. E., & Weiss, S. M. (1978). Behavioral medicine revisited: An amended definition. *Journal of Behavioral Medicine*, 1(3), 249–251.
- Silenzio, V. M. B., Pena, J. B., Duberstein, P. R., Cerel, J., & Knox, K. L. (2007). Sexual orientation and risk factors for suicidal ideation and suicide attempts among adolescents and young adults. *American Journal of Public Health*, 97(11), 2017–2019.
- Stuber, J., & Quinnett, P. (2013). Making the case for primary care and mandated suicide prevention education. *Suicide and Life-Threatening Behavior*, 43(2), 117–124.
- Suarez, L., Beach, S. R., Moore, S. V., Mastromauro, C. A., Januzzi, J. L., Celano, C. M., ... Huffman, J. C. (2015). Use of the Patient Health Questionnaire-9 and a detailed suicide evaluation in determining imminent suicidality in distressed patients with cardiac disease. *Psychosomatics*, 56(2), 181–189.
- Sudak, D., Roy, A., Sudak, H., Lipschitz, A., Maltsberger, J., & Hendin, H. (2007). Deficiencies in suicide training in primary care specialties: A survey of training directors. *Academic Psychiatry*, 31(5), 345–349.
- Swanson, J. W., McGinty, E. E., Fazel, S., & Mays, V. M. (2015). Mental illness and reduction of gun violence and suicide: Bringing epidemiologic research to policy. *Annals of Epidemiology*, 25(5), 366–376.
- Taylor, S. E., Repetti, R. L., & Seeman, T. (1997). Health psychology: What is an unhealthy environment and how does it get under the skin? *Annual Review of Psychology*, 48(1), 411–447.
- Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*, 96(2), 234.
- Weiland, A., Blankenstein, A. H., Van Saase, J. L. C. M., Van der Molen, H. T., Jacobs, M. E., Abels, D. C., ... Arends, L. R. (2015). Training medical specialists to communicate better with patients with medically unexplained physical symptoms (MUPS). A randomized, controlled trial. *PLoS One*, 10(9), e0138342.
- World Health Organization (WHO). (2013). *Reports on Suicide*. Geneva, Switzerland: Author.

Suggested Reading

- Marks, D. F., Murray, M., & Evans, B. (2005). *Health psychology: Theory, research and practice*. Thousand Oaks, CA: Sage.
- Morrison, J. (2015). *When psychological problems mask medical disorders: A guide for psychotherapists*. New York, NY: Guilford Publications.
- Sarafino, E. P., & Smith, T. W. (2014). *Health psychology: Biopsychosocial interactions*. Hoboken, NJ: Wiley.
- Taylor, S. E., & Sirois, F. M. (1995). *Health psychology*. New York: McGraw-Hill.

Adherence to Behavioral and Medical Regimens

Tricia A. Miller^{1,2}, Summer L. Williams³, and
M. Robin DiMatteo²

¹*Santa Barbara Cottage Hospital, Santa Barbara, CA, USA*

²*Department of Psychology, University of California Riverside, Riverside, CA, USA*

³*Department of Psychology, Westfield State University, Westfield, MA, USA*

Introduction

Patient adherence (also called compliance) involves the degree to which patients follow treatment recommendations as directed by their healthcare provider. Although empirical evidence demonstrates that quality health outcomes depend heavily upon patients' adherence to recommended treatments, 25% of patients, on average, are nonadherent to prevention and disease management activities (DiMatteo, 2004). These activities include taking medications as prescribed, keeping medical appointments, engaging in regular disease screening, and following specific diet and exercise regimens (DiMatteo, Haskard-Zolnierrek, & Martin, 2012; Martin, Williams, Haskard, & DiMatteo, 2005). For patients with chronic disease, adherence can be as low as 50% or less (Vermeire, Hearnshaw, Royen, & Denekens, 2001).

Patient nonadherence can be intentional or unintentional. "Unintentional nonadherence" describes situations in which patients incorrectly believe they are adherent, or try to be adherent but fail, whereas "intentional nonadherence" describes the purposeful choice to dismiss treatment or change the regimen without consulting the provider (Lehane & McCarthy, 2007). Whether purposeful or accidental, nonadherence contributes to suboptimal health outcomes. Research has identified some clinical factors that may influence patient nonadherence, such as the complexity of the regimen prescribed, and patients' existing beliefs about the recommended treatment and the severity or seriousness of the disease condition (DiMatteo, Haskard, & Williams, 2007).

Patient nonadherence also places an appreciable economic burden on the US healthcare system. Empirical evidence suggests that as many as \$100 billion dollars are wasted annually

due to the cost of preventable hospitalizations (Cutler & Everett, 2010). Failure to properly adhere to treatment can also cause unnecessary complications or disease progression, with significant increases in the cost of care (Dunbar-Jacob, Schlenk, & McCall, 2012).

Current research suggests that improving adherence requires patients' knowledge and understanding of their disease and treatment regimen, a collaborative and trusting therapeutic partnership between patients and health professionals, and satisfaction with care.

The purpose of this chapter is to highlight the importance of patient adherence, discuss several factors that affect treatment adherence, and examine the Information–Motivation–Strategy Model (IMS Model) as a way in which clinicians and healthcare professionals can improve their patient's adherence behaviors.

Factors that Affect Patient Adherence

Several factors influence whether or not patients engage in disease management and adherence behaviors. Decades of research have identified both real and perceived doubts about the expected benefits and efficacy of treatment, potential financial constraints, and the lack of resources as factors that significantly affect treatment adherence (DiMatteo, Giordani, Lepper, & Croghan, 2002; Zolnierak & DiMatteo, 2009). Socioeconomic factors, financial constraints (e.g., cost of medications), and patient characteristics can also negatively affect adherence (Peltzer & Pengpid, 2013).

Some of the unique demands of patients' prescribed treatment regimens have been associated with lower rates of adherence. These include the number of medications prescribed, the frequency of dosing, and the specific routines of administering medications for complex treatments. Patient nonadherence can be as high as 70% for treatment regimens that are complex and/or require complex lifestyle changes (Chesney, 2003). Dezii, Kawabata, and Tran (2002) found that for patients with type 2 diabetes, adherence was higher for patients on a one-medication-per-day regimen compared with patients who were on a three-medication-per-day regimen. Forgetting to take (or how to take) prescribed medications also contributes to patient nonadherence. Over 56% of patients forget details of their medical instructions shortly after leaving their physician's or healthcare provider's office (Martin et al., 2005). Thus, it is important that healthcare providers explain specific steps of their patient's treatment regimens, review the most important details of their patient's treatment, and use written instructions to encourage patients to ask questions and recall important information.

Patients' level of health literacy can also affect treatment adherence (Gazmararian, Williams, Peel, & Baker, 2003; Zaghoul & Goodfield, 2004). Health literacy is defined as a patient's ability to appropriately obtain, process, and understand basic health information needed to make health decisions (Baker, 2006; Schillinger et al., 2003). The Institute of Medicine (2004) estimates that more than 90 million people in the United States lack the necessary health literacy skills needed to understand and act on health information given by their providers. Patients with low health literacy often use fewer preventive services, incur higher medical costs, have more limited understanding of their treatment regimen, and have poorer health status than those with higher health literacy (Gazmararian et al., 2003; Schillinger et al., 2003; Schillinger, Bindman, Wang, Stewart, & Piette, 2004). Thus, low health literacy increases health disparities and negatively impacts patients' self-management and collaborative care (Schillinger et al., 2004). The interpersonal dynamics of the clinician–patient relationship can also influence patterns of adherence behaviors. For example, patients who feel that their physicians communicate well with them and actively

encourage them to be involved in their own care are more motivated to adhere (O'Malley, Forrest, & Mandelblatt, 2002). Such agreement fosters a cohesive partnership and allows physicians and patients to work together toward a mutually agreed upon treatment plan (Jahng, Martin, Golin, & DiMatteo, 2005). Training physicians to be better communicators results in significant improvements in patient adherence. Research by Zolnierек and DiMatteo (2009) found that the odds of patient adherence were 1.62 times higher for physicians who were trained in communication skills compared with the physicians who did not receive communication training. Effective communication also gives patients the ability to build rapport and trust; clinicians who promote trust through collaborative partnerships and who express compassion and "bedside manner" for their patients are more likely to succeed in fostering adherence and cooperation for a variety of treatment regimens (Schillinger et al., 2003).

Improving Adherence Using the Information–Motivation–Strategy (IMS) Model

Current empirical research highlights the complexity of establishing adherence-enhancing interventions and suggests that interventions should be tailored to meet the specific needs of patients for the most optimal effectiveness (DiMatteo et al., 2012; Martin et al., 2005). Evaluating and targeting patients' specific needs can be a difficult task for clinicians and health-care providers, however, especially given the limited time constraints of the medical visit itself.

A well-organized and simple model known as the Information–Motivation–Strategy Model^(c) can be used by clinicians and healthcare teams during medical encounters to help guide and promote patients' adherence behaviors (DiMatteo et al., 2012). Utilizing these three elements, that is, providing patients with *information*, building *motivation*, and offering effective *strategies*, in the context of effective clinician–patient communication can result in substantial and significant improvements in adherence and overall health outcomes.

The *information* element of the IMS Model outlines the importance of empowering patients to become more knowledgeable about their disease or condition by means of effective clinician–patient communication. In a review assessing more than 300 studies, researchers found that patients often do not understand much of the information they receive from their health-care provider (Kindig, Panzer, & Nielsen-Bohlman, 2004). Proper understanding can be achieved when clinicians communicate effectively with their patients, however (Zolnierек & DiMatteo, 2009). Empirical research suggests a 19% higher risk of nonadherence among patients whose physician communicated poorly than among patients whose physician communicated well (Zolnierек & DiMatteo, 2009). Thus, cohesive partnerships, in which clinicians work together with their patients, make it possible for clinicians to both effectively and thoroughly provide clear information and check the adequacy of their patients' understanding (DiMatteo et al., 2012). Patients who feel that their clinicians communicate well with them and encourage them to be involved in their own care tend to be more motivated to adhere (Martin et al., 2005).

A patients' level of trust in their physician is also an essential component of the physician–patient relationship and can greatly affect patient adherence. Patients must believe that their physician is someone who can understand their unique experience and who can provide them with reliable and honest advice. Martin et al. (2005) found that adherence rates were almost three times higher in primary care relationships consisting of higher levels of patient trust and physicians' knowledge of their patient as a person. In fact, patients' trust in their physician has

been found to exceed many other variables in promoting both patient adherence and overall satisfaction with care (Martin et al., 2005).

The second element of the IMS Model, *motivation*, emphasizes the importance of shared decision making between patients and their physician in order to develop a treatment plan the patient believes in and one to which the patient can make a commitment (DiMatteo et al., 2012; Zolnierek & DiMatteo, 2009). Helping patients to believe in the potential efficacy of a recommended treatment and the establishment of informed collaborative choice can influence a patients' motivation to adhere (Zolnierek & DiMatteo, 2009). Some research suggests that when a patient believes in the importance of their recommended treatment, healthcare teams can use behavioral contracts to gain further commitment (Martin et al., 2005). Ultimately, patients are more adherent to treatment if they believe that the consequences of nonadherence are more detrimental to their health than if they take the consequences of nonadherence less seriously. Thus, it is extremely important for health professionals to discuss openly, with their patients, any beliefs and specific perceptions or expectations of treatment that may hinder adherence behaviors.

The final element of the IMS Model involves developing workable *strategies* or resources to help patients overcome any (real or perceived) barriers that may hinder adherence. Members of the healthcare team must be willing to direct patients to resources to overcome such barriers as the financial cost of medication, the disadvantages of complex treatment regimen, or the lack of transportation to scheduled appointments. Patients also need assistance and encouragement to access supportive resources from their social support group (such as spouse, family, or friends) and any other aid that might be available to them to support the regimen (e.g., community- or work-based interventions) (Miller & DiMatteo, 2013). Health professionals should routinely assess the levels of social support available to their patients, as research by DiMatteo (2004) has shown that the absence of social support is a significant barrier to adherence.

The IMS Model can be used as simple heuristic through which clinicians can target the specific needs of their patients in order to establish the most effective treatment. The IMS Model illustrates that knowledge, commitment, and ability are essential for improving patient adherence (DiMatteo et al., 2012).

Conclusion

Patient adherence is essential for the achievement of quality healthcare outcomes. Effective patient communication can increase patient satisfaction, improve overall quality of life, and ultimately reduce the risk of nonadherence. Current empirical research continues to try to understand, assess, and predict the many challenges that patients face in the management of their care. The elements of the *Information–Motivation–Strategy* (IMS) model can be used by clinicians and health professionals to assist patients in building effective long-term disease management strategies to improve overall health outcomes and ultimately maximize adherence to treatment.

Author Biographies

Tricia A. Miller, PhD, is the research scientist for Cottage Children's Medical Center. She received her doctorate from the University of California, Riverside, in social-personality psychology. Her research focuses on the social psychological process of health and medical

care delivery including provider–patient communication and treatment adherence. Dr. Miller is a lead contributor to grant-funded projects examining Pediatric Care Coordination among medically complex children and improvements in clinical and social outcomes for very low birth weight infants and their families.

Summer L. Williams, PhD, received her doctorate in social-personality psychology from the University of California, Riverside. Her research expertise is in health communication and health outcomes research, where she has spent the last decade examining doctor–patient communication in diverse populations, patient adherence, and patient satisfaction. She is an associate professor at Westfield State University in Massachusetts, currently teaching courses in introductory psychology, health psychology, social psychology, psychology of illness, and health of vulnerable populations.

M. Robin DiMatteo, PhD, is a distinguished professor emeritus of psychology at the University of California, Riverside. She received her BS in mathematics and psychology from Tufts University, and her MA and PhD degrees from Harvard University. She has served as a resident consultant in health policy at the RAND Corporation. Dr. DiMatteo has been elected Fellow of the American Association for the Advancement of Science. Dr. DiMatteo received the Distinguished Teaching Award from the University of California, Riverside, and is a member of the UCR Academy of Distinguished Teachers.

References

- Baker, D. W. (2006). The meaning and the measure of health literacy. *Journal of General Internal Medicine*, 21, 878–883.
- Chesney, M. (2003). Adherence to HAART regimens. *AIDS Patient Care STDS*, 17, 168–177.
- Cutler, D. M., & Everett, W. (2010). Thinking outside the pillbox-medication adherence as a priority for health care reform. *The New England Journal of Medicine*, 362, 1553–1555.
- Dezii, C. M., Kawabata, H., & Tran, M. (2002). Effects of once-daily and twice-daily dosing on adherence with prescribed glipizide oral therapy for type 2 diabetes. *Southern Medical Journal*, 95, 68–71.
- DiMatteo, M. R. (2004). Variations in patients' adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care*, 43, 200–209.
- DiMatteo, M. R., Giordani, J., Lepper, S., & Croghan, T. W. (2002). Patient adherence and medical treatment outcomes: A meta-analysis. *Medical Care*, 40, 794–811.
- DiMatteo, M. R., Haskard, K. B., & Williams, S. L. (2007). Health beliefs, disease severity, and patient adherence: A meta-analysis. *Medical Care*, 45, 521–528.
- DiMatteo, M. R., Haskard-Zolnierck, K. B., & Martin, L. R. (2012). Improving patient adherence: A three factor model to guide practice. *Health Psychology Review*, 6, 74–91.
- Dunbar-Jacob, J., Schlenk, E., & McCall, M. (2012). Patient adherence to treatment regimen. In A. Baum, T. A. Revenson, & J. Singer (Eds.), *Handbook of health psychology* (pp. 271–283). New York: Psychology Press.
- Gazmararian, J. A., Williams, M. V., Peel, J., & Baker, D. W. (2003). Health literacy and knowledge of chronic disease. *Patient Education and Counseling*, 51, 267–275.
- Institute of Medicine. (2004). *Health literacy: A prescription to end confusion*. Retrieved from <http://www.nap.edu/openbook.php?isbn=0309091179>
- Jahng, K. H., Martin, L. R., Golin, C. E., & DiMatteo, M. R. (2005). Preferences for medical collaboration: Patient-physician congruence and patient outcomes. *Patient Education and Counseling*, 57, 308–314.
- Kindig, D. A., Panzer, A. M., & Nielsen-Bohlman, L. (Eds.) (2004). *Health literacy: A prescription to end confusion*. Washington, DC: National Academies Press.

- Lehane, E., & McCarthy, G. (2007). Intentional and unintentional medication nonadherence: A comprehensive framework for clinical research and practice? A discussion paper. *International Journal of Nursing Studies*, 44, 1468–1477.
- Martin, L. R., Williams, S. L., Haskard, K. B., & DiMatteo, M. R. (2005). The challenge of patient adherence. *Therapeutics and Clinical Risk Management*, 1, 189–199.
- Miller, T. A., & DiMatteo, M. R. (2013). Importance of family/social support and impact on adherence to diabetes therapy. *Journal of Diabetes Metabolic Syndrome and Obesity: Targets and Therapy*, 6, 421–426.
- O'Malley, A. S., Forrest, C. B., & Mandelblatt, J. (2002). Adherence of low-income women to cancer screening recommendations. *Journal of General and Internal Medicine*, 17, 144–154.
- Peltzer, K., & Pengpid, S. (2013). Socioeconomic factors in adherence to HIV therapy in low and middle income countries. *Journal of Health, Population and Nutrition*, 31, 150–170.
- Schillinger, D., Bindman, A., Wang, F., Stewart, A., & Piette, J. (2004). Functional health literacy and the quality of physician-patient communication among diabetes patients. *Patient Education and Counseling*, 52, 315–323.
- Schillinger, D., Piette, J., Grumbach, K., Wang, F., Wilson, C., Daher, C., ... Bindman, A. B. (2003). Closing the loop physician communication with diabetic patients who have low health literacy. *Archives of Internal Medicine*, 163, 83–90.
- Vermeire, E., Hearnshaw, H., Royen, P. V., & Denekens, J. (2001). Patient adherence to treatment: Three decades of research a comprehensive review. *Journal of Clinical Pharmacy and Therapeutics*, 26, 331–342.
- Zaghloul, S. S., & Goodfield, M. J. (2004). Objective assessment of compliance with psoriasis treatment. *Archives of Dermatology*, 140, 408–414.
- Zolnierck, K. B. H., & DiMatteo, M. R. (2009). Physician communication and patient adherence to treatment: A meta-analysis. *Medical Care*, 47, 826–834.

Suggested Reading

- Riekert, K. A., Ockene, J. K., & Pbert, L. (Eds.) (2014). *Handbook of health behavior change* (4th ed.). New York: Springer.

Tobacco Use Disorder and Its Treatment

Victoria A. Torres¹, Lee M. Cohen¹, and
Suzy Bird Gulliver^{2,3}

¹*Department of Psychology, College of Liberal Arts, University of Mississippi, Oxford, MS, USA*

²*Baylor and Scott and White Hospitals and Clinics, Waco, TX, USA*

³*School of Public Health, Texas A&M University, College Station, TX, USA*

Overview

The diagnosis of tobacco use disorder is a relatively new name for a long-standing health problem that was previously referred to as nicotine dependence (Britt, Cohen, Collins, & Cohen, 2001). Though the terms “nicotine” and “tobacco” are often synonymously used, “tobacco” technically refers to a plant, while “nicotine” refers to a primary addictive chemical in the tobacco plant. Although this classification was somewhat recently amended, the use of products containing tobacco has been around for centuries. In fact, the popularity of such products swelled in the years preceding the establishment of the United States, and since that time, the tobacco industry has worked tirelessly to increase their consumer base. More recently, tobacco companies have marketed electronic nicotine delivery systems (ENDS). The popularization of these and other tobacco products have contributed to tobacco’s role as the number one cause of preventable death in our society (World Health Organization [WHO], 2011).

It is alarming to consider that tobacco use is currently linked to 6 million deaths annually (WHO, 2016). To put this in perspective, during the twentieth century, tobacco use was linked to more deaths than both world wars combined (Chalabi, 2013). Further, one-quarter of all deaths among US women and men aged 35–69 are directly linked to a smoking-related illness (Jha et al., 2013). In addition to high mortality rates, tobacco use is associated with declines in health status, economic productivity, and environmental conditions (WHO, 2011).

History and Landmark Studies

Tobacco use has a long history, dating back to the pre-Columbian period where indigenous peoples chewed, smoked, and inhaled tobacco for medicinal and ritualistic purposes (Kohrman & Benson, 2011). This cash crop spread across Europe in the 1500s after Jean Nicot, a diplomat and scholar, first introduced tobacco to the French court. It was not until the beginning of the twentieth century, however, that researchers began warning the public about the harm caused by the use of tobacco. Specifically, in 1930, researchers in Germany noted a correlation between smoking and cancer. However, tobacco was not publically scrutinized until Reader's Digest published their 1952 article, *Cancer by the Carton*. As a result of this article, the public's attention was piqued and cigarette sales plummeted (CNN, 2000). Twelve years later, the surgeon general of the United States released a landmark report warning Americans of tobacco's adverse effects for the first time (U.S. Department of Health and Human Services [USDHHS], 1964). At that time, an estimated 42.4% of the US population were classified as current smokers (Wilson, 1967).

Since the release of the first surgeon general's report, tobacco companies and public health advocates have been at odds. Specifically, public health advocates have fought for harsher policies to protect the public, while big tobacco companies have worked to persuade the public that they have designed a "cleaner smoke." As consumers became more aware of the dangers associated with tobacco use, many attempted to quit. Others sued tobacco companies for failing to warn them of the deadly consequences of tobacco use. In 1996, the US Department of Health and Human Services appointed a panel of experts to create clinical practice guidelines. These guidelines were designed to assist in the delivery and support of evidence-based treatments for tobacco use and dependence (Abrams et al., 2003). Since that time, the guidelines have undergone several rounds of revisions utilizing an evidence base of over 8,700 research articles. Ten key recommendations, mostly focusing on brief and intensive treatments and system-wide changes, were put forth in the most recent update.

Despite an overall decrease in smoking rates and discovering more effective ways to help people quit, problems associated with the use of tobacco products remain. Although smoking rates declined by 10% between 1997 and 2015, 36.5 million Americans continued to smoke in 2015 despite awareness of the harmful consequences to themselves and others (Centers for Disease Control and Prevention [CDC], 2016). Additionally, since the release of the surgeon general's report, over 2.5 million *nonsmoking* Americans have died due to the dangerous effects of secondhand smoke (USDHHS, 2014).

Another more recent concern involves the misperception that alternative tobacco products (e.g., e-cigarettes and smokeless tobacco) are safe. In 2003, ENDS became globally available. Now a multibillion dollar industry, ENDS advertisers promise a "cleaner smoke," leading many to mistakenly assume that these products are less harmful or represent a viable, safe substitute for cigarettes that can be used to aid in cessation (USDHHS, 2016). The available data, however, suggest this is not the case. Specifically, e-cigarettes contain aerosol, while smokeless tobacco products contain known carcinogens and toxic chemicals (USDHHS, 2016). In addition to this misperception, tobacco companies appear to be targeting youth, adding various flavors that mimic familiar sweet tastes (e.g., cotton candy, gummy bears, and sweet tarts) to these products. Sadly, it appears to be working as e-cigarette use has increased by 900% among high school students between 2011 and 2015 (USDHHS, 2016). In addition to adolescent smokers, several populations are considered particularly vulnerable to developing tobacco use disorder. Broadly, those experiencing negative affect, women, blue-collar workers, low-income populations, ethnic minorities, and those without health insurance have

a higher risk of adopting tobacco use patterns leading to disorder (Cohen, McCarthy, Brown, & Myers, 2002; Orleans et al., 1998).

Classification of Tobacco Use Disorder

Researchers and clinicians have historically grouped tobacco use with other substances of abuse. However, tobacco influences the human body in a distinct way as nicotine acts as both a stimulant and a depressant. Therefore, it causes the body to relax while simultaneously causing blood pressure to increase. Accordingly, several diagnostic manuals, namely, the APA's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and the *International Classification of Diseases* (ICD), began to address nicotine use disorders as distinct from other substance use disorders.

Exactly what symptoms constitute tobacco use disorder has changed over time. Most recently, the changes made in the current version of the DSM (5th Edition; DSM-5; American Psychiatric Association [APA], 2013a) includes increased clarity with regard to smoking patterns and severity of cravings (APA, 2013b). Additionally, the term "tobacco abuse" was added, a severity criterion was implemented (i.e., experiencing 2–3 criteria indicates "mild" severity, 4–5 indicates "moderate" severity, and 6 or more indicates "severe"), new specifiers "in a controlled environment" and "on maintenance therapy" were included, and the "physiological" subtype specifier was removed (APA, 2013a). Further, early remission is defined as "at least 3 but less than 12 months without meeting substance use disorder criteria (except craving)," and sustained remission is defined as "at least 12 months without meeting the criteria (except craving)" (APA, 2013a).

The DSM-5 (APA, 2013a) defines tobacco use disorder as "a problematic pattern of tobacco use leading to clinically significant impairment or distress" where a minimum of two of the following symptoms occur within a 12-month period:

- 1 Tobacco is used in larger amounts or for a longer period than intended.
- 2 There is a persistent desire to quit use or there are unsuccessful quit attempts.
- 3 A great deal of time is spent obtaining or using tobacco.
- 4 There are cravings or strong urges to use tobacco.
- 5 There is interference in daily activities that occurs as a result of tobacco use.
- 6 Use continues despite adverse consequences in personal or social situations.
- 7 Important activities (social, occupational, recreational) are given up due to tobacco use.
- 8 Tobacco use persists in situations where it is physically hazardous.
- 9 Despite having a physical or psychological tobacco-related problem, tobacco use persists.
- 10 Tolerance to tobacco is observed.
- 11 A well-defined withdrawal syndrome is experienced when use is discontinued (p. 571).

Tolerance and withdrawal are other constructs associated with tobacco use disorder. Tolerance is defined by either "a need for markedly increased amounts of tobacco to achieve the desired effect" or "a markedly diminished effect with continued use of the same amount of tobacco." Withdrawal is recognized by either "the characteristic withdrawal syndrome for tobacco" or use of tobacco "to relieve or avoid withdrawal symptoms." The withdrawal syndrome is characterized by an "abrupt cessation of tobacco use" or a "reduction in the

amount of tobacco used.” Further, four or more of the symptoms below are experienced within 24 hr of reduced use or cessation (p. 575):

- 1 Irritability, frustration, or anger
- 2 Anxiety
- 3 Difficulty concentrating
- 4 Increased appetite
- 5 Restlessness
- 6 Depressed mood
- 7 Insomnia

Initiation and Maintenance

A common question that has plagued researchers and clinicians alike is, “Why do individuals start smoking in the first place given that we have data to support that it is unhealthy?” While the answer to this question has not yet been identified, much has been learned about the process of initiation and maintenance. Generally, tobacco use begins before 18 years of age, and recent research indicates that a person can become addicted to nicotine after just one cigarette (University of Massachusetts Medical School, 2007). Since 20–30% of those who experiment with tobacco will meet lifetime criteria for tobacco use disorder over the course of their lifetime, it is not surprising that many teen smokers who experiment with tobacco several times as a means of fitting in socially will progress to more frequent use (Abrams et al., 2003; American Academy of Addiction Psychiatry [AAAP], 2015). Further, those who experiment with smoking commonly develop associations between smoking, daily life occurrences (e.g., socializing, drinking, waking up, finishing a meal), emotional states (e.g., anxiety, anger), and sensory aspects of tobacco use. These associations frequently contribute to the maintenance of tobacco use. Since nicotine is highly addictive and tobacco use becomes easily engrained in a person’s daily life, quitting becomes difficult. Lastly, the interaction of nicotine and human physiology also helps to explain why people maintain this unhealthy behavior and find it difficult to quit. Tobacco companies have worked untiringly to design the perfect nicotine delivery system. After a person’s first inhalation, nicotine takes only 8 s to reach the brain, where it targets areas associated with feelings of pleasure and reward.

Benefits to Tobacco Cessation

The detrimental health effects of using tobacco products have been well established. Regardless of the manner in which it is used, tobacco harms the human body. It increases heart rate and blood pressure while simultaneously decreasing the health of respiratory and circulatory systems. In addition, protracted use of tobacco changes cellular characteristics, which may support the development of cancer. This said, research has indicated that vast improvements in health can occur in the absence of tobacco use (CDC, 1990). Specifically, in 2013, Jha et al. used the US National Health Interview Survey to obtain smoking and smoking cessation information from 202,248 men and women between 1997 and 2004. This data set revealed that adults who quit smoking reduced their risk of mortality from smoking-related illnesses by close to 90%. Researchers have also discovered tremendous health improvements including reduced smoking-related symptoms such as coughing and shortness of breath and decreased

risk of smoking-related illnesses (e.g., heart attack and diabetes), some of which occur within 20 min after a person stops smoking (American Cancer Society, 2016).

Evidence-Based Treatment

Now that it has been established that nicotine is a highly addictive substance, is delivered to the human brain in an optimal way, and becomes part of many aspects of individuals' lives, it should be clear that most individuals who wish to quit smoking find it difficult. In fact, 44% of smokers' quit attempts result in relapse within 14 days, and 60–98% of smoking quit attempts lead to relapse (Abrams et al., 2003). In addition to the more common challenges described thus far, people tend to present to treatment with an array of problems or “comorbidities” that may complicate the process of smoking cessation. Accordingly, clinicians should take an individual's unique barriers to smoking cessation into account when creating a treatment plan. Thankfully, a variety of options are available. Currently, the most effective treatment includes a combination of behavioral therapy and pharmacotherapy (i.e., nicotine replacement therapy [NRT]). Before delving into an overview of these broad treatment modalities (as well as other more specific ones), it is important to mention the importance of assessment, which is a common and important component throughout treatment.

Assessment

It is best practice to begin any cessation program with a comprehensive assessment. The goal of assessment is to equip the clinician with a better idea of how to work most effectively with a particular individual. Assessment should take different forms throughout treatment and can be useful in understanding an individual's barriers that may undermine a quit attempt. Generally, healthcare providers attend to five clinical areas when conducting smoking cessation assessments: (a) motivation, (b) nicotine dependence, (c) past quit attempts and smoking history, (d) substance abuse comorbidity, and (e) psychiatric comorbidity (Abrams et al., 2003). By attending to these five areas, clinicians can tailor smoking cessation treatment to specific clients to give them the best chance of success.

Additionally, assessment can inform which type of treatment may be best suited for a particular client given that different types of therapy have led to better treatment outcomes among certain populations (e.g., pregnant women, racial and ethnic minorities, low-income smokers; Vidrine, Cofta-Woerpel, Daza, Wright, & Wetter, 2006; Weinberger, Pittman, Mazure, & McKee, 2015). Lastly, given that a person's level of motivation and other relevant individual factors may change across time, it is important that assessment occur throughout treatment so that the clinician is quantitatively and qualitatively aware of how their clients are adjusting during the various stages of treatment.

Pharmacotherapy

Beginning with the development of nicotine gum, pharmacotherapy has been the principal treatment modality in most smoking cessation research studies. Since smoking is perpetuated by habitual behavior and the addictive nature of nicotine, it makes sense that the combination of nicotine replacement and behavioral therapies may be helpful for people attempting to quit. By using pharmacotherapy, smokers can focus more on the behavioral aspects of their habit, while at the same time, receiving help with respect to the severity of withdrawal symptoms that

they may be experiencing. Currently there are five modalities of nicotine replacement therapy (NTR; i.e., gum, patches, lozenges, inhalers, and nasal spray) and two oral medications that do not contain nicotine [Chantix (varenicline tartrate) and Zyban (bupropion hydrochloride)]. Of these options, nicotine gum, patches, and lozenges are available over the counter, while the others require a prescription. It is important to note that while each option in this category may come with its own side effects, use of such products has evidenced higher rates of smoking cessation success when compared with behavioral therapy alone (Fiore et al., 2000). Still, for those who are unable or unwilling to use pharmacotherapy, other options such as nicotine fading are available. Finally, while use of Chantix and Zyban can be used before the quit date, the NTRs should not start until one has quit smoking.

Behavioral Therapy: Preparing to Quit

Whether a person decides to receive brief or more intensive behavioral treatment, the first phase of such treatment involves preparation. While behavioral treatment may differ somewhat based on individuals' specific needs, it is generally the case that the early stage of this approach focuses on preparing the individual to quit. This includes the use of psychoeducation (e.g., learning about the effects of nicotine), motivational enhancement techniques (e.g., eliciting reasons for wanting to quit), preparation for overcoming withdrawal symptoms (e.g., brand fading or gradual reduction in number of cigarettes), unlearning a behavior (e.g., reducing the number of places an individual smokes), practicing urge control strategies (e.g., delaying the next cigarette), letting others know that you are working on quitting, and ultimately, setting a definitive quit date (which is a critical step given that many have indefinite plans to quit).

During the preparation stage, individuals learn as much as they can about their smoking behavior (i.e., when they smoke, what "triggers" their smoking) by self-monitoring and recording data relevant to this behavior. This phase enables individuals to plan their quit attempt (with the use of data) so that their chance for success is optimized. During this phase, individuals may be advised to purchase cigarettes in smaller quantities than usual so that they do not have too many cigarettes readily available to them or too many left over once they get to their quit date. This strategy is both economical and creates an inconvenience to their normal routine. Individuals may also wish to slowly reduce the number of cigarettes they smoke per day (or gradually change their brand of cigarettes to a brand with less nicotine) in order to minimize withdrawal when they quit (especially if they prefer to not use one of the available pharmacotherapies). It is during this phase of treatment that healthcare providers can help patients identify what in their environments may lead to failure. For instance, evaluation of an individual's context may reveal that stress at work leads to smoking. Learning and mastering stress reduction techniques as well as planning for what they can do rather than take a "smoke break," before the quit date, will likely help that individual increase the chance of a successful quit attempt (Abrams et al., 2003). Additionally, clinicians and smokers should consider their time commitments and availability before beginning treatment. The amount of time a smoker can dedicate to treatment must be considered to avoid gaps. Finally, past quit attempts that ended in relapse should be reframed as learning opportunities prior to beginning anew. Utilizing previous quit attempts in a positive way will likely contribute to success in a new quit attempt.

Behavioral Therapy: Quit Day

As mentioned above, a quit date is generally set during the preparation phase of treatment. Hopefully by this day, the individual has learned a great deal about their smoking behaviors

and is ready for any challenges they face. It is recommended that on this day, individuals change their regular routine. The reason for this is to lessen the occurrence of paired associations that will ultimately be too difficult to resist. For example, if a cigarette is paired with a cup of coffee at home in the morning, either drink tea or get the cup of coffee on the way to work and drink it in the car (where there are presumably no cigarettes available). It is also important on this day (and every day that follows) to be on guard for triggers and difficult situations.

Behavioral Therapy: Relapse Prevention

Being smoke-free will get easier and easier as time goes on; however, it is important for ex-smokers to not get overconfident and always be prepared to deal with the stressors or cravings they experience in ways other than smoking. It is important for individuals to utilize any urge control strategies they practiced during the preparation stage. Whereas in the preparation stage, a helpful urge control strategy was to delay the amount of time until the next cigarette, in this stage appropriate strategies include, escaping or avoiding situations that historically led to smoking, using distraction techniques such as staying busy, and substituting smoking with other, healthy behaviors, like eating carrots or chewing gum. It is also important to set up a reward structure for being successful (e.g., buying something with the money being saved from not smoking). Additionally, asking friends and family for support when things get rough may be helpful. Finally, if a cigarette is smoked, it is important to frame the occurrence as a “slip” rather than a failure or excuse to continue smoking regularly. If this is not possible, consider setting a new quit date and use all that has been learned from previous quit attempts.

Complementary and Alternative Treatments

Despite the advances in more traditional smoking cessation treatments, some people prefer to use methods that are less mainstream and not typically considered standard practice. While some of the treatments that fall into this category have been carefully evaluated, others have not. Complementary treatments are those that are used in conjunction with more traditional methods, whereas alternative treatments are those that are used instead of other options. Although a growing number of these types of treatment are available, a comprehensive discussion of all that are currently used falls outside the scope of this entry. Below are summaries for three such therapies including, hypnotherapy, acupuncture, and confectionery chewing gum. For a more thorough discussion, refer to the National Center for Complementary and Integrative Health’s Clinical Digest (2014), which can be found online at nccih.nih.gov/health/providers/digest/smoking.

Hypnotherapy

Hypnotherapy is a method that aims to decrease a person’s desire to smoke, strengthen the will to quit, and increase a person’s focus on quitting by acting on underlying impulses. Hypnotherapy treatments differ in regard to hypnotic induction, amount of sessions, duration of treatment, and use in combination with other therapeutic methods.

While research in this area is quite varied, hypnotherapy’s effectiveness has been studied alongside a number of other smoking cessation methods. For example, in 2010 Barnes and colleagues reviewed literature pertaining to the efficacy of hypnotherapy for smoking cessation

and found 11 studies with data from 1,120 participants. Overall, Barnes et al. (2010) found conflicting results with regard to hypnotherapy's efficacy. In comparison to no intervention, hypnotherapy was not demonstrated to be more effective 6 months after the beginning of treatment. Additionally, these studies did not reveal a greater effect when compared with rapid smoking or psychological treatments such as counseling (Barnes et al., 2010). More recently, Hasan and coauthors conducted a randomized controlled trial (RCT) among individuals who were hospitalized for a variety of health concerns including cardiac and pulmonary illnesses. This study revealed that at 12 (43.9 vs. 28.2%; $p = 0.14$) and 26 weeks post-hospitalization (36.6 vs. 18.0%; $p = 0.06$), hypnotherapy patients were more likely than NRT patients to be nonsmokers. These findings indicate that hypnotherapy may aid in smoking cessation; however, more research is needed to determine the extent to which hypnotherapy is efficacious and with which populations it is most useful. For a more complete review of the use of hypnotherapy for the treatment of smoking cessation, refer to the Cochrane report at www.cochrane.org/CD001008/TOBACCO_does-hypnotherapy-help-people-who-are-trying-to-stop-smoking.

Acupuncture

Acupuncture, a traditional Chinese method that uses needles to stimulate energy points, has also been promoted as an aid for smoking cessation. The aim of acupuncture techniques for smoking cessation is to reduce nicotine withdrawal symptoms. Similar to current research on hypnotherapy, studies on the efficacy of acupuncture for smoking cessation are mixed. While some lines of research support the use of acupuncture techniques in the short term, it is likely that these results are attributed to a placebo effect. Overall, acupuncture research has not revealed clear evidence in support of its effectiveness above and beyond other treatments (White, Rampes, & Ernst, 2014). For a more complete review of the use of acupuncture for the treatment of smoking cessation, refer to the Cochrane report at www.cochrane.org/CD000009/TOBACCO_do-acupuncture-and-related-therapies-help-smokers-who-are-trying-to-quit.

Confectionery Chewing Gum

One healthy alternative to smoking that has received empirical attention is the use of confectionery chewing gum. Previous research has shown that confectionery gum appears to aid in the reduction of withdrawal symptoms, particularly those related to negative affect, among individuals dependent on nicotine. In a series of studies, Cohen, Britt, Collins, al'Absi, and McChargue (2001); Cohen, Britt, Collins, Stott, and Carter (1999); and Cohen, Collins, and Britt (1997) have evaluated the utility of confectionery chewing gum as an aid to help smokers when they are asked to abstain from smoking for brief periods of time in the laboratory. Results from two of these studies (1997 and 2001) revealed that the use of chewing gum reduces the severity of self-reported withdrawal symptoms following 3–4 hr of abstinence. Results from the other study show that the use of chewing gum is associated with changes in smoking behavior (Cohen et al., 1999). Specifically, participants showed significant decreases in the number of cigarette puffs that were taken as well as increased periods of abstinence. More recently, the use of chewing gum has been shown to be useful in reducing symptoms of withdrawal over 24 (Cortez-Garland, Cohen, VanderVeen, & Cook, 2010) and 48 (Cohen, Collins, VanderVeen, & Weaver, 2010) hr of abstinence.

In conclusion, confectionery chewing gum has demonstrated its utility as an aid for smokers during periods of abstinence ranging from 3 to 48 hr. Chewing gum may be a useful adjunct to existing smoking cessation treatments given that it is easily accessible, inexpensive, and

simple to administer. Due to the well-documented risk of smoking and the difficulty individuals who smoke face during the cessation process, a behavioral substitute that can positively influence the symptoms of negative affect associated with nicotine withdrawal may improve upon treatment success.

Future Research Directions

Many important questions concerning tobacco use have been addressed over the past 50 years. Yet, better treatments for tobacco use disorder are needed as it is estimated that 1.1 billion people smoked tobacco in 2015 alone (WHO, 2017). Five key areas concerning tobacco use disorder treatment remain to be addressed.

First, since we know smoking initiation typically begins in adolescence and young adulthood, it may be helpful to understand more about underlying mechanisms unique to these age groups. By pinpointing smoking behavior, prevention and effective treatments should aim to reduce the number of people who attempt to begin smoking in the first place. High-risk and low-risk thinking patterns in adolescents can be identified and may aid in the development of critical prevention efforts for youth (Choi, Gilpin, Farkas, & Pierce, 2001). Since those who experiment with smoking are highly likely to become regular users, prevention efforts seem to be most effective during this developmental period. Second, more effective treatments are needed for adult smokers. Particularly, current research exploring the relationship of the neurobiology of nicotine addiction to behavioral, pharmacological, and combined treatments is needed. Third, more information about tailoring treatments to smokers with distinct profiles may be helpful. Continued research on how to most successfully match smoker profiles to treatment options may result in more efficacious smoking cessation outcomes. Fourth, although smoking and drinking alcohol seem to go hand in hand, we are still uncertain about how these two addictive behaviors are connected or why they tend to be seen regularly in conjunction with one another. More information about underlying smoking and drinking pathways could be helpful for understanding why people start smoking and drinking and how we can help them stop if they wish to do so. Finally, broad system-wide considerations must be taken into account to reduce the prevalence of tobacco use disorder. Strengthened collaborative efforts between researchers and practitioners will likely aid in this process. As researchers continue to pioneer new treatments and clinicians implement changes into practice, tobacco use disorder will hopefully become less of an issue in our society.

Conclusion

Based on recent data, 60–80% of Americans who currently report smoking cigarettes meet diagnostic criteria for tobacco use disorder (AAAP, 2015). A recent survey by the CDC indicates that while rates of cigarette smoking have been generally trending downward in the United States (CDC, 2016), there is a 900% increase in youth e-cigarette usage from 2011 to 2015. As such, more work in this area remains. Researchers have asserted that the initiation of tobacco use rates in a population predict future smoking patterns. However, little is currently known about adolescent and young adult smoking patterns. Future efforts must target youth tobacco prevention, more effective treatments, and improved system-wide cessation efforts. As public health advocates continue to be vigilant about this issue, a reduction or elimination of a widespread, long-standing problem will hopefully soon be seen.

Author Biographies

Victoria A. Torres, BA, is a doctoral candidate in the clinical psychology program at the University of Mississippi. Her current research focuses primarily on factors that contribute to tobacco use. Before Ms. Torres came to UM, she received her baccalaureate degree in psychology from Baylor University. After graduating from Baylor, she worked for 2 years as a research assistant at Baylor Scott & White as part of the Warriors Research Institute, where she contributed to several federally funded research projects.

Lee M. Cohen, PhD, is dean of the College of Liberal Arts and professor in the Department of Psychology at the University of Mississippi. Dean Cohen came to UM from Texas Tech University, where he served in a number of administrative roles including the director of the nationally accredited doctoral program in clinical psychology and the chair of the Department of Psychological Sciences. As a faculty member, he received several university-wide awards for his teaching and academic achievement. As a researcher, he received more than \$1.5 million from funding agencies, including the US Department of Health and Human Services, the National Science Foundation, and the National Institutes of Health/National Institute on Drug Abuse. His research program examines the behavioral and physiological mechanisms that contribute to nicotine use, and he worked to develop optimal smoking cessation treatments.

He is a fellow of the American Psychological Association and received his PhD in clinical psychology from Oklahoma State University.

Suzy Bird Gulliver, PhD, is a licensed clinical psychologist and clinical researcher. Currently, she serves as director and chief of the Warriors Research Institute (WRI) and as a professor of psychiatry at Texas A&M Health Science Center. She earned her PhD in clinical psychology from the University of Vermont and went on to work as a National Institute of Alcohol Abuse and Alcoholism-funded postdoctoral fellow at Brown University. Dr. Gulliver later spent 18 years in a variety of roles within the VA in Boston, Massachusetts. Before founding the WRI within Baylor Scott & White Health in 2013, Dr. Gulliver served as the director of the VA VISN 17 Center of Excellence in Waco, Texas. Dr. Gulliver's team has been federally funded and has published 88 peer reviewed manuscripts, 2 book chapters, as well as hundreds of presentations to academic, medical, and emergency response audiences.

References

- Abrams, D. B., Niaura, R., Brown, R. A., Emmons, K. M., Goldstein, M. G., & Monti, P. M. (Eds.) (2003). *The tobacco dependence treatment handbook: A guide to best practices*. New York, USA: Guilford Press.
- American Academy of Addiction Psychiatry. (2015). *Nicotine dependence*. Retrieved from <http://www.aaap.org/wp-content/uploads/2015/06/AAAP-nicotine-dependence-FINAL.pdf>
- American Cancer Society. (2016). *Benefits of quitting smoking over time*. Retrieved from <https://www.cancer.org/healthy/stay-away-from-tobacco/benefits-of-quitting-smoking-over-time.html#references>
- American Psychiatric Association. (2013a). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013b). *Highlights of changes from DSM-IV-TR to DSM-5*. Retrieved from <http://dsm.psychiatryonline.org/doi/abs/10.1176/appi.books.9780890425596.changes>
- Barnes, J., Dong, C. Y., McRobbie, H., Walker, N., Mehta, M., & Stead, L. F. (2010). Does hypnotherapy help people who are trying to stop smoking? *Cochrane Database of Systematic Reviews*, 10.

- Retrieved from http://www.cochrane.org/CD001008/TOBACCO_does-hypnotherapy-help-people-who-are-trying-to-stop-smoking
- Britt, D. M., Cohen, L. M., Collins, F. L., & Cohen, M. L. (2001). Cigarette smoking and chewing gum: Response to a laboratory-induced stressor. *Health Psychology, 20*, 361–368.
- CDC. (1990). Centers for Disease Control, Report, Department of Health and Human Services of U.S. Government, Atlanta, Georgia, USA.
- Centers for Disease Control and Prevention. (2016). Early release of selected estimates based on data from the national health interview survey, 2015. Retrieved from http://www.cdc.gov/nchs/data/nhis/earlyrelease/earlyrelease201605_08.pdf?version=meter+at+0&module=meterLinks&pgtype=article&contentId=&mediaId=&referrer=&priority=true&action=click&contentCollection=meter-links-click
- Chalabi, M. (2013). Cigarettes or war: Which is the biggest killer? *The Guardian*. Retrieved from <https://www.theguardian.com/news/reality-check/2013/dec/18/cigarettes-or-war-which-is-the-biggest-killer>
- Choi, W. S., Gilpin, E. A., Farkas, A. J., & Pierce, J. P. (2001). Determining the probability of future smoking among adolescents. *Addiction, 96*(2), 313–323.
- CNN. (2000). CNN media announcement via TV broadcast.
- Cohen, L. M., Britt, D. M., Collins, F. L., al'Absi, M., & McChargue, D. E. (2001). Multimodal assessment of the effect of chewing gum on nicotine withdrawal. *Addictive Behaviors, 26*, 289–295.
- Cohen, L. M., Britt, D. M., Collins, F. L., Stott, H., & Carter, L. (1999). Chewing gum affects smoking topography. *Experimental and Clinical Psychopharmacology, 7*, 444–447.
- Cohen, L. M., Collins, F. L., & Britt, D. M. (1997). The effect of chewing gum on tobacco withdrawal. *Addictive Behaviors, 22*, 769–773.
- Cohen, L. M., Collins, F. L., VanderVeen, J. W., & Weaver, C. C. (2010). The effect of chewing gum flavor on the negative affect associated with tobacco abstinence among dependent cigarette smokers. *Addictive Behaviors, 35*, 955–960.
- Cohen, L. M., McCarthy, D. M., Brown, S. A., & Myers, M. G. (2002). Negative affect combines with smoking outcome expectancies to predict smoking behavior over time. *Psychology of Addictive Behaviors, 16*, 91–97.
- Cortez-Garland, M., Cohen, L. M., VanderVeen, J. W., & Cook, K. (2010). The effect of chewing gum on self-reported nicotine withdrawal: Is it the flavor, the act of chewing, or both? *Addictive Behaviors, 35*, 224–228.
- Fiore, M. C., Bailey, W. C., Cohen, S. J., Dorfman, S. F., Goldstein, M. G., Gritz, E. R., ... & Mecklenburg, R. E. (2000). Treating tobacco use and dependence: Clinical practice guideline. US Department of Health and Human Services, 00-0032.
- Jha, P., Ramasundarahettige, C., Landsman, V., Rostron, B., Thun, M., Anderson, R. N., & Peto, R. (2013). 21st-century hazards of smoking and benefits of cessation in the United States. *The New England Journal of Medicine, 368*(4), 341–350.
- Kohrman, M., & Benson, P. (2011). Tobacco. *Annual Review of Anthropology, 40*, 329–344.
- National Center for Complementary and Integrative Health's Clinical Digest. (2014). Complementary health approaches for smoking cessation. Retrieved from <https://nccih.nih.gov/health/providers/digest/smoking>
- Orleans, C. T., Boyd, N. R., Bingle, R., Sutton, C., Fairclough, D., Heller, D., ... Baum, S. (1998). A self-help intervention for African American smokers: Tailoring cancer information service counseling for a special population. *Preventive Medicine, 27*, S61–S70.
- U.S. Department of Health and Human Services. (1964). Report of the Advisory Committee to the surgeon general of the Public Health Service. Retrieved from <https://profiles.nlm.nih.gov/ps/access/nnbbmq.pdf>
- U.S. Department of Health and Human Services. (2014). The health consequences of smoking—50 years of progress: A report of the surgeon general. Retrieved from <https://www.surgeongeneral.gov/library/reports/50-years-of-progress/index.html#execsumm>

- U.S. Department of Health and Human Services. (2016). E-cigarette use among youth and young adults: A report of the surgeon general. Retrieved from https://e-cigarettes.surgeongeneral.gov/documents/2016_sgr_full_report_non-508.pdf
- University of Massachusetts Medical School. (2007). Inhaling from just one cigarette can lead to nicotine addiction: Kids show signs of addiction almost immediately. Retrieved from www.sciencedaily.com/releases/2007/07/070703171843.htm
- Vidrine, J. I., Cofta-Woerpel, L., Daza, P., Wright, K. L., & Wetter, D. W. (2006). Smoking cessation 2: Behavioral treatments. *Behavioral Medicine*, 32(3), 99–109.
- Weinberger, A. H., Pittman, B., Mazure, C. M., & McKee, S. A. (2015). A behavioral smoking treatment based on perceived risks of quitting: A preliminary feasibility and acceptability study with female smokers. *Addiction Research & Theory*, 23(2), 108–114.
- White, A. R., Rampes, H., Liu, J., Stead, L. F., & Campbell, J. (2014). Acupuncture and related interventions for smoking cessation. *Cochrane Database of Systematic Reviews*, 1. Retrieved from <http://doi/10.1002/14651858.CD000009.pub4>
- Wilson, R. W. (1967). Cigarette smoking and health characteristics. Retrieved from http://www.cdc.gov/nchs/data/series/sr_10/sr10_034acc.pdf
- World Health Organization. (2011). WHO Report on the Global Tobacco Epidemic 2011. Retrieved from http://www.who.int/tobacco/global_report/2011/en/
- World Health Organization. (2016). *Tobacco* [Fact sheet]. Retrieved from <http://www.who.int/mediacentre/factsheets/fs339/en/>
- World Health Organization. (2017). Prevalence of tobacco smoking. Retrieved from <http://www.who.int/gho/tobacco/use/en/>

Suggested Reading

- Baker, T. B., Breslau, N., Covey, L., & Shiffman, S. (2012). DSM criteria for tobacco use disorder and tobacco withdrawal: A critique and proposed revisions for DSM-5. *Addiction*, 107(2), 263–275.
- Holford, T. R., Meza, R., Warner, K. E., Meernik, C., Jeon, J., Moolgavkar, S. H., & Levy, D. T. (2014). Tobacco control and the reduction in smoking-related premature deaths in the United States, 1964–2012. *Journal of the American Medical Association*, 311(2), 164–171.
- Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology*, 54, 25–53.

Psychological Assessment in Medical Settings: Overview and Practice Implications

Kelsey A. Maloney and Adam T. Schmidt

Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

The field of health psychology is a rapidly changing and expanding area of clinical and research focus. Skills of psychologists are increasingly being utilized in medical settings, including primary care, rehabilitation, and specialty medical clinics including oncology, cardiology, and organ transplantation. Approximately 60 medical disorders frequently encountered in primary care settings can also cause or exacerbate mental health conditions (Kush, 2001). Many primary care physicians may not have sufficient time to routinely detect, diagnose, or provide treatment for common psychological problems in their patients; moreover, many referrals that are made are not followed-up on by patients because of constraints of access, insurance coverage, or unfamiliarity with mental health assessment and intervention. Because so many common medical disorders impact mental health and vice versa, many physicians are beginning to work collaboratively with psychologists as part of a multidisciplinary treatment team in order to provide holistic, integrated care to their patients. Through the formal assessment of psychological symptoms, psychologists can help turn the focus of treatment either to or away from primary psychological issues and help streamline treatment for patients (Kush, 2001).

Anxiety disorders, followed by depressive disorders, are the most common presenting problems for mental health professionals practicing across medical settings. Both the occurrence of anxiety and depression can complicate treatment of other health conditions either by interfering with reporting of symptoms, reducing compliance with medically recommended treatments, or by contributing to behaviors that complicate the course of the medical ailment (e.g., a patient smokes to reduce stress caused by anxiety thereby exacerbating their cardiovascular disease). Not only can primary medical concerns be complicated by secondary mental health disorders, but primary mental health concerns can also be complicated with secondary medical disorders. Psychologists can provide a more thorough description of a patient's

psychosocial milieu and a better understanding of mental health concerns that may exacerbate the course of the presenting medical issue or, at the very least, complicate the treatment of this condition. Efficient and thorough psychological assessment is a key portion of the role of psychology in a medical setting. In this chapter, our goal is to present a brief overview of the specific roles for psychologists and of psychological assessment in particular within different types of medical settings in which their expertise is routinely employed.

Primary Care Settings

“Primary Care Psychologists are experts in: (a) assessment & evaluation of common psychosocial symptoms...[seen in primary care setting]; (b) [able to distinguish between symptoms associated with a medical disease and a mental health condition]; (c) collaborate with primary care teams; (d) have the knowledge to triage appropriately; (e) have an understanding of biomedical conditions commonly seen in primary care and applicable pharmacological interventions (p. 8)” (Frank et al., 2004, in James, 2006). Primary care psychologists can be helpful in screening for common psychological conditions impacting certain age groups such as autism and attention deficit hyperactivity disorder (ADHD) in pediatric settings or mild cognitive impairment in older adults, common psychiatric symptoms including depression and anxiety (Kush, 2001; Thombus et al., 2012), and screening for psychoactive substance use (Kush, 2001).

Psychologists working with primary healthcare providers may be called on to facilitate screening for disorders common in specific age groups. For example, a psychologist working with a pediatrician may administer screening measures to assess for developmental disabilities such as autism spectrum disorders in toddlers or ADHD in school-aged children. These could be direct administration of screening measures by the psychologist or, more commonly, consultation and interpretation of instruments administered by the medical staff. A positive result on one of these measures may trigger consultation with a psychologist to either perform a more in-depth assessment or to provide additional guidelines or context for the findings. Similarly, physicians working with older adults may refer for additional assessment following a physical exam in which a patient appears confused or somewhat disoriented. This model of working with primary care providers can greatly facilitate the continuum of care and help to ensure that individuals who may not otherwise be evaluated for these, relatively common, disorders are able to be screened and, if necessary, assessed in a timely fashion.

Screening for depression can be another important role for psychologists working in primary care settings. Screening for depression includes “the use of questionnaires concerning the symptoms of depression or small sets of questions about depression to identify patients who may have depression but who have not sought treatment and whose depression has not already been recognized by health care providers” (Thombus et al., 2012). Using depression rating scales can help physicians identify depression, sometimes increasing their ability to do so by 2.5- to 25-fold (Kush, 2001). Certain medical illnesses have been associated with high risk for depression, including “seizure disorders, diabetes, hypothyroidism, hyperthyroidism, Huntington’s disease, Parkinson’s disease, and cancer” (Kush, 2001). Depression in these populations can complicate treatment and exacerbate the course of the medical disorders. More general patient stressors such as disability resulting from the medical condition and/or significant psychosocial stressors (e.g., the loss of a job, grief from the loss of a loved one, or stress resulting from rigorous treatment regimens) should also trigger evaluations for depressive symptoms. A common and quick depression questionnaire that

can be used for screening in primary care settings is the Beck Depression Inventory, Second Edition (BDI-II) (Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report measure that can be completed in 5–10 min, can be scored rapidly, and can be used from age 13 to adulthood. Mean BDI-II scores “have been established for Major Depression (single episode, 28.065, recurrent 29.45), Dysthymic Disorder 24.02, Bipolar-Depressed 20.59, and Adjustment Disorder with Depressed Mood 17.29” (Kush, 2001). The BDI for Primary Care (BDI-PC) (Steer, Cavalieri, Leonard, & Beck, 1999), an abridged version, is also an option in primary care settings (Kush, 2001).

Although advantages for screening for depression has potential value, “no trials have found that patients who undergo screening have better outcomes than patients who do not when the same treatments are available to both groups” (Thombus et al., 2012). Routine screening of all patients for depression is labor intensive and can lead to misidentification of depression in some individuals, resulting in treatment for depression in patients with subclinical or mild symptoms and resources allocated away from patients who truly have depression (Thombus et al., 2012). As an alternative to screening, the National Institute for Health and Clinical Excellence 2010 guidelines recommend physicians be alert to possible depressive disorders, especially when there is (a) a previous history of depression in the target patient, (b) when patients have a chronic physical condition, and (c) when the patient has a new or worsening physical impairment that causes functional limitations, and screen for depressive symptoms when there is a specific concern such as the recent loss or change in the individual’s living situation, support network, or quality of life (Thombus et al., 2012).

Anxiety disorders have significant comorbidity with medical disorders including heart and lung diseases, fibromyalgia, and diabetes (Kush, 2001). Screening for anxiety disorders in primary care health settings is especially important in patients reporting somatic concerns including shortness of breath and chest pains. If undertaken in an efficient manner so as to not interfere with legitimate medical problems, screening can lead to reduced medical costs and improved outcomes by helping physicians make differential diagnoses and guide the selection of appropriate treatment (Kush, 2001). The Beck Anxiety Inventory (BAI) (Beck & Steer, 1993) is a 21-item self-report measure that can be completed and scored quickly. Fourteen items measure biological symptoms of anxiety, and seven items measure psychological anxiety symptoms; no items specifically measure phobias or obsessions and compulsions (Kush, 2001). Mean BAI norms have been established for “Panic Disorder with Agoraphobia ($M = 27.27$, Panic Disorder without Agoraphobia ($M = 28.81$), Social Phobia ($M = 17.77$), Obsessive-Compulsive Disorder ($M = 21.69$), and [Generalized Anxiety Disorder] ($M = 18.83$)” (Kush, 2001).

Another consideration for psychologists practicing in primary care settings where time and resources are commodities is the significant comorbidity between anxiety and depression. In settings where this is of concern, other measures that screen for both types of symptoms may be most appropriate. The Hamilton Rating Scale for Depression (HAM-D) (Hamilton, 1960) is clinician administered and is used to screen for depression; it also contains three anxiety items and can be used to assess a patient’s overlap in anxiety and depression symptoms. Both the BDI-II and BAI have shown discrimination between depression and anxiety (Kush, 2001).

In addition to internalizing concerns, drug and alcohol abuse are aspects of concern when patients present to primary care settings. According to Kush (2001), approximately 10% of adults experience significant dysfunction from alcohol or drug use at any time, yet most of these adults can present to medical appointments without any outward presentation of their substance use. Substance use and complications arising from this use can complicate the course of chronic health conditions and can attenuate the effectiveness of medical interventions. As a

result, psychologists need to incorporate screening measures of substance abuse in their standard assessment protocols for primary care patients. The Drug Abuse Screening Test (DAST-20) (Skinner, 1982) is a 20-item assessment for drug abuse over 12 months, including both prescription drug and illicit drug use, and it can be administered in approximately 5 min via self-report or interview (Kush, 2001). The Alcohol Dependence Scale (ADS) (Skinner & Horn, 1984) is a 28-item assessment for alcohol abuse over 12 months, and it can be administered in approximately 10 min via self-report or interview (Kush, 2001). In cases of suspected drug or alcohol abuse, it is important to establish a timeline of whether drug or alcohol use preceded medical problems or vice versa as this distinction may have implications for etiology, prognosis, and treatment.

Rehabilitation Settings

In addition to primary care settings, psychologists also frequently play a role in physical rehabilitation settings where patients present with a wide range of physical injuries/disabilities and co-occurring mental health concerns. Important components of psychological assessment in rehabilitation settings include the measurement of social problem solving that may impact treatment, functional impairments that may influence daily living, measures of disability status, and coping skills/behavioral adjustment (Dreer et al., 2009; Hall, 1999). Empirically supported measures of social problem solving include “the Means-End Problem Solving Procedure (MEPS; Platt & Spivack, 1975), the Problem Solving Inventory (PSI; Heppner, 1988), and the Social Problem Solving Inventory-Revised (SPSI-R; D’Zurilla, Nezu, & Maydeu-Olivares, 2002)” (in Dreer et al., 2009). These and similar tools are helpful adjuncts to treatment in rehabilitation because social problem skills can help those living with chronic health conditions or disabilities cope with psychological distress (Dreer et al., 2009).

Functional assessment measures assess disability across domains including “self-care, mobility, and, more variably, cognition, communication, and behavior” (Hall, 1999). Measures of functional disability include the Functional Independence Measure (FIM), which measures motor and cognitive functioning; the Functional Assessment Measure (FAM), which is an addition to the FIM (known together as the FIM+FAM) and adds 12 items on cognitive, behavioral, communication, and community function (Hall, 1999); and the Disability Rating Scale (DRS; Rappaport, Hall, Hopkins, Belleza, & Cope, 1982). Other measures of functional disability include length of inpatient stay, hospital or rehabilitation charges (excluding physician fees), and the intensity and type of treatment (i.e., service utilization; Hall, 1999). Finally, client satisfaction with services is an important target variable for ascertaining the quality of outcomes (Hall, 1999). These measures can help psychologists understand the limitations of their patients and provide a road map for cognitive behavioral interventions targeting specific areas of concern. Moreover, using these screening instruments can augment information gained from traditional psychological assessments by providing context as to the physical and cognitive limitations, patients may face upon returning home or during their stay at the facility.

Measures of disability status typically include analyses of community integration, service utilization, and independence. The Community Integration Questionnaire (CIQ) (Willer, Ottenbacher, & Coad, 1994) is a 15-item self-administered or interview-based questionnaire that assesses community integration for individuals with TBI across three dimensions: home integration, social integration, and productivity (Hall, 1999). The Craig Handicap Assessment and Reporting Technique (CHART) (Whiteneck, Charlifue, Gerhart, Overholser, &

Richardson, 1992) assesses five domains of “physical independence, mobility, occupation, social integration, and economic self-sufficiency” in individuals with spinal cord injury (Hall, 1999). Other measures of handicap status include employment, which can be measured by the CIQ, CHART, FIM + FAM, DRS, or a monthly employment ratio; living arrangements, which provides an estimation of an individual’s “level of dependence...and cost to society”; and service utilization (Hall, 1999). Measures of handicap status are useful additions to assessments of functional limitations as they illustrate a patient’s level of independence in the community and the extent to which their functional impairments impact their occupational and social functioning all of which may interact with mental health conditions and personality characteristics.

It is also important to assess psychosocial and behavioral adjustment in patients in rehabilitative settings (Hall, 1999). The Neurobehavioral Rating Scale (NRS) (Levin et al., 1987) is a 27-item assessment of cognitive and emotional disturbances and has been “the scale of choice for assessing behavioral changes following TBI in neurosurgical trials,” although it requires substantial training, judgment, and “familiarity with the patient” to administer (Hall, 1999). Additionally, quality of life should be assessed as it is “arguably an ultimate aim of rehabilitation”; quality of life can be assessed using the Satisfaction with Life Scale (Diener, Emmons, Larsen, & Griffin, 1985) or the Quality of Life Scale (Chubon, 1987, in Hall, 1999).

Specialty Settings

Cardiology

Anxiety and depression are the most prevalent classes of psychiatric disorders, and coronary heart disease (CHD) is a highly prevalent cardiovascular issue in the general population. Anxiety is an independent predictor of later CHD events and cardiac mortality, and depression is associated with elevated risk of later cardiac episodes (Compare, Germani, Proietti, & Janeway, 2011). “In 2008, the American Heart Association recommended [and the American Psychiatric Association endorsed] that ‘screening tests for depressive symptoms should be applied to identify cardiology patients who may require further assessment and treatment’ if appropriate referral for further depression assessment and treatment is available” (Compare et al., 2011). As patients who have anxiety or depression are at increased risk for cardiovascular complications, it is important to assess patients with cardiovascular disease, in particular, for emotional conditions that may compound their risk for adverse cardiovascular events (Compare et al., 2011).

Although general measures of anxiety and depression mentioned previously (BDI, BAI, Hamilton Anxiety Scale) can be used to assess these symptoms, there are several assessment measures for anxiety and depression that were developed for specific use in cardiovascular patient populations. The Diagnostic Interview with Structures Hamilton (DISH) (Freedland et al., 2002) is used with patients with acute coronary syndrome as a measure conducive to brief hospital visits, whereas the Maastricht Questionnaire (Appels, 1989) specifically assesses exhaustion (Compare et al., 2011). If assessment identifies patients with moderate to severe symptoms of anxiety or depression who have also either failed several trials of psychotropic medication, who have a history of previous psychiatric diagnosis, and/or who possess any one of a variety of risk factors (e.g., history of physical abuse or sexual victimization), the recommendation would be for these individuals to receive a more thorough assessment (Compare et al., 2011).

Organ Transplantation

Patients on the wait list for or who have received a donor organ are vulnerable to a variety of psychological issues. Adaptive and mental health challenges can occur at each stage of illness and treatment, including “organ failure/chronic illness, pretransplant evaluation, waiting for a donor, surgery, recovery, rehabilitation, [and] permanent maintenance (Olbrisch, Benedict, Ashe, & Levenson, 2002).” The selection process to receive a donor organ includes a process for determining a patient’s fitness to receive a donor organ such as psychological functioning and determination of any contraindications to transplantation (Maldonado et al., 2012).

As the gap between organ donation and patients awaiting transplant increases, it is becoming more important to identify potential risk factors (i.e., “substance abuse, compliance issues, serious psychopathology”) that could result in greater risk of postoperative noncompliance through the utilization of pretransplant psychological evaluations (Olbrisch et al., 2002). Psychological evaluations also help “[promote] fairness and equal access to care, [provide] a description of the patient’s neuropsychiatric and cognitive functioning, [serve] as a guide for the clinical management of the patient and [address] the psychological needs of the transplant team with regard to patient care” (Olbrisch et al., 2002). A typical pretransplant psychological evaluation consists of a clinical interview on the patient’s background and functioning, the possible use of standardized instruments, possible brief screenings for cognitive deficits such as mental status evaluations or neuropsychological measures of memory and executive functioning, collateral information (especially in patients with substance abuse history), and assessing for the quality of the patient’s support system (Olbrisch et al., 2002).

Standardized measures that can be used to assess patients as candidates for organ transplant include the Psychosocial Assessment of Candidates for Transplantation (PACT) (Olbrisch, Levenson, & Hamer, 1989), the Stanford Integrated Psychosocial Assessment for Transplantation (SIPAT) (Maldonado et al., 2012), and the Readiness for Transition Questionnaire (RTQ) (Gilleland, Amaral, Mee, & Blount, 2012) (Maldonado et al., 2012; Marchak, Reed-Knight, Amaral, Mee, & Blount, 2015; Olbrisch et al., 2002). The PACT consists of eight items on a five-point Likert scale and includes the rater’s impressions. The SIPAT consists of 18 items across 4 domains: “Patient’s Readiness Level & Illness Management, Social Support System Level of Readiness, Psychosocial Stability and Psychopathology, and Lifestyle and Effect of Substance Use” (Maldonado et al., 2012). The RTQ consists of three parallel versions—the RTQ-Provider, RTQ-Teen, and RTQ-Parent—all designed to be completed by healthcare providers (Marchak et al., 2015).

Oncology

Cancer is a significant physical health ailment in modern society with approximately 14.5 million people currently diagnosed with cancer in the United States (Stanton, Rowland, & Ganz, 2015). Diagnosis and treatment of cancer can cause significant psychological distress in patients, including elevated symptoms of depression and anxiety that can affect more than one in four patients with cancer (Stanton et al., 2015; Thalen-Lindstrom, Larsson, Glimelius, & Johansson, 2013). Stanton et al. (2015) proposed three periods during the survivorship phase—reentry (completion of treatment to up to 1 year), early survivorship (approximately 5 years post-diagnosis), and long-term survivorship (beyond 5 years post-diagnosis)—and proposed that psychological distress may be more pronounced in certain periods than in others. As a result, different assessment questions may predominate depending upon the stage of treatment a patient is in.

For cancer patients, anxiety can center on a fear of cancer recurrence. Fear of recurrence is common in long-term survivors and can be triggered by “follow-up medical visits, symptoms that mimic illness (e.g., pain that may be attributed to disease spread), death of a public figure from cancer, or a family member’s illness” (Stanton et al., 2015). Depression is also present in cancer survivors, and there is a higher risk for depressive symptoms in those with advanced cancer, receiving chemotherapy, and with more physical symptoms. However, it should be noted that at 7 years after diagnosis, cancer survivors’ rates of depression are not significantly different from healthy controls. Nonetheless, cancer treatments themselves can also result in a number of cognitive effects including impairments in attention, working memory, concentration, and executive functioning (Stanton et al., 2015).

Similar to organ transplant assessment, assessment in oncology settings can help maximize limited resources and identify patients in the most distress (Vodermaier, Linden, & Siu, 2009). Brief, self-report measures can be used to screen for oncology patients who are most in need of psychological services, and systematic screening helps promote equal access to resources better than physician- or patient-initiated referrals, which can fail to identify emotionally distressed patients (Vodermaier et al., 2009). For a detailed review of ultrashort, short, and long assessment measures that can be used as screeners for emotional distress in cancer patients, see Vodermaier et al. (2009).

The Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983) is a 14-item self-report measure for anxiety and depression, with scores greater than 10 signifying clinical cases of anxiety and depression. The HADS has been used as a screener for oncology patients in clinical settings and does increase referral rates; those referred using the HADS, however, had no difference in improvement from those in standard care (Thalen-Lindstrom et al., 2013).

Beyond anxiety and depression, cancer survivors are also likely to experience fatigue, cognitive impairment, pain, and sexual and urinary/bowel problems, as well as difficulty with more global issues such as finding benefits in their cancer experience and returning to work (Stanton et al., 2015). Anxiety and depression symptoms can be risk factors for fatigue; other risk factors for fatigue include elevated body mass, catastrophic thinking, loneliness, and early life adversity (Stanton et al., 2015). Cancer survivors often endorse problems in memory, attention, concentration, and executive function abilities; these problems can be exacerbated by risk factors such as older age, lower education, or lower IQ (Stanton et al., 2015). Therefore, in addition to psychosocial stressors and mental health symptoms, evaluations involving oncology patients should also include screening measures for cognitive functioning in order to determine if additional referrals for a comprehensive neuropsychological assessment is appropriate.

Quality of life is also an important outcome to consider for cancer survivors. Quality of life measures can include indicators of strength of interpersonal relationships and support systems, life appreciation, personal regard, and attention to health behaviors (Stanton et al., 2015; Thalen-Lindstrom et al., 2013). Although quality of life measurement is not typically included in standard psychological assessments, it is understandable how these may facilitate treatment of cancer survivors or those patients dealing with continued cancer treatment. An example measure for evaluating quality of life in oncology patients is the European Organization for Research and Treatment of Cancer (EORTC) quality of life questionnaire (QLQ-C30) (Aaronson et al., 1993) that includes five functional scales (higher scores reflect better functioning), nine symptom scales (higher scores reflect more severe problems), and a global quality-of-life (QOL) scale (higher scores reflect better functioning; Thalen-Lindstrom et al., 2013).

Conclusions

Having psychologists in medical care settings can provide useful insight into issues that arise when there is comorbidity between medical and psychological disorders in medical health settings. Psychological assessments can be used to screen for common psychological syndromes, such as depression and anxiety, autism spectrum disorders, ADHD, and dementia, and to evaluate conditions that may directly impact medical interventions such as substance abuse. The role of psychology in specialty care settings is less focused on direct diagnosis of psychological disorders (although that may continue to be a focus of screening) but rather on issues that are important for treatment and rehabilitation planning such as psychological distress, contraindications of treatment, functional impairment, behavioral/social adjustment, and coping skills. Psychological evaluations have already demonstrated usefulness in a variety of medical settings, and their use and utility will likely increase as we continue to appreciate the role of mental health in the etiology and maintenance of physical disease and the impact of positive psychological adjustment on long-term physical health and well-being.

Author Biographies

Kelsey A. Maloney is a doctoral student in clinical psychology at Texas Tech University. She received her MA from Sam Houston State University in 2016 and BS from Mississippi State University in 2014. Her research interests include the intersection of health and forensic psychology, such as improving health outcomes in juvenile justice populations.

Adam T. Schmidt is an assistant professor of clinical psychology at Texas Tech University. He received his PhD in psychology from the University of Minnesota. His research interests are in the areas of psychological and neuropsychological assessment with at risk populations and in using psychological assessment data to improve treatment outcomes.

References

- Aaronson, N. K., Ahmedzai, S., Bergman, B., Bullinger, M., Cull, A., Duez, N. J., ... Kaasa, S. (1993). The European Organization for Research and Treatment of Cancer QLQ-C30: A quality-of-life instrument for use in international clinical trials in oncology. *Journal of the National Cancer Institute*, 85(5), 365–376.
- Appels, A. (1989). Loss of control, vital exhaustion and coronary heart disease. In A. Steptoe & A. Appels (Eds.), *Stress, personal control and health* (pp. 215–235). Oxford, England: John Wiley & Sons.
- Beck, A. T., & Steer, R. A. (1993). *BAI, beck anxiety inventory manual*. San Antonio, TX: The Psychological Corporation.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI II: Beck depression inventory—Second edition manual*. San Antonio, TX: The Psychological Corporation.
- Chubon, R. A. (1987). Development of a quality-of-life rating scale for use in health-care evaluation. *Evaluation & the Health Professions*, 10(2), 186–200.
- Compare, A., Germani, E., Proietti, R., & Janeway, D. (2011). Clinical psychology and cardiovascular disease: An up- to- date clinical practice review for assessment and treatment of anxiety and depression. *Clinical Practice and Epidemiology in Mental Health*, 7, 148–156. doi:10.2174/1745017901107010148
- Diener, E. D., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment*, 49(1), 71–75.

- Dreer, L. E., Berry, J., Rivera, P., Snow, M., Elliott, T. R., Miller, D., & Little, T. D. (2009). Efficient assessment of social problem-solving abilities in medical and rehabilitation settings: A rasch analysis of the social problem-solving inventory-revised. *Journal of Clinical Psychology*, 65(7), 653–669. doi:10.1002/jclp.20573
- Freedland, K. E., Skala, J. A., Carney, R. M., Raczynski, J. M., Taylor, C. B., de Leon, C. F. M., ... Veith, R. C. (2002). The depression interview and structures Hamilton (DISH): Rationale, development, characteristics, and clinical validity. *Psychosomatic Medicine*, 64(6), 897–905.
- Gilleland, J., Amaral, S., Mee, L. L., & Blount, R. L. (2012). Getting ready to leave: Transition readiness in adolescent kidney recipients. *Journal of Pediatric Psychology*, 37(1), 85–96.
- Hall, K. M. (1999). Functional assessment in traumatic brain injury. In R. Rosenthal, E. R. Griffith, J. S. Kreutzer, & B. Pentland (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (pp. 131–146). Philadelphia, PA: F. A. Davis Company.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, 23, 56–62.
- James, L. C. (2006). Integrating clinical psychology into primary care settings. *Journal of Clinical Psychology*, 62(10), 1207–1212. doi:10.1002/jclp.20306
- Kush, K. R. (2001). Primary care and clinical psychology: Assessment strategies in medical settings. *Journal of Clinical Psychology in Medical Settings*, 8(4), 219–228. doi:10.1023/A:1011973027283
- Levin, H. S., High, W. M., Goethe, K. E., Sisson, R. A., Overall, J. E., Rhoades, H. M., ... Gary, H. E. (1987). The neurobehavioural rating scale: Assessment of the behavioural sequelae of head injury by the clinician. *Journal of Neurology, Neurosurgery & Psychiatry*, 50(2), 183–193.
- Maldonado, J. R., Dubois, H. C., David, E. E., Sher, Y., Lolak, S., Dyal, J., & Witten, D. (2012). The Stanford integrated psychosocial assessment for transplantation (SIPAT): A new tool for the psychosocial evaluation of pre- transplant candidates. *Psychosomatics Journal of Consultation and Liaison Psychiatry*, 53(2), 123–132. doi:10.1016/j.psych.2011.12.012
- Marchak, J. G., Reed-Knight, B., Amaral, S., Mee, L., & Blount, R. L. (2015). Providers' assessment of transition readiness among adolescent and young adult kidney transplant recipients. *Pediatric Transplantation*, 19, 849–857. doi:10.1111/petr.12615
- Olbrisch, M. E., Benedict, S. M., Ashe, K., & Levenson, J. L. (2002). Psychological assessment and care of organ transplant patients. *Journal of Consulting and Clinical Psychology*, 70(3), 771–783. doi:10.1037//0022-006X.70.3.771
- Olbrisch, M. E., Levenson, J. L., & Hamer, R. (1989). The PACT: A rating scale for the study of clinical decision making in psychosocial screening of organ transplant candidates. *Clinical Transplantation*, 3, 164–169.
- Rappaport, M., Hall, K. M., Hopkins, K., Belleza, T., & Cope, D. N. (1982). Disability rating scale for severe head trauma: Coma to community. *Archives of Physical Medicine and Rehabilitation*, 63(3), 118–123.
- Skinner, H. A. (1982). The drug abuse screening test. *Addictive Behaviors*, 7, 363–371.
- Skinner, H. A., & Horn, J. L. (1984). *Alcohol Dependence Scale (ADS) user's guide*. Toronto, OT: Addiction Research Foundation.
- Stanton, A. L., Rowland, J. H., & Ganz, P. A. (2015). Life after diagnosis and treatment of cancer in adulthood. *American Psychologist*, 70(2), 159–174. doi:10.1037/a0037875
- Steer, R. A., Cavalieri, T. A., Leonard, D. M., & Beck, A. T. (1999). Use of the Beck Depression Inventory for primary care to screen for major depression disorders. *General Hospital Psychiatry*, 21(2), 106–111.
- Thalen-Lindstrom, A., Larsson, G., Glimelius, B., & Johansson, B. (2013). Anxiety and depression in oncology patients; a longitudinal study of a screening, assessment and psychosocial support intervention. *Acta Oncologica*, 52(1), 118–127. doi:10.3109/0284186X.2012.707785
- Thombus, B. D., Coyne, J. C., Cuijpers, P., Jonge, P. D., DPhil, S. G., Loannidis, J. P. A., ... Ziegelstein, R. C. (2012). Rethinking recommendations for screening for depression in primary care. *Canadian Medical Association Journal*, 184(4), 413–418. doi:10.1503/cmaj.111035

- Vodermaier, A., Linden, W., & Siu, C. (2009). Screening for emotional distress in cancer patients: A systematic review of assessment instructions. *Journal of the National Cancer Institute*, 101(21), 1464–1488. doi:10.1093/jnci/djp336
- Whiteneck, G. G., Charlifue, S. W., Gerhart, K. A., Overholser, J. D., & Richardson, G. N. (1992). Quantifying handicap: A new measure of long-term rehabilitation outcomes. *Archives of Physical Medicine and Rehabilitation*, 73(6), 519–526.
- Willer, B., Ottenbacher, K. J., & Coad, M. L. (1994). The community integration questionnaire: A comparative examination. *American Journal of Physical Medicine & Rehabilitation*, 73(2), 103–111.
- Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*, 67(6), 361–370.

Suggested Reading

- Hall, K. M. (1999). Functional assessment in traumatic brain injury. In R. Rosenthal, E. R. Griffith, J. S. Kreutzer, & B. Pentland (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (pp. 131–146). Philadelphia, PA: F. A. Davis Company.
- Kush, K. R. (2001). Primary care and clinical psychology: Assessment strategies in medical settings. *Journal of Clinical Psychology in Medical Settings*, 8(4), 219–228. doi:10.1023/A:1011973027283
- Vodermaier, A., Linden, W., & Siu, C. (2009). Screening for emotional distress in cancer patients: A systematic review of assessment instructions. *Journal of the National Cancer Institute*, 101(21), 1464–1488. doi:10.1093/jnci/djp336

Complementary, Alternative, and Integrative Interventions in Health Psychology

Jeffrey E. Barnett

College of Arts and Sciences, Loyola University Maryland, Baltimore, MD, USA

Complementary and alternative treatments and interventions (widely known as CAM or complementary and alternative medicine) are composed of a wide range of healthcare practices, interventions, systems, and products that are not considered to be a part of conventional medicine and healthcare practices (e.g., National Center for Complementary and Integrative Health [NCCIH], 2015d). Complementary practices are those used along with conventional healthcare practices, and alternative practices are those used instead of conventional healthcare practices. In contrast, integrative practices involve utilizing conventional and complementary healthcare practices in a coordinated manner (NCCIH, 2015a).

While CAM interventions have received greater scientific scrutiny in recent years, many forms of CAM have been practiced for thousands of years and have rich histories of use in many cultures. Examples include Traditional Chinese medicine (TCM), which dates back to 800 BCE and Ayurveda, a whole medical system with roots in Hinduism over 10,000 years ago (Duke Center for Integrative Medicine [DCIM], 2006). At present, over 100 different interventions, products, systems, and modalities are included in widely accepted definitions of CAM (Cochrane Reviews, 2015).

Despite the historical acceptance and use of some forms of CAM, the level of support for each form of CAM varies, so a careful review of available research is needed before recommending or utilizing any CAM modalities with patients. In addition to searching for recent studies on the efficacy of various CAM modalities for the treatment of specific health conditions as well as for health promotion, a careful reading of relevant meta-analytic studies available through Cochrane Reviews is recommended (Cochrane Reviews, 2015). Additionally, the NCCIH, an institute of the National Institutes of Health, provides up-to-date information on the efficacy of various CAM modalities to include defining “through rigorous scientific

investigation, the usefulness and safety of complementary and integrative health interventions and their roles in improving health and healthcare” (NCCIH, 2015b, para. 2).

Various CAM modalities may be used to promote health as well as to treat disease. While traditional explanations for the efficacy of many CAM modalities have been questioned by the scientific community, recent research has demonstrated the effectiveness of a range of CAM interventions for a wide variety of disorders. This research has also shed light on the underlying mechanisms of many CAM modalities, helping to explain scientifically what many individuals have accepted based on religious and cultural beliefs and many years of anecdotal support.

CAM has a significant presence in the United States. Nahin, Barnes, Stussman, and Bloom (2009) found that there were over 354 million visits to CAM practitioners in 2007 with over 42 billion dollars spent on these services that year. Additionally, it is estimated that 38.3% of adults and 12% of children in the United States use at least one CAM modality each year (Barnes, Bloom, & Nahin, 2008; NCCIH, 2015a). Further, as research demonstrates the efficacy of various CAM modalities and as individuals embrace lifestyles consistent with the practice of CAM, the number individuals utilizing CAM has steadily increased.

In the most recent extensive survey of CAM practices in the United States, the National Health Interview Survey (National Institutes of Health [NIH], 2012) found that CAM practices are used to promote health, to help treat chronic conditions, and to alleviate the side effects of medications and other conventional treatments with these interventions largely being used along with conventional healthcare and not in place of it. Dietary supplements have consistently been found to be the most widely used form of CAM with 17.7% of all US adults found to be using them (NIH, 2012). While differences exist within and between groups, overall, CAM is used by individuals across the age span from a wide range of cultural backgrounds, socioeconomic levels, and educational levels.

For children and adolescents ages 4–17, the most widely used CAM approaches in order of frequency are dietary supplements and natural products; chiropractic or osteopathic manipulation; yoga, tai chi, or qi gong; deep breathing; homeopathy; meditation; special diets; massage; guided imagery; and movement therapy. These are used to promote health and wellness as well as to treat conditions (in order of frequency) such as back or neck pain, other musculoskeletal symptoms, head or chest cold, anxiety or stress, ADHD, and insomnia (NCCIH, 2015c).

In general, psychologists tend to hold positive views of CAM, both for their personal use and for their patients’ use. Additionally, many are actively integrating various CAM modalities into their treatment of patients and/or referring patients to CAM practitioners in addition to the psychological treatment they are providing (Barnett & Shale, 2012). Members of the general public also tend to hold positive views of CAM and are open to its use in their lives. Increases in the use of CAM by members of the public in recent years are attributed to factors such as dissatisfaction with conventional medicine to include its increasingly technological focus, a focus on more holistic health promotion and healthcare, and a desire to take a more active role in, and have increased control over, their health and healthcare (Stapleton et al., 2015).

The NCCIH (2015b) views CAM as being made up of five domains. These are the following:

- Whole medical systems. These include homeopathy, naturopathy, TCM, and Ayurveda.
- Mind–body medicine. These include meditation, biofeedback, hypnosis, spirituality and prayer, art therapy, music therapy, dance movement therapy (DMT), and yoga.

- Biologically based practices. These include dietary supplements, herbal supplements, and aromatherapy.
- Manipulative and body-based practices. These include massage therapy and spinal manipulation such as chiropractic and osteopathic.
- Energy therapies. These include qigong, reiki, therapeutic touch, and electromagnetic therapy.

While a wide range of CAM modalities exist, not all are commonly used, and not all receive equal support and acceptance in our nation today. Information will be shared on the 14 most widely used CAM modalities in the United States as reported by Barnes et al. (2008) and NCCIH (2015c). These are, in order of the frequency of their use from greatest to least, dietary supplements; meditation; chiropractic; aromatherapy; massage therapy; yoga; progressive muscle relaxation (PMR); spirituality, religion, and prayer; DMT; acupuncture; reiki; biofeedback; hypnosis; and music therapy. For additional information on each of these CAM modalities to include their origins, uses, relevant research, and limitations, readers are referred to the website of NCCIH at www.nccih.nih.gov.

Dietary Supplements

Dietary supplements include a wide range of typically orally ingested substances that are used to enhance general health and well-being or to help treat a wide range of ailments and health concerns. Dietary supplements include one or more of many “dietary ingredients (including vitamins; minerals; herbs or other botanicals; Amino Acids; and other substances) or their constituents” (Office of Dietary Supplements [ODS], 2011 para. 3).

While dietary supplements are regulated by the US Food and Drug Administration (FDA), they are regulated quite differently than prescription and over-the-counter medicines and vitamins. In fact, as Barnett and Shale (2012) emphasize, “the FDA does not review the safety and/or effectiveness of any supplements prior to them being sold” (p. 577). Additionally, for many supplements, there can be significant differences between brands and even between batches within brands for the potency, purity, type, and quantity of the active ingredients and other substances included in them. Additionally, while many dietary supplements are marketed as “natural products,” the use of some of them, both alone and in combination with other products, can result in harmful effects and side effects. Yet, 17.7% of adults use herbal products, and when including vitamins and minerals, 52% of adults are using dietary supplements (Radimer et al., 2004).

Psychologists should not make recommendations to patients about the use of dietary supplements and should not integrate their use into psychological treatment. Yet, it is recommended that psychologists ask all patients about their use of dietary supplements (as well as their use of prescription and over-the-counter medications). While many of these substances may be quite helpful, there are numerous significant health risks associated with their use and misuse. Additionally, when certain herbal supplements are taken along with widely prescribed medications, serious side effects are common. Therefore, it is recommended that psychologists keep informed about the literature on the use of dietary supplements and educate patients about their possible use and attendant risks. All patients currently using, considering using, or who in the psychologist’s view may benefit from the use of dietary supplements (as well as prescription and over-the-counter medications) should be referred to their primary care physician to oversee and/or recommend their use to help ensure optimal outcomes are achieved.

Meditation

Meditation is a mind–body practice that has long been integrated into a wide range of religious and spiritual practices. The most widely used and studied forms of meditation at present are mindfulness meditation and concentrative meditation. Mindfulness meditation involves paying attention in a nonjudgmental way in the present moment (Kabat-Zinn, 1994). It has formed the basis of integrative treatments such as mindfulness-based stress reduction (MBSR) and mindfulness-based cognitive therapy (MBCT) and is a component of acceptance and commitment therapy (ACT) and dialectical behavior therapy (DBT). Concentrative meditation involves focusing on a specific word or phrase to alter one’s conscious state and to achieve a deep state of relaxation. In the most commonly used and studied form of concentrative meditation, Transcendental Meditation, it “allows the mind to simply, naturally and effortlessly transcend thinking and to experience a deep state of restfully alert consciousness” (e.g., Nagendra, Maruthai, & Kutty, 2012).

With appropriate training and competence, meditation practices may be integrated into the treatment of patients with a wide range of presenting problems. Patients may be referred to various commercially available meditation programs to augment the work they are doing in psychotherapy, meditation practices may be integrated into ongoing psychotherapy, and meditation-based treatments such as MBSR and MBCT may be offered.

Numerous studies have demonstrated the usefulness of meditation practices for the treatment of conditions such as high blood pressure, irritable bowel syndrome, ulcerative colitis, anxiety, depression, insomnia, and acute respiratory illnesses, and initial findings indicate some promise for pain management and for smoking cessation (NCCIH, 2015e). For example, in a randomized controlled study, Barnhofer et al. (2015) demonstrated the effectiveness of mindfulness approaches in reducing depressive and suicidal cognitions. While no significant contraindications or side effects are reported with the use of meditation practices, as with all healthcare interventions, consultation with each patient’s primary care physician before recommending or integrating meditation into psychological treatment is recommended.

Chiropractic

Chiropractic is a system of spinal adjustments or manipulations that are performed to realign the spine in a manner that is believed to relieve pain and to promote health and wellness. Chiropractic is most widely used to treat back pain, neck pain, pain in the joints of the arms and legs, and headaches (American Chiropractic Association [ACA], 2015). Chiropractic has a long history dating back to 2700 BCE in ancient China and Greece (ACA, 2015). There exists a range of techniques used by in chiropractic treatment, but their primary aim is to realign the spine to reduce or correct spinal nerve impingement and to improve joint mobility. Chiropractic manipulation may be provided as a stand-alone treatment or in conjunction with conventional healthcare interventions.

Research has been conducted on the use of chiropractic for acute low back pain, chronic low back pain, and headaches with moderate to significant effectiveness being found, depending on the studies reviewed. It has also been studied for the treatment of phobias, ADHD, and nocturnal enuresis, with questionable effectiveness being found (Barnett, Shale, Elkins, & Fisher, 2014).

Chiropractic is a profession that is regulated by state law. Only licensed doctors of chiropractic may offer these treatments in the United States. Thus, if chiropractic care is to be considered for patients, referral to a licensed doctor of chiropractic is essential. But these professionals may use a wide range of techniques, with some offering nutritional counseling and other services, so being clear on their training, areas of competence, and techniques used is essential. Additionally, due to the very forceful physical nature of chiropractic spine manipulations, side effects such as muscular soreness are common (Ernst, 2007).

Aromatherapy

Aromatherapy is the use of essential oils from herbs, flowers, and trees to promote physical, emotional, and spiritual health and wellness. Proponents of aromatherapy believe that the inhalation of various essential oils from plants positively influences “mood, behavior, and spiritual well-being by stimulation of the olfactory or somatic senses” (Barnett et al., 2014, p. 181). While aromatherapy has a long history of use dating back to ancient Egypt, China, and India as parts of whole medical systems such as TCM and Ayurveda, its use and effectiveness have only recently begun to be studied.

Aromatherapy may be used to treat specific symptoms and disorders such as stress, anxiety, insomnia, and pain (Chalifour & Champagne, 2008). It has also been used holistically to promote overall wellness and for esthetic purposes such as to promote relaxation and to create a pleasing and peaceful home environment. Reviews of controlled studies do not yield significant findings for the treatment of any disorders, yet many individuals report that smelling the aroma of a range of plant extracts helps them to feel more relaxed and peaceful.

Aromatherapy should be used with caution as there are serious side effects that may result from applying essential oils in an incorrect manner or with incorrect doses. Additionally, some essential oils are toxic and should be avoided completely. Further, some essential oils are contraindicated with pregnant women and children. Thus, the possible use of aromatherapy should be given careful consideration that includes a review of the relevant literature and appropriate oversight for any aromatherapy use.

Massage Therapy

Massage therapy is a manipulative and body-based practice that involves “pressing, rubbing, and moving muscles and other soft tissues of the body, primarily by using the hands and fingers” (Barnett et al., 2014, p. 210). It is primarily used as a means of reducing stress and promoting relaxation and to reduce muscular tightness and strain. It has also been used in the rehabilitation of sport-related injuries and to improve mood and reduce anxiety and depression as well as to promote overall wellness. Research findings indicate strong support for the use of massage therapy in the treatment of anxiety, depression, and stress, with moderate effectiveness in the treatment of low back pain and neck pain. Massage therapy has also been shown to positively impact immune functioning for individuals with breast cancer, HIV, and leukemia (Field, 2006).

There are numerous massage therapy techniques that are commonly used. Additionally, there are four main forms of massage presently being practiced in the United States. These are Swedish, deep tissue, sports, and trigger point massage. Massage therapy is regulated in the

United States, and referrals for massage therapy should only be to those who are licensed or certified. Care should be taken to ensure that when massage is included in patients' overall treatment, it is done so with appropriate oversight. While few side effects or contraindications are reported in the literature, it is recommended that massage therapy only be used after clearance is received from the patient's primary care physician. Some patients with musculoskeletal difficulties or injuries should not participate in massage therapy.

Yoga

Yoga is a form of CAM with ancient roots that has become increasingly popular in recent years. There are a large number of yoga "schools" with each emphasizing different techniques and goals, with different styles existing within each school. But, overall, yoga involves physical postures, meditation, breathing exercises, and sustained concentration to promote wellness, relaxation, strength, and flexibility. The most widely practiced form of yoga in the United States is Hatha yoga with some of its popular styles including Bikram, Ashtanga, Iyengar, and Kundalini yoga.

Yoga is practiced by more than 13 million adults in the United States; 58% practice it to promote overall health and well-being, 16% to treat specific medical conditions such as low back pain, and 11% to seek relief from musculoskeletal conditions. Yet, only 22% reported that their physician recommended the use of yoga (NCCIH, 2015e). Thus, it is likely that many individuals are seeking out the practice of yoga on their own initiative.

Psychologists may recommend yoga for a wide range of health concerns. Strongest support has been found for yoga's use in the treatment of pain and to promote coping skills, with weak evidence for its use in treating depression, anxiety, and eating disorders. Yet, it is widely used to enhance wellness and quality of life overall, with many individuals reporting significant benefits from its use.

While few risks are associated with the use of yoga, not all forms of yoga are suitable for all individuals. Each patient's needs should be assessed, the different forms of yoga researched, and then an individual decision made. Before engaging in some of the more strenuous forms of yoga, clearance by the patient's physician is recommended.

Progressive Muscle Relaxation

PMR is a widely used CAM modality that is now so widely accepted for use in psychological treatment that it soon may not be considered complementary or alternative. PMR is a technique in which the patient learns to progressively relax different muscle groups throughout the body. Its use may also incorporate guided imagery and deep breathing exercises. Psychologists may easily integrate PMR into ongoing psychological treatment, and its use is quite frequently taught in graduate training for psychologists. No additional licensure or certification is needed to teach patients PMR. Scripts and recordings of PMR instructions are widely available online and may be utilized by psychologists and patients alike.

Research indicates that PMR can be used quite effectively to treat anxiety, muscular tension, and other stress-related symptoms to include tension headaches, neck ache, and back pain. PMR is useful for promoting general relaxation and as a component of the treatment of a wide range of stress-related disorders. PMR may be utilized in conjunction with other forms

of CAM such as meditation, yoga, massage therapy, and biofeedback in the treatment of stress-related disorders.

Spirituality, Religion, and Prayer

While many mental health professionals may not consider religion, spirituality, and prayer as relevant to the provision of psychological services, for many individuals who seek out psychological treatment, these are important parts of their lives. In fact, many patients' suffering and distress may be related to religious or spiritual issues or concerns. For others, religion, spirituality, and prayer may be important sources of strength and support that can be tapped into to assist them in treatment.

Spirituality, religion, and prayer are commonly incorporated into the treatment of addictions, trauma, and depression, but they may be utilized in a wide range of treatments to assist patients to achieve their goals. They may also be useful for helping patients find meaning in their suffering, for enhancing coping ability and resilience, and to promote general well-being. Research support is found for the use of prayer as a coping mechanism for patients with cancer or other terminal illnesses, to treat pain, and for stress reduction (Barnett et al., 2014).

Psychologists should be cautious about imposing personal religious beliefs on patients or not valuing and respecting patients' beliefs and preferences. Further, for those psychologists who will integrate prayer and other religious or spiritual interventions into psychological treatment, care must be exercised not to take on the role of the clergy. When concerns exist, consultation with, and referrals to, members of the clergy are recommended.

Dance Movement Therapy

DMT is a mind–body practice that incorporates dance and movement and that views motion and emotion interconnected, using expressive movement to assist patients to address unresolved emotional conflicts. The dance movement therapist analyzes the patient's movements to better understand the patient's underlying emotional drives and conflicts and, with this deeper understanding, assists the patient to work through and resolve them.

DMT has been used to treat anxiety, distorted body image, depression, and fatigue and to promote psychological adjustment and quality of life. Well-designed studies are uncommon in the literature, and support for DMT is predominantly anecdotal (Barnett et al., 2014). Available research on the effectiveness of DMT indicates some support for its use in the treatment of the abovementioned issues, but due to methodological issues, caution should be exercised in recommending its use.

Acupuncture

Acupuncture is a component of TCM that has been in use for thousands of years. In TCM it is believed that each individual's life energy, or *qi* (pronounced *chi*), flows through 16 pathways throughout the body called *meridians*. Acupuncturists place needles in combinations of some of the 360 specific *acupoints* along these meridians and stimulate them either by manually manipulating the needles or with electrical stimulation, with the goal of rebalancing the life energy and thus ameliorating the symptoms being experienced (DCIM, 2006).

In recent years acupuncture has been studied extensively with well-designed studies and has been found to be moderately effective for the treatment of chronic low back pain, cancer-related pain, anxiety, migraines and headaches, and insomnia. While it has also been used for the treatment of depression and schizophrenia, support for these uses is quite limited (Barnett et al., 2014). While some may question traditional beliefs about acupuncture's underlying mechanisms, recent research provides support for the stimulation of nerve endings that result in changes in the brain's pain centers (White & Ernst, 2004).

Acupuncture is found to generally to be safe with minimal side effects when provided by a trained and competent practitioner, and it has fewer side effects than many conventional treatments for chronic low back pain (Thomas et al., 2005). Acupuncture should only be provided by a trained and credentialed acupuncture professional. In many states licensure and/or certification is required to provide acupuncture, and each practitioner's credentials should be assessed before making referrals.

Reiki

Reiki is a form of energy therapy in which the practitioner transfers energy, or life force, into the patient by placing her or his hands slightly above or lightly on the patient's body. By passing this energy to the patient at a series of known hand positions, it is believed that this energy transfer promotes healing in the participant by allowing her or his own life force to flow more freely (Barnett et al., 2014). While limited research has been conducted on the use and effectiveness of reiki, evidence exists that demonstrates some benefit in the treatment of depression and anxiety and for relieving stress and promoting general wellness. It has also been shown to reduce pain, fatigue, and insomnia in cancer patients.

No known side effects or risks are associated with the use of reiki, but due to the limited research support for it at present, caution should be taken when recommending it, and reiki should only be used as a complementary treatment and not a replacement for well-researched treatments. Reiki practitioners must be certified, yet at present there are numerous certification agencies with no single standard for training and for demonstrating competence. Thus, the competence of reiki practitioners should be carefully assessed when considering making referrals.

Biofeedback

Biofeedback is another CAM modality that has been extensively studied and that has been widely integrated into psychological practice. As such, many practitioners are now considering biofeedback to be a component of conventional psychological practice. Biofeedback involves utilizing one of several possible monitoring devices to provide feedback to the patient on underlying physiologic processes such as respiration, heart rate, muscle tension, skin temperature, and brain wave activity. By use of the ongoing feedback, patients learn to impact and control these underlying physiologic processes to reduce or ameliorate symptoms associated with them.

Extensive research has demonstrated biofeedback's effectiveness in treating tension headaches, migraines, Raynaud's disease, anxiety, and ADHD. Some support is also found for its use in treating fibromyalgia, phantom limb pain, and depression (Barnett et al., 2014). There are no known side effects of biofeedback, and its practitioners provide it within the scope of

their professional license when appropriately trained. Optional board certification is available through the Biofeedback Certification International Alliance (see www.bcia.org) that also maintains a list of board certified professionals for use when making referrals. Biofeedback is widely used and regularly integrated into treatment provided by appropriately trained psychologists.

Hypnosis

Hypnosis is a mind–body therapy that also has been extensively studied and widely integrated into conventional healthcare by appropriately trained practitioners. Hypnosis, also known as hypnotherapy, involves a hypnotic induction that places the patient into an altered state of consciousness during which the patient is receptive to suggestions that assist the patient to achieve desired goals and symptom reduction. While some individuals are more hypnotizable than others, most individuals are able to benefit from its use. Hypnosis has been utilized as a stand-alone treatment as well as integrated with other healthcare practices. There is strong research support for the use of hypnosis to treat acute and chronic pain and as an analgesic during medical and surgical procedures. It has also been shown effective in the treatment of irritable bowel syndrome and in the reduction of the side effects of chemotherapy. While it has been used for smoking cessation and other types of habit control, the evidence for these uses is more limited (Barnett et al., 2014).

When offered by competent professionals, side effects from the use of hypnosis are quite rare. Most professionals who utilize hypnosis do so within the scope of their licenses as health professionals. Yet, board certification is available through the American Society of Clinical Hypnosis (www.asch.net), and this should be considered when making referrals. Appropriately trained psychologists may easily integrate the appropriate use of hypnosis into their patients' treatment.

Music Therapy

Music therapy involves the use of music appreciation, creation, and performance for emotional expression. As a mind–body intervention, music therapy may assist patients to explore and express their thoughts and feelings through music, with the goal of this process being improved health.

Music therapy has been used in the treatment of depression, anxiety, chronic pain, PTSD, chemotherapy side effects, dementia, and schizophrenia. While mixed results have been seen thus far, music therapy does appear to be at least somewhat helpful in the treatment of depression and anxiety, and for promoting general relaxation in response to various stressors. Thus, it may be useful when integrated with other treatments. Music therapy practitioners should be certified by the American Music Therapy Association (www.musictherapy.org).

Conclusion

A wide range of CAM modalities exist that have received strong scientific support for the treatment of a wide range of conditions and disorders either alone or in conjunction with other treatments. Yet, not all CAM modalities are helpful to all patients, and some modalities

may prove harmful if not applied appropriately. One must possess the education and training necessary to be knowledgeable about CAM to make the best possible treatment decisions for patients. Some CAM modalities may effectively be integrated into conventional treatments by psychologists, and for others, referrals to competent and appropriately credentialed CAM practitioners should be made. With so many individuals utilizing CAM on their own and with so many patients open to, or even requesting, the use of CAM in their psychological treatment, it is important that psychologists educate themselves about the use, benefits, limitations, and contraindications of CAM in psychological practice.

Author Biography

Jeffrey E. Barnett, PsyD, ABPP, is a licensed psychologist as well as associate dean and professor of psychology at Loyola University Maryland. He is board certified by the American Board of Professional Psychology in clinical psychology and in Clinical Child and Adolescent Psychology, and he is a distinguished practitioner in psychology of the National Academies of Practice.

References

- American Chiropractic Association. (2015). What is Chiropractic? Retrieved from http://www.acatoday.org/level2_css.cfm?T1ID=13&T2ID=61
- Barnes, P. M., Bloom, B., & Nahin, R. (2008). Complementary and alternative medicine use among adults and children: United States, 2007. In *National Health Statistics Reports* (Vol. 12, pp. 1–23). Atlanta, GA: Centers for Disease Control and Prevention.
- Barnett, J. E., & Shale, A. J. (2012). The integration of complementary and alternative medicine (CAM) into the practice of psychology: A vision for the future. *Professional Psychology: Research and Practice*, 43, 576–585.
- Barnett, J. E., Shale, A. J., Elkins, G., & Fisher, W. I. (2014). *Complementary and alternative medicine for psychologists: An essential resource*. Washington, DC: American Psychological Association.
- Barnhofer, T., Crane, C., Brennan, K., Duggan, D. S., Crane, R. S., Eames, C., ... Williams, J. M. G. (2015). Mindfulness-based cognitive therapy (MBCT) reduces the association between depressive symptoms and suicidal cognitions in patients with a history of suicidal depression. *Journal of Consulting and Clinical Psychology*, 83(6), 1013–1020.
- Chalifour, M., & Champagne, T. (2008). Aromatherapy. Retrieved from <http://www.ot-innovations.com/content/view/35/46/>
- Cochrane Reviews. (2015). Complementary and Alternative Medicine Reviews in Cochrane Library through Issue 8, 2014. Retrieved from <http://www.compmed.umm.edu/cochrane/reviews.asp>
- Duke Center for Integrative Medicine. (2006). *The Duke encyclopedia of new medicine: Conventional and alternative medicine for all ages*. London, UK: Rodale Books International.
- Ernst, E. (2007). Adverse effects of spinal manipulation: A systemic review. *Journal of the Royal Society of Medicine*, 100, 330–338.
- Field, T. (2006). *Massage therapy research*. New York, NY: Elsevier.
- Kabat-Zinn, J. (1994). *Wherever you go, there you are*. New York, NY: Hyperion.
- Nagendra, R.P., Maruthai, N., & Kutty, B.M. (2012, April 18). Meditation and its regulatory role on sleep. *Published online: Frontiers of Neurology*, vol. 3, p. 54. PMID: 22529834.
- Nahin, R. L., Barnes, P. M., Stussman, B. J., & Bloom, B. (2009). Cost of complementary and alternative medicine (CAM) and frequency of visits to CAM practitioners, 2007. In *National Health Statistics Reports No. 18* (pp. 1–14 (DHHS Publication No. 2009-1250)). Hyattsville, MD: National Center for Health Statistics.

- National Center for Complementary and Integrative Health (NCCIH). (2015a). Health Info. Retrieved from <https://nccih.nih.gov/health/integrative-health>
- National Center for Complementary and Integrative Health (NCCIH). (2015b). NCCIH Facts at a Glance. Retrieved from <https://nccih.nih.gov/about/atag glance>
- National Center for Complementary and Integrative Health (NCCIH). (2015c). Children and Complementary Health Approaches. Retrieved from <https://nccih.nih.gov/health/children>
- National Center for Complementary and Integrative Health (NCCIH). (2015d). Health Topics A-Z. Retrieved from <https://nccih.nih.gov/health/atoz.htm>
- National Center for Complementary and Integrative Health (NCCIH). (2015e). Yoga as a Complementary Health Care Approach. Retrieved from <https://nccih.nih.gov/news/multimedia/infographics/yoga>
- National Institutes of Health. (2012). National Health Interview Survey. Retrieved from <https://nccih.nih.gov/research/statistics/NHIS>
- Office of Dietary Supplements. (2011). Dietary supplements: Background information. Retrieved from <http://ods.od.nih.gov/factsheets/DietarySupplements-HealthProfessional/>
- Radimer, K., Bindwald, B., Hughes, J., Ervin, B., Swanson, C., & Picciano, M. F. (2004). Dietary supplement use by US adults: Data from the National Health and Nutrition Examination Survey, 1999–2000. *American Journal of Epidemiology*, 160, 339–349.
- Stapleton, P., Chatwin, H., Boucher, E., Crebbin, S., Scott, S., Smith, D., & Purkis, G. (2015). Use of complementary therapies by registered psychologists: An international study. *Professional Psychology: Research and Practice*, 46, 190–196.
- Thomas, K. J., MacPherson, H., Radcliffe, J., Thorpe, L., Brazier, J., Campbell, M., ... Nicholl, J. P. (2005). Longer term clinical and economic benefits of offering acupuncture care to patients with chronic low back pain. *Health Technology Assessment*, 9, 1–109.
- White, A., & Ernst, E. (2004). A brief history of acupuncture. *Rheumatology*, 43, 662–663.

Resources

NCCIH Clearinghouse
nccih.nih.gov
 PubMed®
www.ncbi.nlm.nih.gov/pubmed
 MedlinePlus
www.medlineplus.gov
 Cochrane Database of Systematic Reviews
www.cochranelibrary.com

Hypnosis and Health Psychology

Steven Jay Lynn¹, Craig P. Pollizi¹, Joseph P. Green²,
Damla E. Aksen¹, Ashwin Gautam¹, and James Evans¹

¹*Department of Psychology, Binghamton University, Binghamton, NY, USA*

²*Department of Psychology, Ohio State University at Lima, Lima, OH, USA*

From Mesmer's early ventures in using suggestion to alleviate physical and psychological distress in the eighteenth century to the use of hypnosis to facilitate surgery prior to effective anesthetics, and extending to the present, hypnosis has played an increasingly prominent role in treating medical conditions and in ameliorating pain and suffering (e.g., Flammer & Bongartz, 2003). As hypnosis has moved into the mainstream of psychological science and clinical practice, there is increasing recognition that hypnosis can be used as a stand-alone intervention and, more commonly, as an adjunct intervention in the field of health psychology. This review will feature qualitative reviews, meta-analyses, and randomized controlled trials (RCTs) that have documented the promise of hypnosis in treating many health-related conditions, ranging from acute and chronic pain and obesity to irritable bowel syndrome.

Applications of hypnosis to the field of health psychology have been spurred by research that has debunked many popular myths about hypnosis and has, instead, documented that hypnosis is not a special trance or sleep-like state in which participants respond in robot-like fashion to suggestions delivered by a powerful hypnotist, lose awareness of their surroundings, and spontaneously forget what transpired during hypnosis. Rather, participants who experience hypnotic suggestions retain their ability to resist or oppose suggestions; are aware of what occurs before, during, and after hypnosis; and achieve therapeutic gains not because of a trance but because merely defining the context as hypnosis can catalyze motivation to experience suggestions fully and bolster positive treatment expectancies relevant to successful clinical outcomes.

An Introduction to Clinical Hypnosis

Hypnosis—defined as any set of suggestions that are either administered to participants in a context that is presented as “hypnosis” or participants understand to be “hypnosis”—provides

health psychologists with tremendous leverage in crafting suggestions that are individually tailored or can be administered in manualized interventions. Hypnosis traditionally involves a so-called induction that can be virtually any array of verbal (e.g., suggestions for relaxation) and nonverbal communications (e.g., suggestions delivered in a calm, soothing manner) presented by the person in the role of the hypnotist that define or establish the proceedings as “hypnosis.” Inductions can range from highly ritualized methods (e.g., the proverbial “swinging watch” popularized in the media) to more naturalistic conversations to experience an increasingly deep hypnotic state. Hypnotic inductions typically contain imaginative suggestions for relaxation, focusing on suggested events, and deepening experiential involvement in suggestions. The attention individuals receive from the hypnotist, accompanied by the soothing, calming way hypnotists often converse with participants, can promote a close therapeutic alliance, bolster motivation to respond, and diminish anxiety. After the induction, suggestions often hone in on the participant’s problem or condition and can be tailored to address specific patient needs.

Given the prevalent views that hypnosis can produce positive outcomes, the very idea of undergoing hypnosis can engender positive expectancies for treatment gains. The context of hypnosis lends itself well to adaptation to a health psychology setting because participants can detach themselves from everyday concerns and attend to therapeutic suggestions and helpers can talk to patients in deeply personal ways and exhort participants to modify deeply ingrained cognitive behavioral patterns and to comply with medical recommendations and treatment regimens.

The experience of hypnosis depends much more on the motivation and willingness of participants to think and imagine along with suggestions, and the nature of the suggestions themselves, rather than on any special skills of the hypnotist. Accordingly, hypnosis can justifiably be presented to individuals as self-hypnosis, and participants can learn to “self-administer” suggestions apart from the hypnotist, which can increase the “portability” of hypnosis and generalize treatment gains to everyday life. Additionally, so-called posthypnotic suggestions for comfort and pain relief, for example, can extend the benefits of hypnosis well beyond the consulting room. Such suggestions may be reinforced with tapes or DVDs that contain therapeutic suggestions that the patient can use to revivify and reinstate key points and elements of the hypnotic proceedings.

Hypnotic Suggestibility

In many health-related interventions, hypnotic suggestibility does not play a prominent role. Although about 15% of people are low or non-suggestible and pass no or few suggestions, and a comparable percentage of people are highly suggestible and pass most suggestions, the majority of individuals (approximately 70%) can respond to a variety of suggestions. Many of the suggestions (e.g., relaxation, focused attention) delivered in the context of medical interventions do not require a high level of suggestibility. Even people who score relatively low on standardized scales of hypnotic suggestibility can benefit from hypnotic suggestions. In the case of hypnotic treatment of clinical pain, for example, there is only a weak association of hypnotic suggestibility and pain relief.

Although a formal hypnotic induction increases suggestibility by approximately 10–20%, compared with identical waking suggestions administered in a nonhypnotic context, the addition of hypnosis can provide incremental benefits beyond nonhypnotic interventions. Kirsch, Montgomery, and Sapirstein’s (1995) meta-analysis documented the ability of hypnosis to

enhance treatment gains of psychotherapeutic approaches that span psychodynamic and cognitive behavioral psychotherapies. Another meta-analysis provided preliminary evidence for the use of hypnosis in treating numerous psychosomatic disorders (e.g., conversion disorder, gastrointestinal problems, enuresis, asthma; Flammer & Alladin, 2007).

Applications of Hypnosis in Health Psychology

Pain Relief

Hypnosis exerts its effects largely via altering subjective experience and therefore impacts perceptions of pain. The effects of hypnotic analgesia are reflected in brain and spinal cord functioning and vary as a function of the suggestions administered (Jensen & Patterson, 2014). In addition to suggestions for relaxing and imagining a “favorite place” of comfort, calmness, and soothing, for example, suggestions for easing pain can involve reinterpreting pain sensations (e.g., as pressure), distraction (e.g., watching absorbing scenes on a mental television or movie or reliving positive past events), and transforming sensations and perceptions (e.g., imagining pain as a color and suggesting the patient transform the color).

Meta-analyses (Montgomery, DuHamel, & Redd, 2000) have found that (a) hypnosis produces moderate-to-large effects in relieving both clinical pain (e.g., coronary disease, cancer, headache, burns) and experimental pain (e.g., pressure, ischemic pain, cold pressure) and that hypnosis produced salutary effects for 75% of the population and (b) hypnosis is associated with greater pain reductions and better recovery outcomes in adults undergoing surgical or medical procedures compared with standard care alone and attention control interventions (Tefikow et al., 2013). Montgomery, David, Winkel, Silverstein, and Bovbjerg (2002) documented that surgical patients undergoing hypnosis treatment experienced better clinical outcomes (e.g., reduced emotional distress, pain medication, recovery time) than 89% of patients in comparison groups. Moreover, hypnosis has proved useful in reducing pain associated with needle procedures (Birnie et al., 2014) and in treating fibromyalgia, compared with control interventions (e.g., relaxation, pharmacological treatment, usual care, wait list; Bernardy, Klose, Busch, Choy, & Häuser, 2012). In a RCT of a 15-min hypnosis intervention administered preoperatively to women undergoing excisional breast biopsy or lumpectomy, hypnosis was superior to attention control, as reflected in decreased medication use; reports of pain, nausea, fatigue and discomfort; emotional upset at discharge time in surgery; and institutional cost savings (\$772.71, on average, per patient). Importantly, hypnosis appears to reduce anxiety associated with medical conditions, as a meta-analysis has revealed in cancer patients (Chen, Liu, & Chen, 2017).

Qualitative reviews (e.g., see Elkins, 2016; Elkins, Jensen, & Patterson, 2007; Jensen & Patterson, 2014) confirm that hypnosis (a) is associated with decreases in chronic pain conditions (e.g., arthritis, sickle cell disease, cancer, low back pain, headaches) and (b) produces salutary effects in reducing nausea and vomiting associated with cancer and (c) that hypnosis is generally more efficacious in reducing chronic pain compared with attention control, physical therapy, and psychoeducation. Moreover, hypnosis can be combined with virtual reality to relieve pain across the age spectrum and that hypnosis outperforms attention placebo in reducing pain in burn wound debridement (see Elkins, 2016). Hypnosis for pain control and modulation is the best established of all applications of hypnosis in the field of behavioral health psychology.

Irritable Bowel Syndrome (IBS)

Meta-analyses, RCTs, and narrative reviews document the promise of hypnosis in treating IBS, even among medically treated refractory patients, and that the benefits of hypnosis are not limited to short-term success. Gonsalkorale, Miller, Afzal, and Whorwell (2003) followed over 200 patients consecutively treated with hypnosis for IBS and reported that 71% of patients responded positively to treatment and that over 80% of these patients maintained their treatment gains over 5 years. Nevertheless, a Cochrane Review (Webb, Kukuruzovic, Catto-Smith, & Sawyer, 2007) tempered these positive conclusions in indicating that the evidence base, while promising, requires additional rigorous studies to permit more definitive conclusions regarding the effectiveness of hypnosis in treating IBS.

Obesity and Weight Loss

Hypnosis produces weight loss when combined with cognitive behavioral interventions. Milling, Gover, and Moriarity (2018) reported two meta-analyses, which revealed that hypnosis is a highly promising treatment for obesity, especially when incorporated into cognitive behavioral therapy for weight loss. The average participant reported greater weight reductions than about 94% of participants in control conditions at the conclusion of treatment and 81% of control participants at follow-up. The average individual who received CBT combined with hypnosis lost more weight than 60% of participants that received CBT alone at the end of treatment and 79% of individuals that received only CBT at follow-up. The reviewers suggested that studies of long-term effects of hypnosis on weight loss are needed, on the order of 1–5 years, to determine the persistence of treatment effects over more lengthy time intervals.

Smoking Cessation

Hypnosis shows promise in treating smoking. Meta-analyses indicate that hypnotic interventions are associated with quit rates on average of 31% for men and 23% for women (Green, Lynn, & Montgomery, 2008). Nevertheless, a 2010 Cochrane Review (Barnes *et al.*, 2010) reported considerable heterogeneity of findings and that the claims regarding hypnosis for smoking cessation based on case studies extended beyond evidence secured from relatively few RCTs reported in the literature. Clearly, more RCTs that include biochemical measures to verify self-reports of smoking cessation are warranted.

Other Applications

More preliminary yet promising findings indicate that hypnosis can be effective in treating a variety of other medical conditions, such as skin disorders, asthma, insomnia, hot flashes, and hypertension, and be useful in palliative care and in assisting women in labor, as summarized in Elkins (2016). Hypnosis awaits further evaluation before more confident conclusions can be drawn.

Conclusions

The evidence for hypnosis for treating pain-related and medical conditions is promising, although findings across studies are mixed. Additionally, firm conclusions are limited by the lack

of adequate follow-up periods and assessments and the difficulty of disentangling hypnosis-specific effects from nonspecific treatment variables, including placebo/expectancy effects, a positive therapeutic alliance, and participating in a symptom-relief endeavor. It is important to distinguish the effects of “hypnosis” from adjunctive cognitive behavioral strategies and to control for placebo/expectancy effects, naturally occurring changes in symptoms, and relaxation and visualization. Further, the effects of hypnosis remain to be disambiguated from the effects of suggestion alone. Additionally, as the response to hypnosis is variable, researchers would do well to evaluate hypnotic suggestibility and to also ensure that studies use naïve raters of treatment effects and that participants are adequately randomized to conditions in research with samples adequate to discern clinically meaningful treatment outcomes. Finally, the mechanisms by which suggestion exerts salutary effects have yet to be well established.

In conclusion, hypnosis is a brief, cost-effective, and popular intervention that can be easily learned to administer by a medical or mental health professional. A steadily accumulating literature, marked by studies of increasing rigor and sophistication, indicates that hypnosis can be employed as a vehicle to administer “direct suggestions” in the context of pain or as an adjunctive intervention to augment the effects of cognitive behavioral and other interventions for diverse medical conditions. Again, the positive effects of hypnosis in a health or medical setting are neither the product of a trance nor unique to hypnosis, but are associated with variables that likely mediate the effectiveness of many nonhypnotic interventions, including positive treatment-relevant expectations, attitudes, and beliefs; a viable therapeutic alliance; goal-directed motivation; and suggestions that target processes (e.g., relaxation for stress management) that are directly or secondarily related to medical symptoms and conditions.

Author Biographies

Steven Jay Lynn is Distinguished Professor of Psychology (SUNY) at Binghamton University. He received his bachelor's degree in psychology from the University of Michigan and his PhD in psychology (clinical) from Indiana University. He is the editor of *Psychology of Consciousness: Theory, Research, and Practice*, and he is on the editorial board of 10 other journals, including the *Journal of Abnormal Psychology*. Dr. Lynn has authored or edited 25 books, and he has published more than 360 articles and chapters on the topics of hypnosis, dissociation, trauma, fantasy, psychotherapy, and scientific thinking in psychology. He is the recipient of the Chancellor's Award from the State University of New York and numerous awards bestowed by the American Psychological Association and other professional societies. His research has been supported by the National Institute of Mental Health and has garnered substantial media attention.

Craig P. Polizzi is a clinical psychology doctoral student at Binghamton University. He has collaborated on randomized controlled trials investigating integrative and complementary interventions for veterans with posttraumatic stress disorder (PTSD). He has also participated in trials examining educational interventions promoting self-regulation in children. His current research focuses on clarifying the relations among acceptance, mindfulness, emotion regulation, and resilience and on elucidating how each functions as a self-regulation strategy. Recently, he has served as principal investigator on a longitudinal study examining novel ways to facilitate the salutary effects of mindfulness meditation.

Joseph P. Green is a professor of psychology at The Ohio State University and works at one of OSU's regional campuses in Lima, Ohio. He has published 70 journal articles and book chapters primarily in the area of hypnosis, imagination, and suggestion-based approaches to

psychotherapy. He has authored or edited two volumes on applying clinical hypnosis for health care and tobacco addiction. Dr. Green served two terms as president of the American Psychological Association, Division 30 (Society of Psychological Hypnosis). His work has been recognized with numerous awards, including *Early Career, Distinguished Contributions to Scientific Hypnosis* and *Distinguished Contributions to Professional Hypnosis* (APA D30), *Clark Hull Award for Scientific Excellence in Writing on Experimental Hypnosis* (American Journal of Clinical Hypnosis), *Arthur Shapiro Best Book Award* (Society for Clinical and Experimental Hypnosis), and the *Distinguished Teaching Award* (OSU Alumni Association).

Damla E. Aksen is a clinical psychology doctoral student at Binghamton University. She has worked in interdisciplinary psychology laboratories that studied perception, development, and evolutionary perspectives. She is interested in studying impulsivity, mindfulness, and emotion dysregulation.

Ashwin Gautam is a clinical psychology doctoral candidate at Binghamton University. He has coordinated studies investigating the effect of mindfulness inductions on experimental fear conditioning, inhibition, and extinction. He is currently serving as a principal investigator on a study exploring the association between mindfulness and procrastination. His current research interests involve the application of acceptance and mindfulness-based therapies for treatment of posttraumatic stress and anxiety related disorders.

James Evans is a clinical psychology doctoral candidate at Binghamton University, where he is a research associate in the Laboratory of Consciousness and Cognition. His primary research interests are in mindfulness and states of consciousness, including those reported in hypnosis, mystical, and other unusual experiences. His clinical interests are in disorders of personality and emotion regulation.

References

- Barnes, J., Dong, C. Y., McRobbie, H., Walker, N., Mehta, M., & Stead, L. F. (2010). Hypnotherapy for smoking cessation. *The Cochrane Database of Systematic Reviews*, (10), CD001008.
- Bernardy, K., Klose, P., Busch, A. J., Choy, E. H., & Häuser, W. (2012). Cognitive behavioural therapies for fibromyalgia syndrome. *The Cochrane Database of Systematic Reviews*, (9), CD009796.
- Birnie, K. A., Noel, M., Parker, J. A., Chambers, C. T., Uman, L. S., Kisely, S. R., & McGrath, P. J. (2014). Systematic review and meta-analysis of distraction and hypnosis for needle-related pain and distress in children and adolescents. *Journal of Pediatric Psychology*, 39(8), 783–808.
- Chen, P. Y., Liu, Y. M., & Chen, M. L. (2017). The effect of hypnosis on anxiety in patients with cancer: A meta-analysis. *Worldviews on Evidence-Based Nursing*, 14(3), 223–236.
- Elkins, G. (Ed.) (2016). *Handbook of medical and psychological hypnosis: Foundations, applications, and professional issues*. New York, NY: Springer.
- Elkins, G., Jensen, M. P., & Patterson, D. R. (2007). Hypnotherapy for the management of chronic pain. *International Journal of Clinical and Experimental Hypnosis*, 55(3), 275–287.
- Flammer, E., & Alladin, A. (2007). The efficacy of hypnotherapy in the treatment of psychosomatic disorders: Meta-analytic evidence. *International Journal of Experimental Hypnosis*, 55(3), 251–274.
- Flammer, E., & Bongartz, W. (2003). On the efficacy of hypnosis: A meta-analytic study. *Contemporary Hypnosis*, 20(4), 179–197.
- Gonsalkorale, W. M., Miller, V., Afzal, A., & Whorwell, P. J. (2003). Long term benefits of hypnotherapy for irritable bowel syndrome. *Gut*, 52(11), 1623–1629.
- Green, J. P., Lynn, S. J., & Montgomery, G. H. (2008). Gender-related differences in hypnosis-based treatments for smoking: A follow-up meta-analysis. *American Journal of Clinical Hypnosis*, 50(3), 259–271.
- Jensen, M. P., & Patterson, D. R. (2014). Hypnotic approaches for chronic pain management: Clinical implications of recent research findings. *American Psychologist*, 69(2), 167–177.

- Kirsch, I., Montgomery, G., & Sapirstein, G. (1995). Hypnosis as an adjunct to cognitive-behavioral psychotherapy: A meta-analysis. *Journal of Consulting and Clinical Psychology*, 63(2), 214–220.
- Milling, L., Gover, M. C., & Moriarity, C. L. (2018). The effectiveness of hypnosis as an intervention for obesity: A meta-analytic review. *Psychology of Consciousness: Theory, Research and Practice*, 5(1), 29–45.
- Montgomery, G. H., David, D., Winkel, G., Silverstein, J. H., & Bovbjerg, D. H. (2002). The effectiveness of adjunctive hypnosis with surgical patients: A meta-analysis. *Anesthesia & Analgesia*, 94(6), 1639–1645.
- Montgomery, G. H., DuHamel, K. N., & Redd, W. H. (2000). A meta-analysis of hypnotically induced analgesia: How effective is hypnosis? *International Journal of Clinical and Experimental Hypnosis*, 48(2), 138–153.
- Tefikow, S., Barth, J., Maichrowitz, S., Beelmann, A., Strauss, B., & Rosendahl, J. (2013). Efficacy of hypnosis in adults undergoing surgery or medical procedures: A meta-analysis of randomized controlled trials. *Clinical Psychology Review*, 33(5), 623–636.
- Webb, A. N., Kukuruzovic, R., Catto-Smith, A. G., & Sawyer, S. M. (2007). Hypnotherapy for treatment of irritable bowel syndrome. *The Cochrane Database of Systematic Reviews*, (4), CD002823.

Suggested Reading

- Jensen, M. P. (2011). *Hypnosis for chronic pain management: Therapist guide*. Oxford, UK: Oxford University Press.
- Lynn, S. J., Rhue, J. W., & Kirsch, I. (Eds.) (2010). *Handbook of clinical hypnosis* (2nd ed.). Washington, DC: American Psychological Association.
- Patterson, D. R. (2010). *Clinical hypnosis for pain control*. Washington, DC: American Psychological Association.

Motivational Interviewing

Thad R. Leffingwell

Department of Psychology, Oklahoma State University, Stillwater, OK, USA

MI's technical and theoretical roots trace to the client-centered counseling strategies popularized by Carl Rogers and classic social psychological theories of cognitive consistency (Festinger, 1962) and psychological reactance (Brehm, 1966). The first clinical application of the method that would come to be known as MI appeared in the research literature in 1988 in the form of the *Drinker's Check-up*, which utilized nonjudgmental client-centered listening skills while reviewing assessment feedback (Miller, Sovereign, & Krege, 1988). William Miller continued to develop the method, and, after developing a collaborative partnership with Stephen Rollnick, the first book fully describing the MI approach was published in 1991 (Miller & Rollnick, 1991).

For the first few decades of the development, investigation, and dissemination of MI, applications of the method were focused almost exclusively upon intervening with misuse of and addiction to psychoactive substances, including alcoholism and drug addiction. Indeed, the subtitle of the first book was *Preparing People to Change Addictive Behaviors* (Miller & Rollnick, 1991). MI targets ambivalence in the behavior change process, which often manifests as resistance to change and is common among people struggling with addictions. After the initial empirical success and accelerating dissemination of MI among substance use professionals, others began applying and investigating MI in other contexts with other target behaviors and populations including primary healthcare (Rollnick, Miller, & Butler, 2008), health promotion (Resnicow & Rollnick, 2011), and mental health (Arkowitz & Westra, 2004), with both adults and adolescents (Cushing, Jensen, Miller, & Leffingwell, 2014; Jensen et al., 2011). The growing recognition of the wider applicability of the MI approach was reflected in the second book, subtitled *Preparing People for Change* (Miller & Rollnick, 2002). Today MI is recognized as a broadly useful tool for helping me make a broad range of behavior changes, especially when ambivalence or resistance to change is present.

The Problem of Resistance to Change

It is a well-known problem that habits are difficult to change even when the status quo is maladaptive or unhealthy and that people often resist professional attempts to assist with change. According to the transtheoretical model, people may be in the pre-action stages of precontemplation (not even thinking about change) or contemplation (acknowledging need or desire to change, but not now or soon) for months or years (Prochaska, DiClemente, & Norcross, 1992). The contemplation stage is characterized by ambivalence about change—a part of the person sees the need or wants the change, but another part prefers and defends the status quo. The preference for the status quo can be driven by many factors including, for example, lacking confidence about the likely success of change attempts, valuing other priorities more highly right now, or favoring immediate benefits more than long-term payoffs that are subject to delay or probability discounting (Bickel, Johnson, Koffarnus, MacKillop, & Murphy, 2014). From the MI perspective, these individuals are viewed as *stuck* in ambivalence, and the goal of the MI conversation is to help patients get *unstuck* and move toward attempting and sustaining necessary behavior changes.

Patient Language About Change

In conversations about a behavior change, patient language basically takes one of two forms—*change talk* or *sustain talk* (referred to as “*resistance*” in earlier versions of MI). Change talk is defined as any expressions that favor or encourage change, including concerns about the status quo, fears about the future if the behavior does not change, ideas about how change might be accomplished, or intentions to change. Sustain talk, on the other hand, is defined as any expressions from the patient that argue for or defend the status quo, including descriptions of advantages or benefits of the status quo, denigration or barriers to change, or hopelessness about change.

The MI approach takes several important positions about patient language in conversations about behavior change. First, the MI model posits that the patient’s language is much more influential on subsequent behavior than the provider’s language. As a result, MI de-emphasizes motivational strategies that are more provider centered such as education, warning of dangers, direct persuasion, or professional advice. Second, the MI model suggests patients are better persuaded by their own language and that the more they hear themselves expressing *change talk* in the conversation (and, as a result, less *sustain talk*), the more subsequent attempts at change are to occur. Miller and Rose (2009) proposed a *theory of MI* that proposes that when *change talk* grows and *sustain talk* falls away during an MI conversation, the patient is more likely to experience and/or express a sense of commitment to change that is supported by a sense of intrinsic motivation, and it is this inner psychological experience that sets the stage for subsequent behavior change. As a result, the primary goal during an MI conversation is to increase the frequency and/or intensity of *change talk* and decrease the frequency and/or intensity of *sustain talk* as a way of building intrinsic motivation and evoking commitment to change.

Style and Spirit of MI

MI has been described as have both a “*style and spirit*” as well as a set of proscribed conversational strategies and recommended techniques. The style and spirit can be thought of as the attitude, worldview, or stance of the MI provider. Rather than being about what the

MI provider is to “do,” the MI style and spirit is about how the MI provider should “be” during the conversation. The style and spirit have been described as the music or melody of MI, giving it pace and feeling, when used with the “lyrics” of the skills and strategies.

In the original formulations of MI (Miller & Rollnick, 1991, 2002), the spirit of MI was described as consisting of three main characteristics. First, MI should have a spirit of *collaboration*. The interaction between provider and patient should have a sharing of power, as opposed to a “one up, one down” character. The provider is to avoid taking on a role as expert or authority and instead try to develop a sense of equal partnership in exploring the topic of behavior change. Second, MI should be *evocative*. In MI, the most valued words in the conversation are the patient’s own. If change is to happen, it is mostly likely to be as a result of the patient being persuaded by his or her own words, rather than succumbing to the arguments or persuasion of the provider. For this reason, it is important for the MI provider to act in ways that encourage and evoke the patient’s side of the conversation. Finally, MI should be *autonomy supportive*. Ideally, in an MI conversation, the patient would experience an expansion of their sense of personal choice and autonomy, rather than experiencing attempts to control or limit choices (e.g., “you must...”). This aspect of MI reduces the influence of psychological reactance and improves the chances that the patient may overcome ambivalence and reactance and choose to make necessary changes.

The latest description of the MI technique (Miller & Rollnick, 2013) expanded a bit upon these basic aspects of the spirit of MI. The aspects of collaborative partnership and evocation remain, but autonomy support has been subsumed under a broader concept of *acceptance*. Autonomy support is viewed as one facet of acceptance, but the concept also includes a fundamental belief in the *absolute worth* of the individual, accurate *empathy*, and *affirmation*, or actively seeking out and elevate an individual’s strengths. Finally, the third edition also adds *compassion* as an important aspect of the essential spirit of MI. *Compassion* refers to the intentional application of MI in the service of what is best for the patient, not the provider.

Skills and Strategies of MI

MI also includes a number of proscribed or recommended strategies for the competent practice of MI. These strategies represent the “microskills” by which the spirit of MI is translated into practice and are in service of enacting the spirit. The skills can be reliably coded, and more consistent use of the strategies in the context of MI conversations has been found to lead to more favorable outcomes (Magill et al., 2014; Moyers, Martin, Manuel, Hendrickson, & Miller, 2005).

The most fundamental skills of the MI conversation involve the strategic use of active listening skills. In MI, the provider is expected to utilize evocative strategies such as open-ended questions, accurate reflections, and frequent summaries. MI de-emphasizes the value of provider-centered language of advice, education, or persuasion. The goal of the MI provider is to strategically and intentionally apply the skills of active listening to evoke and grow *change talk* and quiet *sustain talk* from the patient.

One highly effective MI strategy is the use scaled questions, or what are sometimes referred to as *readiness rulers* (Rollnick et al., 2008). MI providers may wish to assess readiness to change, confidence in change, or the importance of change using a 0–10 scale. For example, “How ready would you estimate you are to quit smoking today, from zero to ten, with zero being ‘no interest at all’ and ten being ‘totally committed?’” The answers provided to such questions provide a very useful and quick assessment of the constructs of interest, but they are especially valuable if follow-up questions are asked. For example, if patient were to respond to the example question

with “four,” a useful follow-up question might be something like “Why did you choose four, and not zero?” The answers to that question would almost certainly take the form of change talk. This strategy reliably evokes valuable change talk into the conversation. Scaled questions are often recommended in brief and specialized adaptations of MI like the “5 A’s” and Screening, Brief Intervention, and Referral to Treatment (SBIRT) models, discussed below.

Two other broad MI strategies are essential to the competent practice of MI—*rolling with resistance* and *developing discrepancies*. Argumentation and confrontation in response to patient resistance are seen as counterproductive, largely because they tend to *increase* frequency and intensity of *sustain talk*. Rolling with resistance is an alternative strategy for responding that is intended to quiet *sustain talk* or, ideally, evoke the “other side of the coin” of *change talk*. MI includes many specific strategies rolling with resistance including the use of reflective listening strategies but also reframing or emphasizing choice and autonomy, among others.

Developing discrepancy is based upon theories of cognitive consistency and dissonance that posit that people strive for consistency between attitudes and actions (Festinger, 1962). When individuals are confronted with inconsistency, they experience a sense of dissonance that is uncomfortable, and they are thus motivated to act in such a way to reduce the discrepancy. When one’s behavior is experienced as inconsistent with an attitude or belief that is highly valued, that individual may be expected to change their behavior to better fit, or “square,” with the attitude or belief. For this reason, the MI practitioner is constantly listening for ways that the status quo may be inconsistent with a value, goal, or aspect of self-image of the patient. If such discrepancies are found and made salient, they may help move the patient in the direction of change. For example, an MI provider working with a smoker may at some point in the conversation say something to the patient like “you strike me as the kind of person who really values your independence, but at the same time you find yourself quite dependent on those cigarettes. In a sense, you don’t choose them – they choose you.”

Four Processes

In the latest iteration of MI (Miller & Rollnick, 2013), a new concept was introduced of the *four processes* of MI. The four processes essentially define the beginning, middle, and end of an MI encounter, though they do not necessarily always proceed in a linear fashion. The four processes are *engaging*, *focusing*, *evoking*, and *planning*. Engaging is the first step into an MI conversation and involves establishing a rapport and relationship as a foundation for proceeding. Focusing is a process of narrowing the agenda for the conversation onto one, or a few, important target behaviors for the conversation. Evoking is the process that most providers may recognize as the “meat” of the MI conversation. It involves an intentional conversation about the target behavior, with a focus of evoking and growing change talk and minimizing resistance. The final process of planning involves the transition from *whether* change should or will occur (the primary focus of MI) into *how* a change may occur. This process might involve plans to simply revisit the issue if now is not the right time to change, or might involve a detailed plan for change including the use of professional assistance.

Evidence Base for Motivational Interviewing

MI has emerged and developed in the age of evidence-based practice. As a result, MI has been rigorously studied from its very outset, and today there have been hundreds of randomized

clinical trials and several meta-analyses evaluating the efficacy of MI for a broad range of outcomes and populations. In general, the data overwhelmingly support the conclusion that MI does have a reliable effect on a broad range of target behaviors including alcohol misuse (Burke, Arkowitz, & Menchola, 2003), tobacco dependence (Heckman, Egleston, & Hoffmann, 2010), obesity (Armstrong et al., 2011), and gambling (Yakovenko, Quigley, Hemmelgarn, Hodgins, & Ronksley, 2015) and as an adjunct for treatment of psychological disorders (Arkowitz, Miller, & Rollnick, 2015), among both adults and adolescents (Cushing et al., 2014; Jensen et al., 2011). The effect sizes for MI interventions are typically small to moderate, but that may not be surprising given that the durations of the interventions studied are often quite brief, typically ranging from only a few minutes to two brief sessions. As a result, the cost-effectiveness of MI interventions is quite strong.

Training in MI

MI is a complicated clinical intervention approach for which training is necessary. The available evidence suggests that a broad array of health professionals, including both medical providers and social service providers, can be trained to effectively implement MI competently (Madson, Loignon, & Lane, 2009). However, in order for improvements toward the competent practice of MI to be maintained, a single training or workshop is unlikely to be sufficient. Sustained competent practice of MI typically requires intensive training with performance feedback followed by several months of ongoing coaching and supervision (Schwalbe, Oh, & Zweben, 2014).

Applications and Adaptations of Motivational Interviewing

MI proper is a principle-driven approach that relies upon the expertise of the provider to identify appropriate targets for MI and to weave MI interventions together with other strategies that suit the needs of the particular client or problem at the moment. However, due to the demands of clinical trials to have replicable interventions and the demands of real-world settings to have more easily disseminable models for specific target behaviors (e.g., tobacco use) or settings (e.g., hospital emergency rooms), there have been a number of efforts to develop specific MI-based treatment and intervention models.

One example of an MI-based treatment is motivational enhancement therapy (MET), a treatment designed and tested initially as part of Project MATCH, a large multisite clinical trial of interventions for alcohol abuse and dependence (Miller, Zweben, DiClemente, & Rychtarik, 1992). MET was a brief intervention consisting of four treatment sessions and utilized the basic principles of MI at the time. Like the original Drinker's Check-up (Miller et al., 1988) and many early trials of MI, the MI conversation occurred in the context of reviewing the results of a comprehensive clinical assessment, especially in the first two sessions. In the last two sessions, spaced weeks apart, the therapist would use MI principles to encourage motivation and progress.

Other adaptations of MI have attempted to create simplified, targeted intervention models that could be more easily disseminated to a variety of providers. For example, the Surgeon General's influential document *treating tobacco dependence* promoted a "5 A's" model of brief intervention designed to be implemented by a variety of healthcare providers at every encounter with patients who use tobacco (Tobacco Use and Dependence Guideline Panel, 2008).

The five A's are as follows: (a) assess every patient for tobacco use and for those who are current users proceed to (b) advise quitting with brief, personalized advice; (c) assess for readiness to make a quit attempt and motivate those who are not currently ready; (d) assist with a quit attempt by offering guidance, prescriptions, or referrals; and (e) arrange for follow-up at subsequent visits. The recommendations for assessing readiness and motivated unmotivated patients essentially describe the practice of MI. A similar brief intervention model that incorporates MI is SBIRT (Babor et al., 2007). SBIRT is a model for brief intervention designed initially for use in busy hospital emergency departments to address patients for who alcohol or drug misuse may have contributed to their illness or injury. The brief intervention component of SBIRT is essentially MI.

Conclusions

MI is a broadly applicable and essential evidence-based approach to working with patients to overcome resistance and move toward healthy behavior changes. The approach can be learned and applied by many different types of providers and is applicable to many clinical problems and populations.

Author Biography

Thad R. Leffingwell is a clinical health psychologist and is currently a professor and head of the Department of Psychology in the College of Arts and Sciences at Oklahoma State University. His research has explored how people change health risk behaviors and how providers can interact with patients to encourage behavior change more effectively. He is a member of the Motivational Interviewing Network of Trainers and has providing training in MI to thousands of providers over the last 20 years.

References

- Arkowitz, H., Miller, W. R., & Rollnick, S. (2015). *Motivational interviewing in the treatment of psychological problems*. New York, NY: Guilford.
- Arkowitz, H., & Westra, H. A. (2004). Integrating motivational interviewing and cognitive behavioral therapy in the treatment of depression and anxiety. *Journal of Cognitive Psychotherapy, 18*, 337–360. doi:10.1891/jcop.18.4.337.63998
- Armstrong, M. J., Mottershead, T. A., Ronksley, P. E., Sigal, R. J., Campbell, T. S., & Hemmelgarn, B. R. (2011). Motivational interviewing to improve weight loss in overweight and/or obese patients: A systematic review and meta-analysis of randomized controlled trials. *Obesity Reviews, 12*, 709–723. doi:10.1111/j.1467-789X.2011.00892.x
- Babor, T. F., McRee, B. G., Kassebaum, P. A., Grimaldi, P. L., Ahmed, K., & Bray, J. (2007). Screening, Brief Intervention, and Referral to Treatment (SBIRT): Toward a public health approach to the management of substance abuse. *Substance Abuse, 28*, 7–30.
- Bickel, W. K., Johnson, M. W., Koffarnus, M. N., MacKillop, J., & Murphy, J. G. (2014). The behavioral economics of substance use disorders: Reinforcement pathologies and their repair. *Annual Review of Clinical Psychology, 10*, 641–677. doi:10.1146/annurev-clinpsy-032813-153724
- Brehm, J. W. (1966). *A theory of psychological reactance*. Oxford, England: Academic Press.

- Burke, B. L., Arkowitz, H., & Menchola, M. (2003). The efficacy of motivational interviewing: A meta-analysis of controlled clinical trials. *Journal of Consulting and Clinical Psychology, 71*, 843–861. doi:10.1037/0022-006X.71.5.843
- Cushing, C. C., Jensen, C. D., Miller, M. B., & Leffingwell, T. R. (2014). Meta-analysis of motivational interviewing for adolescent health behavior: Efficacy beyond substance use. *Journal of Consulting and Clinical Psychology, 82*, 1212–1218. doi:10.1037/a0036912
- Festinger, L. (1962). Cognitive dissonance. *Scientific American, 207*, 93–107. doi:10.1038/scientificamerican1062-93
- Heckman, C. J., Egleston, B. L., & Hoffmann, M. T. (2010). Efficacy of motivational interviewing for smoking cessation: A systematic review and meta-analysis. *Tobacco Control, 19*, 410–416. doi:10.1136/tc.2009.033175
- Jensen, C. D., Cushing, C. C., Aylward, B. S., Craig, J. T., Sorell, D. M., & Steele, R. G. (2011). Effectiveness of motivational interviewing interventions for adolescent substance use behavior change: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 79*, 433–440. doi:10.1037/a0023992
- Madson, M. B., Loignon, A. C., & Lane, C. (2009). Training in motivational interviewing: A systematic review. *Journal of Substance Abuse Treatment, 36*, 101–109. doi:10.1016/j.jsat.2008.05.005
- Magill, M., Gaume, J., Apodaca, T. R., Walthers, J., Mastroleo, N. R., Borsari, B., & Longabaugh, R. (2014). The technical hypothesis of motivational interviewing: A meta-analysis of MI's key causal model. *Journal of Consulting and Clinical Psychology, 82*, 973–983. doi:10.1037/a0036833
- Miller, W. R., & Rollnick, S. (1991). *Motivational interviewing: Preparing people to change addictive behavior*. New York, NY: Guilford.
- Miller, W. R., & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change*. New York, NY: Guilford.
- Miller, W. R., & Rollnick, S. (2013). *Motivational interviewing: Helping people change* (3rd ed.). New York, NY: Guilford.
- Miller, W. R., & Rose, G. S. (2009). Toward a theory of motivational interviewing. *American Psychologist, 64*(6), 527–537. doi:10.1037/a0016830
- Miller, W. R., Sovereign, R. G., & Krege, B. (1988). Motivational interviewing with problem drinkers: II. The Drinker's Check-up as a preventive intervention. *Behavioural Psychotherapy, 16*, 251–268.
- Miller, W. R., Zweben, A., DiClemente, C. C., & Rychtarik, R. G. (1992). *Motivational enhancement therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence*. (Volume 2, Project MATCH Monograph Series). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.
- Moyers, T. B., Martin, T., Manuel, J. K., Hendrickson, S. L., & Miller, W. R. (2005). Assessing competence in the use of motivational interviewing. *Journal of Substance Abuse Treatment, 28*, 19–26. doi:10.1016/j.jsat.2004.11.001
- Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change: Applications to addictive behaviors. *American Psychologist, 47*, 1102–1114. doi:10.1037/0003-066X.47.9.1102
- Resnicow, K., & Rollnick, S. (2011). Motivational interviewing in health promotion and behavioral medicine. In W. M. Cox & E. Klinger (Eds.), *Handbook of motivational counseling: Goal-based approaches to assessment and intervention with addiction and other problems*. Chichester, UK: Wiley. doi:10.1002/9780470979952.ch25
- Rollnick, S., Miller, W. R., & Butler, C. C. (2008). *Motivational interviewing in health care*. New York, NY: Guilford.
- Schwalbe, C. S., Oh, H. Y., & Zweben, A. (2014). Sustaining motivational interviewing: A meta-analysis of training studies. *Addiction, 109*, 1287–1294. doi:10.1111/add.12558
- Tobacco Use and Dependence Guideline Panel. (2008). *Treating tobacco use and dependence: 2008 update*. Rockville, MD: United States Department of Health and Human Services. Retrieved from: <https://www.ncbi.nlm.nih.gov/books/NBK63952/>

Yakovenko, I., Quigley, L., Hemmelgarn, B. R., Hodgins, D. C., & Ronksley, P. (2015). The efficacy of motivational interviewing for disordered gambling: Systematic review and meta-analysis. *Addictive Behaviors*, 43, 72–82. doi:10.1016/j.addbeh.2014.12.011

Suggested Reading

Miller, W. R., & Rollnick, S. (2013). *Motivational interviewing: Helping people change*. New York, NY: Guilford.

Rollnick, S., Miller, W. R., & Butler, C. C. (2008). *Motivational interviewing in health care*. New York, NY: Guilford.

Rosengren, D. B. (2018). *Building motivational interviewing skills*. New York, NY: Guilford.

Rehabilitation Psychology

Monica F. Kurylo¹ and Kathleen S. Brown²

¹*Departments of Medicine and Public Health, University of Kansas Medical Center,
Kansas City, KS, USA*

²*Private Practice, Fort Myers, FL, USA*

Our wounds are often the openings into the best and beautiful part of us.

– David Richo

Rehabilitation psychologists “enhance the lives of people with disabilities and chronic illness” (American Psychological Association Division 22 Rehabilitation Psychology and the Rehabilitation Psychology Synarchy, 2015, p. 6) as noted in the petition for recognition of rehabilitation psychology as a specialty approved by the American Psychological Association’s (APA) Council of Representatives in August 2015 (e.g., Dunn & Elliott, 2008). Rehabilitation psychologists recognize the wounds and resilience of the people with whom they work. Since World War II, rehabilitation psychologists have partnered with physicians treating patients with a variety of injuries and/or chronic illness, including amputations, spinal cord injuries, and head injuries (Cox, Hess, Hibbard, Layman, & Stewart, 2010). As the field of rehabilitation psychology matured, the specialty expanded to include working with children with disabilities and their families related to the establishment of the National Society of Crippled Children and Adults, working with government vocational rehabilitation organizations funded by state-federal partnerships, and, more recently, advocating for people with disabilities through the passage of the Americans with Disabilities Act (ADA) in 1990.

Rehabilitation psychologists may specialize in working with particular medical diagnoses, such as head injury, stroke, spinal cord injury, amputation, or other neurological illness/injury, or they may work with a more broadly varied group of patients whom they encounter in inpatient or outpatient settings, similar to health psychologists. For example, rehabilitation psychologists may work with people who have become debilitated due to treatments for metastatic cancer, patients who are being considered for or have undergone solid organ transplant, and people who have sustained burn or electrical injuries. While the focus of psychological evaluation and intervention may be similar among both rehabilitation and health psychologists,

the petition for recognition of rehabilitation psychology as a specialty noted that “Rehabilitation Psychology works with individuals who have acquired a disability or chronic health condition. Clinical Health Psychology uses similar assessment and intervention techniques to identify and enact health behavior change, while Rehabilitation Psychology has a ‘specific focus on adaptation to illness or injury’ (Klonoff et al., 2011), and the interactions between the individual, family, and physical, social, and policy environments in order to enhance social role participation” (APA Division 22 Rehabilitation Psychology and the Rehabilitation Psychology Synarchy, 2015, p. 32).

In 1952, rehabilitation psychology was introduced as a new subspecialty within the APA. Rehabilitation psychologists were identified as practitioners, educators, researchers, and advocates for people with disabilities working with interdisciplinary teams. Since that time, the field of rehabilitation psychology has expanded to include expertise as policy makers, healthcare administrators, and program developers. The importance of the role of rehabilitation psychology in the team has also been recognized by regulatory agencies, such as CARE (formerly known as the Commission for Accreditation of Rehabilitation Facilities) that includes psychology in their guidelines for staff inclusion on rehabilitation teams.

The purpose of this chapter is to introduce rehabilitation psychology as a subspecialty particularly with regard to practice areas of assessment, intervention, and interdisciplinary team functioning. Although some assessment instruments and interventions are similar to clinical health psychology, readers will understand how rehabilitation psychology contrasts with health psychology in its roles and focus. The role of rehabilitation psychology in research, advocacy, and training is also explained. For further information on rehabilitation psychologists performing as program developers, policy makers, and healthcare administrators, readers are encouraged to review Cheak-Zamora, Reid-Arndt, Hagglund, and Frank (2012).

Practice of Rehabilitation Psychology: Assessment

In rehabilitation psychology, the assessment process is used to develop a multidisciplinary or interdisciplinary treatment plan that addresses the impact of a disability or other chronic health condition. In addition, adjustment and accommodation to the disability along with the necessary social and community supports to optimize functioning are also evaluated. Reasons for referral are many: individuals may be referred for assessment of physical, cognitive, emotional, and/or social adaptation to injury, illness, and disability in the patient and family; cognitive, emotional, and behavioral dysfunction; neuropsychological evaluation to determine ability to function at home, school, and/or work with or without accommodations; evaluation of self-care and independent living skills; evaluation of psychosexual functioning, with an emphasis on education regarding disability-related changes and use of adaptive technology; evaluation of social and recreational participation; assessment of health self-management and prevention of secondary complications; and assessment of caregiver status and functioning, including caregiver knowledge and skills, social support, and self-care. Assessment of each of these domains requires knowledge of a broad range of quantitative and qualitative assessment instruments and techniques.

Along with instruments for mood, pain, coping, adjustment to disability, and cognitive and physical functioning in health populations, assessment instruments have also been created to be used with diverse rehabilitation populations to address capacity for self-care, work, and independent living. Instruments specific to rehabilitation populations, for example, may include instruments that assess the psychological assimilation of traumatic events, monitor

progress throughout inpatient rehabilitation, and assess how people with disabilities function as active members of their communities (Heinemann & Mallison, 2010). Given the high prevalence of brain injury in traumatic physical injuries, cognitive screening measures are vital to determine the presence and extent of potential cognitive processing issues that can negatively impact treatment and recovery (Novack, Sherer, & Penna, 2010). Assessments of specific environmental or behavioral features relevant to intervention, such as stimulus control or contingency management, often focus on factors to aid integrative assessment for the development of a rationale for a particular intervention, to develop a working relationship with the patient and/or significant others, to address emotional reactions, and to develop expectations and attributions that support the intervention.

The importance of assessing and providing psychological services that best meet the individual and diverse needs of people with disabilities while maximizing their health and welfare, independence and choice, functional abilities, and social participation was highlighted in the *Guidelines for Assessment of and Intervention with Persons with Disabilities* (APA, 2012). Multiple issues relevant to diversity must be considered (e.g., age, gender, sexual orientation, socioeconomic status, religion, race, ethnicity) along with disability identity. An individual's military or veteran status may be an important part of their identity and may differentially affect their adaptation to disability or approach to assessment. Individuals with disabilities need to be included in all aspects of decision making to the best extent possible to ensure that their preferences and values are integrated into the conceptualization of assessment results.

As part of the comprehensive evaluative process, a rehabilitation psychologist may consult with other professionals, such as attorneys, government agencies, educational institutions, the Department of Vocational Rehabilitation, insurance companies, and case managers to optimize an individual's community functioning. Such consultations may include evaluation of acquired cognitive deficits with educational or vocational implications, development of reasonable accommodations for return to school or work, quantification of accident-related "loss" for forensic purposes, recommendations related to hospital discharge plans, or recommendations concerning resumption of premorbid activities such as driving.

Practice of Rehabilitation Psychology: Intervention

Psychotherapeutic interventions are an important part of the rehabilitation plan for individuals with disabilities to improve emotional distress often produced by changes in physical, task, and social functioning; to address the disruption of prior personal, family, and community roles; and to aid motivation, adherence, or participation in rehabilitation efforts. Interventions in rehabilitation psychology focus on applying the findings of assessment and diagnosis to facilitate functional task performance and social role participation in order to maximize the productive engagement of the individual in *all* environments. As disability is seen as a problem of exclusion from ordinary life through a "person-first" perspective (Wright, 1983), interventions are aimed at facilitating adaptation and accommodation to illness or injury while seeking to minimize the attendant limitations. Evidence-based interventions are modified to address the specific challenges an individual, couple, or family face that limit activities and restrict social role participation. Therapeutic interventions can involve emotional, cognitive, and/or behavioral domains. They can help shift the focus of the individual with a disability from premorbid sources of self-esteem, such as physical prowess, to less impaired areas, such as cognitive and personality strengths (Keany & Glueckauf, 1993). At the family and community

level, such emotional, cognitive, and behavioral shifts in emphasis can also significantly reduce disability by helping develop accommodations in the physical and social environments.

Hart and Ehde (2015) developed a theory-driven framework for specifying rehabilitation interventions according to its targets, its active ingredients, and the mechanisms of action that connect them. They suggest that rather than describing rehabilitation treatments by the discipline(s) providing them (e.g., physical therapy, vocational retraining program), or by the problems they are intended to solve (e.g., attention training), treatments must specify the desired changes to narrow choices about specifically what one can do to effect change. Hart and Ehde call for a common system of nomenclature for characterizing and quantifying the treatment targets and ingredients used in clinical interventions and research to advance theory development and improve the quality of intervention research.

In rehabilitation, the team includes not only direct service providers but also the patient, family and significant others, and available community, school, and work relationships. Family members who provide care for persons with disability are themselves at risk for associated physical, mental, emotional, social, and financial problems and therefore may benefit from caregiver-oriented interventions (Chwalisz & Dollinger, 2010). Caring for the caregiver improves and sustains the care provided to the person experiencing chronic illness or disability resulting in improvements in outcomes. Rehabilitation psychologists not only help caregivers understand their loved ones' difficulties but also help to manage problem behaviors, obtain and utilize social supports, and provide interventions to maintain caregiver health and well-being. In caring for others, caregivers must take care of themselves in order to avoid burnout and the disintegration of a previously workable care plan.

Rehabilitation psychologists also intervene to assist with reducing the morbidity and cost of work injuries. Delivering health and rehabilitation services to injured workers in the work environment, instead of in community care settings, has been demonstrated to significantly reduce days away from work and worker's compensation costs (Wegener & Stiers, 2010). Negative experiences, including stereotyping and marginalization, discrimination, and disempowerment in the workplace, may also need to be addressed for individuals with disabilities, as with any individuals of minority status.

Practice of Rehabilitation Psychology: Interdisciplinary Team Functioning

Rehabilitation includes many disciplines working collectively to serve patients with a wide variety of disabilities and chronic conditions. Rehabilitation psychologists contribute across the full spectrum of care from prevention, diagnosis, consultation, treatment, rehabilitation, and/or end-of-life and palliative care. As rehabilitation psychologists practice in these diverse healthcare delivery systems, they have a long tradition of working within multidisciplinary and interdisciplinary teams (Butt & Caplan, 2010). Interdisciplinary typically implies a team that is not only composed of different disciplines working toward a shared set of treatment goals but also have some fluidity of professional boundaries as team members collaborate to best assist the patient to achieve their goals. Functioning within teams requires expertise in communication, behavioral issues and management, patient decision making, and human interaction in systems with attention to the environment, culture, and context in which care is delivered. Rehabilitation psychologists must understand the roles, skills, and contributions of each discipline involved on the team as they educate other disciplines about the potential contributions of psychologists to the team. Awareness of areas of overlap in team member's

contributions is aided by clear communication and respect for each team member's identified role that can change across patients or an individual's rehabilitation program. Barriers to effective team functioning can include hierarchical attitudes, unhealthy stress reactions, the lack of understanding of the advantages of coordinated team care, fear of change, risk aversion, and the challenge of developing an entrepreneurial spirit. Rehabilitation psychologists operate within rehabilitation teams, systems, and programs and provide expertise in the measurement and understanding of rehabilitation team functioning, rehabilitation outcomes, and facilitation of interdisciplinary rehabilitation team functioning (Butt & Caplan, 2010). Participation in interdisciplinary team meetings, case consultations, and rounds allow for the psychologist to aid the integration of problem definition, goals, observations, and services of different providers. As disability results from person-task-environment interactions, a focus on effective and efficient team functioning is necessary to optimize outcomes.

Research in Rehabilitation Psychology

Health and rehabilitation psychology research has grown significantly in the past several decades. Although pioneers in the field of rehabilitation psychology provided an early framework for conceptualizing the work of rehabilitation psychology (i.e., Meyerson, 1948; Wright, 1983), rehabilitation psychology has generally been more focused on empirical evidence and clinical application than theory development (Dunn & Elliott, 2008). This is due in part to the challenge of applying psychological theories in multidisciplinary settings as well as the focus of rehabilitation on the individual and family, whose circumstances are unique and limit generalization as demanded in science. As scientist-practitioners, practitioners often rely on single-subject designs to apply the rigorous methods of science to their daily practice in an effort to determine whether interventions work and to improve patient outcomes. For general and geriatric rehabilitation populations, common areas of rehabilitation research typically focus on the natural history of disability, functional assessment and performance evaluations, intervention issues and outcomes, and rehabilitation service delivery systems.

Rehabilitation has been called a black box as its precise ingredients, their mechanisms of action, and their efficacy and effectiveness remain largely unknown. This lack of a systematic way to characterize interventions in rehabilitation has prevented advances in the field of rehabilitation in several ways (Dijkers, Hart, Tsousides, Whyte, & Zanca, 2014). Rehabilitation research has been disadvantaged by interventions that cannot be readily replicated, tested against one another, or analyzed to try to match specific components to different kinds of patients. Lack of clear evidence about effective treatments and the lack of guidance by which to choose specific therapies for individual patients have hampered progress in clinical applications in rehabilitation. Communication and collaboration across the rehabilitation team, as well as communication with patients and third-party payers, has been impeded by the lack of a common system for naming and describing treatments. Rehabilitation research needs to apply a systematic approach to treatment definition. When we can clearly define the active ingredients and targets of a treatment, then we can reliably study its effects; assess the fidelity of its implementation; replicate it in the correct doses in further studies, both experimental and observational; specify the changes and refinements that might be made to improve it; and disseminate it to the clinic setting (Hart & Ehde, 2015).

Healthcare in the United States suffers from high and rising costs and poor and uneven quality and safety, factors that increase the vulnerability of individuals with disabilities. The increasing prevalence of disability and chronic health conditions across the general population

is largely driven by behavioral and lifestyle factors that are the primary contributors to premature morbidity and mortality and the foundation of the work of rehabilitation psychologists. Theories of health behavior change applied to people with chronic illness and disability help to explain the cognitive mechanisms of behavior change and adherence to treatment in the rehabilitation setting.

Research in positive psychology and posttraumatic growth, which focuses on resilience and promotes the concept that individuals can grow positively in response to challenge, stress, and trauma, has been applied to a variety of medical populations. The integration of positive psychology and rehabilitation psychology research has only more recently been applied to rehabilitation populations. Peter, Geyh, Ehde, Müller, and Jensen (2015) identified three commonalities between the two areas of study: positive principles, focus on individual strengths and resources, and well-being, social participation, and growth as key outcomes.

Within a population health context, MacLachlan and Mannan (2014) identified areas of potential research that rehabilitation psychology could contribute to in an effort to address many of the challenges identified in the *World Report on Disability*. Specific targeted contributions include addressing the human resources needed for health crises in rehabilitation, developing prosocial and community-based interventions and programs, helping to identify and overcome difficulties to accessing healthcare, refining the measurement and classification of disability, and strengthening research, policy, and advocacy for and with people with disabilities.

Research in interdisciplinary team science has also grown significantly within the past decade. Interdisciplinary research in rehabilitation is performed by individuals or teams that integrate information, data, technique, tools, perspectives, concepts, and/or theories from two or more disciplines or bodies of specialized knowledge to advance fundamental understanding or to solve problems whose solutions are beyond the scope of a single discipline or field of research (Committee on Science, Engineering and Public Policy [COSEPUP], 2004). Elements necessary to facilitate interdisciplinary team science include mutual respect among scientists; regular interactions focused on science; common language, constructs, and cultural norms; and institutional leadership and funding that supports transdisciplinary research. For rehabilitation psychologists practicing in the field, interdisciplinary science must address pragmatic concerns that impact how the psychologists can be an effective partner on the team. Concerns may include how the team's mission is determined; how the team and its mission is tied to the larger organization and the climate for the team's functioning; how leadership is determined, if it is shared, and whether and how it may change over time; what are the team's communication patterns; and how does the team make decisions and review and evaluate its progress and decisions. Each of these aspects of effective interdisciplinary team functioning is crucial to understand within our changing healthcare environment and shift toward integrated care in order to optimize patient outcomes within person-task-environment models of care.

Rehabilitation Psychologists as Advocates

The essence of advocacy in rehabilitation psychology is captured in Guideline 21 of the *Guidelines for Assessment of and Intervention With Persons With Disabilities* (APA, 2012), which states that "When working with systems that support, treat, or educate people with disabilities, psychologists strive to keep the clients' perspectives paramount and advocate for client self-determination, integration, choice, and least restrictive alternatives" (p. 26). Rehabilitation psychologists advocate on treatment teams, within their family structure and

social network, and in the community for their patients with regard to their physical and emotional needs. They advocate for needed environmental changes, such as accessibility to/from health provider offices (e.g., ramps, curb cuts), for additional time to complete tasks or other accommodations (e.g., at work or at school), or for therapeutic assistance (e.g., obtaining a therapy dog to assist with physical or sensory needs).

Rehabilitation psychologists also advocate at local, state, and national levels for legislation to assist people with disabilities, including ensuring access to affordable healthcare, social service, and rehabilitative therapy resources, and inclusion in social roles through the ADA (1990) (Cheak-Zamora et al., 2012). They do this because they are “mindful of the fact that limitations of functioning may not only be inherent in the disability itself, but are often attributable, in whole or part, to environmental and institutional barriers, and to negative social attitudes” (APA Division 22 Rehabilitation Psychology and the Rehabilitation Psychology Synarchy, 2015, p. 6).

Training in Rehabilitation Psychology

Graduate students in clinical and counseling psychology programs may first be introduced to physical rehabilitation in practicum experiences in inpatient and/or outpatient medical rehabilitation settings tied to clinical health psychology programs. For example, at the University of Kansas and the University of Kansas Medical Center, where the first author is located, graduate students in clinical psychology (health track) participate in required health practicum medical center experiences, including a neurorehabilitation psychology rotation (the first author is the supervisor) that allows them to participate in interviews, evaluations, and treatment of patients with new onset or chronic physical and medical issues. Externs also have the opportunity to participate in interdisciplinary team meetings, patient/family education, support/education groups, and community outings. The foundation of clinical psychology, learned within the first 2 years of their graduate program, provides a basis from which the students apply their skills in the new setting of inpatient or outpatient physical rehabilitation. Training may also occur during predoctoral internship or in postdoctoral experiences.

According to postdoctoral training guidelines first published in 1995 by Patterson and Hanson, training at the postdoctoral level is expected to occur for a minimum of 1 year with a minimum of two supervisors and include supervised practice, seminars, and coursework, a minimum of 2 hr of weekly didactics, a minimum of 2 hr of weekly supervision, and the patient populations and didactics are to be related to disabilities and chronic health conditions (Patterson & Hanson, 1995). In addition, trainees should be funded, written objectives for the training program are to be provided, formal trainee evaluations are to occur at least twice a year, and program evaluations are to occur annually.

The petition for rehabilitation psychology as a specialty notes that “Training in Rehabilitation Psychology is based upon a disability-specific body of theory and research (Cox, Cox, & Caplan, 2013; Dunn & Elliott, 2005) which focuses on the physical, psychological, social, environmental, and policy aspects of disability and rehabilitation, and includes individual, psychosocial, and cultural aspects of disability. Knowledge acquisition concentrates on the application of psychology principles to understanding the needs of diverse people with disabilities in rehabilitation settings and their families. Content focuses on understanding the impact of physical, cognitive, and/or mental health disabilities on diverse individuals and their relevance in providing rehabilitation services such as assessment, intervention, vocational rehabilitation, case management, and advocacy. Such training can occur in formal experiences, such as

didactics, seminars, and journal clubs, in interdisciplinary forums, such as interdisciplinary team-patient care meetings, case conferences and grand rounds, and in supervision, both individual and group” (APA Division 22 Rehabilitation Psychology and the Rehabilitation Psychology Synarchy, 2015, p. 21).

The American Board of Rehabilitation Psychology has also determined specialty competencies for practice in rehabilitation psychology that guide training in progress toward an individual’s attainment of board certification in the specialty (Cox et al., 2013). Such competencies include, but are not limited to, the ability to provide assessment and treatment related to adjustment to disability, cognitive functioning, family/couples functioning, pain, substance abuse, and educational, vocational, and recreational functioning; the ability to participate in interprofessional collaboration and consultation; knowledge of ethical, legal, and professional issues including laws related to persons with disability; knowledge of research and treatment and program evaluation methods as well as the ability to apply research findings to patients; and knowledge of and attention to diversity and cultural issues (Stiers & Nicholson Perry, 2012).

Summary

In this chapter, we have introduced rehabilitation psychology as a specialty within psychology, simultaneously recognizing its similarities with and differences from clinical health psychology. We have discussed practice aspects including assessment, intervention, and interdisciplinary team functioning that together allow rehabilitation psychologists to assist the team in treating the whole person. Research in rehabilitation psychology was reviewed, and the need for more theory-driven research was described. Advocacy and training in rehabilitation psychology, both vital engagement areas, were also described. While rehabilitation psychologists are involved to varying degrees in research, practice, advocacy, and training, the Division of Rehabilitation Psychology within the American Psychological Association provides a home for those interested in this discipline of psychology. The approval of rehabilitation psychology by the APA Council of Representatives in August 2015 as a recognized specialty within psychology demonstrates the importance of this distinct field, thereby recognizing the ability of rehabilitation psychology to treat the wounds that expose the “best and beautiful” aspects of our patients.

Author Biographies

Monica F. Kurylo, PhD, ABPP(Rp), is an associate professor and director of neurorehabilitation psychology in the Departments of Psychiatry and Rehabilitation Medicine at the Kansas University Medical Center. She received her doctorate in clinical psychology (health/rehabilitation emphases) and has internship and postdoctoral experience in rehabilitation psychology and neuropsychology. She is active in practice, training, advocacy, and research and has served on several local, state, and national professional psychology, rehabilitation psychology, and interdisciplinary groups (<http://www.kumc.edu/school-of-medicine/psychiatry-and-behavioral-sciences/faculty/clinical-psychologists/monica-kurylo-phd-abpp.html>).

Kathleen S. Brown, PhD, is a rehabilitation psychologist involved in consulting, leadership training, and supervision in her independent practice in Fort Myers, Florida. Dr. Brown’s clinical and research interests include pain management, interdisciplinary team development, leadership training in healthcare, and adjustment/coping responses to medical illness.

References

- American Psychological Association. (2012). Guidelines for assessment of and intervention with persons with disabilities. *American Psychologist*, 67(1), 43–62.
- American Psychological Association Division 22 Rehabilitation Psychology and the Rehabilitation Psychology Synarchy. (2015). *Petition for the recognition of a specialty in professional psychology*. Washington, DC: American Psychological Association.
- Americans With Disabilities Act of 1990, Pub. L. No. 101-336, 104 Stat. 328 (1990).
- Butt, L., & Caplan, B. (2010). The rehabilitation team. In R. G. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed., pp. 451–458). Washington, DC: American Psychological Association.
- Cheak-Zamora, N., Reid-Arndt, S. A., Hagglund, K. J., & Frank, R. G. (2012). Health legislation and public policies. In P. Kennedy (Ed.), *The Oxford handbook of rehabilitation psychology* (pp. 511–524). New York, NY: Oxford University Press.
- Chwalisz, K., & Dollinger, S. C. (2010). Evidence-based practice with family caregivers: Decision-making strategies based on research and clinical data. In R. G. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed., pp. 301–312). Washington, DC: American Psychological Association.
- Committee on Science, Engineering and Public Policy (COSEPUP). (2004). *Facilitating interdisciplinary research*. COSEPUP. Washington, DC: National Academies Press.
- Cox, D. R., Cox, R. H., & Caplan, B. (2013). *Specialty competencies in rehabilitation psychology*. New York, NY: Oxford University Press.
- Cox, D. R., Hess, D. W., Hibbard, M. R., Layman, D. E., & Stewart, R. K. (2010). Specialty practice in rehabilitation psychology. *Professional Psychology: Research and Practice*, 41(1), 82–88.
- Dijkers, M. P., Hart, T., Tsaousides, T., Whyte, J., & Zanca, J. M. (2014). Treatment taxonomy for rehabilitation: Past, present, and prospects. *Archives of Physical Medicine and Rehabilitation*, 95(1), S6–S16.
- Dunn, D. S., & Elliott, T. R. (2005). Revisiting a constructive classic: Wright's Physical Disability: A Psychosocial Approach. *Rehabilitation Psychology*, 50(2), 183–189.
- Dunn, D. S., & Elliott, T. R. (2008). The place and promise of theory in rehabilitation psychology research. *Rehabilitation Psychology*, 53(3), 254.
- Hart, T., & Ehde, D. M. (2015). Defining the treatment targets and active ingredients of rehabilitation: Implications for rehabilitation psychology. *Rehabilitation Psychology*, 60(2), 126.
- Heinemann, A. W., & Mallison, T. (2010). Functional status and quality-of-life measures. In R. G. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed., pp. 147–164). Washington, DC: American Psychological Association.
- Keany, K. C., & Glueckauf, R. L. (1993). Disability and value change: An overview and reanalysis of acceptance of loss theory. *Rehabilitation Psychology*, 38(3), 199–210.
- MacLachlan, M., & Mannan, H. (2014). The World Report on Disability and its implications for rehabilitation psychology. *Rehabilitation Psychology*, 59(2), 117.
- Meyerson, L. (1948). Physical disability as a social psychological problem. *Journal of Social Issues*, 4, 2–10.
- Novack, T. A., Sherer, M., & Penna, S. (2010). Neuropsychological practice in rehabilitation. In R. G. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed., pp. 165–178). Washington, DC: American Psychological Association.
- Patterson, D., & Hanson, S. (1995). Joint Division 22 and ACRM guidelines for post-doctoral training in rehabilitation psychology. *Rehabilitation Psychology*, 40, 299–310.
- Peter, C., Geyh, S., Ehde, D. M., Müller, R., & Jensen, M. P. (2015). Positive psychology in rehabilitation psychology research and practice. In S. Joseph (Ed.), *Positive psychology in practice: Promoting human flourishing in work, health, education, and everyday life* (2nd ed., pp. 443–460). Hoboken, NJ: Wiley.

- Stiers, W., & Nicholson Perry, K. (2012). Education and training in rehabilitation psychology. In P. Kennedy (Ed.), *The Oxford handbook of rehabilitation psychology* (pp. 417–431). New York, NY: Oxford University Press.
- Wegener, S. T., & Stiers, W. (2010). Prevention, assessment and management of work-related injury and disability. In R. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed., pp. 407–416). Washington, DC: American Psychological Association.
- Wright, B. A. P. (1983). *Physical disability, a psychosocial approach* (2nd ed.). New York, NY: Harper Collins Publishers.

Suggested Reading

- Dunn, D. (2014). *The social psychology of disability*. Academy of Rehabilitation Psychology Series. New York, NY: Oxford University Press.
- Kennedy, P. (Ed.) (2012). *The Oxford handbook of rehabilitation psychology*. New York, NY: Oxford University Press.
- Kerkhoff, T. R., & Hanson, S. L. (2013). *Ethics field guide: Applications to rehabilitation psychology*. Washington, DC: American Psychological Association.
- Rusin, M. J., & Jongsma, A. E. (2001). *The rehabilitation psychology treatment planner*. New York, NY: Wiley.

Administrative Issues in Primary Care Psychology

Esther N. Schwartz and David R. M. Trotter

*Department of Family and Community Medicine, Texas Tech University
Health Sciences Center, Lubbock, TX, USA*

Introduction

Psychologists and other behavioral health providers (BHPs) have become increasingly integrated into primary care in the United States (Bray, 2016; McDaniel et al. 2014; Miller, Petterson, Burke, Philips, & Green, 2014). The types of administrative issues encountered by BHPs working in primary care will vary by proximity of BHP and medical services locations and the level of integration between medical and behavioral health services. BHPs face administrative challenges that are distinct from providers in traditional mental health settings. In this article, the practicalities of BHPs' daily function such as their appointment structure, coding and billing for services, documentation, availability of administrative support, and position in the organizational hierarchy are discussed. Additionally, BHPs' navigation and resolution of ethical quandaries specific to psychological practice in a medical setting are also explored.

A Brief Context: Behavioral Health Providers in Primary Care Settings

Primary medical care (PC) is defined as “the provision of integrated accessible health care services by clinicians who are accountable for addressing a large majority of personal health care needs, developing a sustained partnership with patients, and practicing in the context of family and community” (Institute of Medicine, 1994, p. 1). PC clinics, including family medicine, general internal medicine, and general pediatrics practices, are the primary entry point for medical and mental healthcare for most Americans. Specifically, more than half (54.6%) of all healthcare visits occur with a primary care physician (PCP) (Centers for Disease Control [CDC], 2012). Integration of BHPs into PC is important as psychosocial and behavioral

factors such as diet, activity level, and substance use critically affect morbidity and mortality in the United States. Additionally, psychological symptoms and disorders are frequently comorbid with medical illnesses (Petterson et al., 2008). Finally, approximately 50–70% of Americans who receive treatment for mental and behavioral health and substance use problems do so from PC (McDaniel & deGruy, 2014). There is mounting evidence supporting that the integration of PC and behavioral health improves health at individual and population levels (Blount et al., 2007). However, primary care behavioral health integration is still not yet widespread.

Administrative Issues as a Function of Level of Integration

The types of administrative issues encountered by BHPs working in PC will vary at least partially as a function of the level of integration between medical and behavioral health services. In general, integration of services requires a team-based collaboration between all providers using a biopsychosocial framework (Bray, 2016; Engel, 1977). There are three broad levels of integration: coordinated, colocated, and fully integrated care (Bray, 2016). In coordinated care, the BHP retains a separate practice (i.e., separate physical spaces, electronic health record [EHR] systems, scheduling and billing staff) and collaborates via referrals from the PCP. In a colocated arrangement, the psychologist and PCP are located within the same office space but maintain separate practices (e.g., scheduling and billing staff, EHR) and financial arrangements (e.g., billing and reimbursement structures). In this arrangement the BHP accepts referrals from PCP and provides consultation (written and face to face). In a fully integrated model, the psychologist is embedded within the primary care system and is an integral part of care delivery and treatment. They share the same space, systems (e.g., EHR), and resources (e.g., scheduling and billing staff). In these settings, psychologists may provide immediate consults to patients during their doctor's visits as requested by the PCP, as well as see patients for brief interventions. For a full discussion of levels of integration, the reader should see Bray (2016). Deft negotiation of administrative issues requires an awareness of the administrative processes operating in each clinical setting and the level of behavioral health service integration.

Basic Practice Management Considerations for BHP Integration

General Practice Organization

BHPs working in colocated or fully integrated primary care settings typically work in a medical facility. Familiarity with the organizational hierarchy and the numerous players in the medical system is helpful for ensuring coordinated care. Key players in these facilities often include a medical director (a physician who makes decisions on standing orders, supervises “mid-levels,” and may provide general oversight of medical services), medical staff (e.g., physicians and mid-level providers like nurse practitioners and physicians assistants), clinic manager (a staff administrator who oversees the day-to-day operations of the clinic and provides general supervision of the staff), various department managers (e.g., nurse manager, billing manager, medical records manager, scheduler/call center manager, lab manager; typically report to the clinic manager), care coordinators (may coordinate referrals and/or manage patient care plans), and various departmental staff (e.g., nurses, billers/coders, call center staff). It is critical that a BHP understands where they fit into this hierarchy, including who they report to, who provides

practice oversight, how they interact with the other key players, and what services and support they will receive from the various clinic departments (e.g., scheduling assistance, billing/coding). For example, will the BHP report to the medical director and be granted privileges in accordance to their license/certification and in a manner similar to the other providers, or will they report to the clinic manager, be considered general staff, and be afforded fewer privileges? Discussions regarding how a BHP will fit into the administrative clinic structure is critical prior to integration.

General Clinic Supports

As stated above, BHPs should consider and negotiate how they will interact with, and what services they will receive, from the various clinic departments. For example, who will take charge of the day-to-day management of the BHPs schedule? Is the BHP responsible for scheduling of patients, taking their phone calls, and contacting them if they have not showed up, or does the scheduling department complete these tasks as they do for the medical providers? In full integration, the BHP's appointments are often marked on the same standardized weekly clinic grids utilized by all other providers. With these grids in place, the BHP's current appointments and total available slots in the future are visible to the entire clinic, increasing transparency and availability for same day access (Kearney, Smith, & Pomerantz, 2015). An additional question is, who does the BHP's billing and coding? Does this responsibility fall under the purview of the BHP's responsibilities, or is this handled by the billing/coding department in accordance with the other providers? It is advisable (when possible) for patients to have a generally equivalent experience (e.g., with scheduling, billing) with the clinic regardless of the type of services (i.e., medical or behavioral health) they are receiving.

Office/Clinic Space

Space in PC settings is often at a premium, and clinics are typically not designed with behavioral health practice in mind. Often, the only available space to see patients is an exam room, and BHPs may be expected to meet with patients in exam rooms. Additionally, it is the norm for many PC practices that providers do not have a private office space for charting and completing other administrative activities (e.g., letter writing, responding to patient messages) and may instead do these activities in a more central provider pod or work hub. While this may take some adjustment on the BHP's part, these centralized work spaces encourage communication between providers in ways that enhance coordination, builds camaraderie, and conveys accessibility. Even if a psychologist is designated a private office space to see patients, simply leaving the door open and being visible conveys accessibility (Robinson & Reiter, 2016). It is advisable that BHPs advocate for space that is roughly equivalent to that of their medical colleagues, use space judiciously, and be flexible.

Appointment Structure

Physician visits are typically scheduled in 15-min increments (high volume), while traditional mental health visits are typically scheduled for 50 min (comparatively low volume). When BHCs work in a coordinated model, they often retain this 50-min structure. However, when working in colocated or fully integrated settings, many move to using a 25-min session structure (a higher volume 16 total appointment slots in a day). When choosing an appointment structure (i.e., either 50 or 25 min), a number of administrative issues should be considered. First, what

are the clinical volume expectations in the clinic? Do the other providers expect you to have a volume more similar to theirs, or is there no expectation of this? If a BHC works in a clinic with many other providers, a 25-min structure is generally preferable as it allows the professional to be available for more of the PCP patients. Second, what level of staffing is available for patient scheduling and check-in/check-out? Are there enough scheduling staff to accommodate an additional 16 patients a day plus the calls they will generate? Finally, will the BHP utilize nursing staff to room patients? While this is not common, some clinical settings will provide nursing support for their BHP to room patients, allowing them to move faster through their clinic without having to make trips to the waiting room or ensure that patients have completed the appropriate paperwork. For a more comprehensive discussion and justification for the use of 25-min appointment structure, the reader may consult Robinson and Reiter (2016).

Documentation

Psychologists' documentation of patient encounters is an important administrative skill, but the style and processes of documentation differ based on level of BHC integration. In a coordinated model, BHP documentation will look similar to other mental health settings, with the addition of workflows and processes to ensure that the referring PCP is provided some level of feedback on the referral (e.g., that the patient was seen, that progress is being made). BHPs working in colocated models often document in a manner similar to traditional mental health providers (i.e., in a separate system, using traditional mental health note structures), while some document more like a BHP in a fully integrated setting. BHPs in fully integrated settings typically document in the same chart used by the PCP. This kind of communication requires that notes are written in a way that is useful to other healthcare team members. Typically, this means that the notes use a structure similar to other providers (e.g., SOAP style), are concise, do not contain psychological jargon, clearly convey the assessment of the patient including risk factors, include a follow-up plan, and provide recommendations for other providers (when necessary). When using this style of documentation, BHPs need to discuss the strengths and limitations of a shared health record during the informed consent discussion with the patient. The psychologist should also clarify with the patient what session information will be shared in the chart.

When using a shared chart, BHPs need to also consider who should have access to their notes. A PCP's notes are typically available to all staff and other providers, including other physicians and mid-levels, nursing staff, scheduling/call center staff, billers/coders, and other general administrative staff. A BHP using a shared EHR should carefully consider which of these groups will have a legitimate need to access to their portion record. For example, it would be important for the BHP to grant access to all referring providers in the clinic, as they will be collaborating on patients. Additionally, it may be important for some of the nursing staff to have access to these notes, as the nursing staff are often in charge of triaging patients who contact the clinic. However, it may not be necessary for scheduling staff and general administrative staff to have access to the notes. The BHP should give careful consideration to this issues and inform patients as to who will have access to their record.

Referral Management

In a coordinated practice, a BHP will receive referrals from a PCP's office, as well as from other sources. However, in colocated and fully integrated clinics, PCPs may be a BHP's sole

source of referrals. Ensuring that the referral process is easy, smooth, and rapid is critical in promoting PCPs' actual placement of the referral and will increase the likelihood that a patient will follow through with the initial appointment. BHPs should consider the process of placing written and in-person referrals. PCPs will be most familiar with creating written referrals, as they refer patients to a host of services (e.g., preventive care screenings, specialist referrals). As most PC practices use an EHR, BHPs should work closely with their IT departments to ensure that their electronic referral process closely matches those that PCPs use for other services. Additionally, BHPs should ensure that common referral barriers (e.g., requirement of prior authorization for service, delays, wait lists) are removed or managed. While PCPs will be most familiar with written referral processes, in-person referrals are one of the BHPs most effective tools for achieving coordinated care. The most effective in-person referral is the "warm hand-off." A warm hand-off is when a PCP consults the BHP in person and then asks the BHP to meet a patient during the patient's regularly scheduled PCP visit. Warm hand-offs by the PCPs to BHPs may be completed in just a few minutes and demonstrably increase the likelihood a patient will initiate therapy. In-person referrals may also occur during a "curbside consultation." A curbside consult is a brief discussion between a BHP and a PCP about a clinical issue that occurs spontaneously. These can occur in an exam room, PCP common work space, or hallway (being mindful of confidentiality issues). Curbside consults are good opportunities to provide accurate, specific, and actionable information on a behavioral problem and support the providers to be more effective in their interactions with patients. Over time, repeated consultations may provide physicians with a good sense of the biopsychosocial model in action and the range of services a psychologist provides. No less important, collaboration communicates to the PCP that they have a potential ally in caring for their patients (Gunn & Blount, 2009). Regardless of how a referral is placed, once a BHP has completed the initial referral visit, he or she should provide the referring PCP with feedback about what the patient is targeting in treatment and how treatment is progressing as a way to close the referral loop.

Billing and Reimbursement

BHPs working in either coordinated or colocated systems typically bill and are reimbursed in ways similar to that of traditional mental health settings. However, billing issues continue to be a serious hurdle in integrated primary care practices (Robinson & Reiter, 2016). Clinic administrators may hire a BHP with no plans for that provider to bring in direct revenue. In this model, the BHP is typically salaried hourly and paid as a member of the team. This system can work well in capitated payment structures, like those found in accountable care organizations (ACO). A capitated payment structure is a reimbursement model in which a health system receives a yearly flat fee from an insurance carrier to provide all services for a patient. Further, insurers may offer additional monetary incentives if the system can reduce overall healthcare costs for its patients by improving quality of care delivered (e.g., decreasing ER visits and hospital stays, reducing redundancy and waste). In fact, there is some evidence to suggest BHPs can reduce healthcare costs at a system level, making their inclusion in these systems attractive. See Blount et al. (2007) for a review of increased cost-effectiveness when behavioral health services are integrated into primary care.

Most BHPs still work in traditional fee-for-services models. BHPs working in these environments are typically either employees of the system who receive payment based on their billing or are contractors who provide services for a contracted fee. In fee-for-services systems, BHPs bill using current procedural terminology codes (CPT) to bill for services. There are two

categories of CPT codes available to BHPs: psychotherapy CPT codes (e.g., 90791) and health and behavior CPT codes (i.e., 96150–96155). The psychotherapy codes are used for billing when a BHP is seeing a patient for a mental health issue (e.g., depressive disorder, anxiety disorder). Available psychotherapy CPT codes include those used for testing and assessment and counseling (based on time; 30, 45, 60 min). The CPT codes are used by those working in traditional mental health settings as well as all levels of PCBH integration. Health and behavior codes allow BHPs to bill for services that address social and behavioral aspects of physical health problems as diagnosed by a medical professional (Robinson & Reiter, 2016). Therefore, a BHP can bill for services to improve the course or outcome of medical issues like obesity, chronic pain, and diabetes. It is important to note that billing for both health and behavior codes and mental health codes in the same encounter is not allowed.

Managing Confidentiality

Protecting patient confidentiality and privacy in the primary care setting is an important administrative issue but can be challenging. Physicians and BHPs have different laws, rules, and ethics codes that govern provider–patient confidentiality. For example, medical care is organized around referrals and informal consultation with others in which protected health information is exchanged. This is permissible under the Health Insurance Portability and Accountability Act (HIPAA), which allows HIPAA-covered healthcare providers to disclose protective health information with another healthcare provider without the patients written consent, if the exchange of information pertains to that provider’s treatment of the patient (see Federal Regulation 45 CFR 164.506). As such, PCPs expect team members to be able to discuss all patient care issues as deemed necessary. However, BHPs may be limited in their ability collaborate in this way as they are not covered under HIPAA and are subject to other privacy and ethical considerations. For example, the American Psychological Association Ethical Standard 4.06 clearly states that when consulting with colleagues, psychologists “do not disclose confidential information that reasonably could lead to the identification of a client/patient.” Therefore, a curbside consult between a PCP and a psychologist BHP in which a patient is referred to by name and medical problem may be permissible under HIPAA, but in violation of APA standards on confidentiality. However, in the primary care culture of openly sharing patient information as a way to coordinate care, a psychologist’s reticence to collaborate may be counterproductive (Gunn & Blount, 2009). BHPs must learn to navigate the complex rules, regulations, and work expectations regarding confidentiality. One of the simplest administrative solutions to this issue is to notify patients in writing during the informed consent process that the BHP may collaborate with other providers in the service of their care. Further, during informed consent, patients can be informed about how their written record will be handled (see above).

Managing Multiple Relationships

Medical providers and BHPs may possess different perspectives on the issue of multiple relationships. For example, consider the example of a provider providing and/or receiving clinical services to/from another colleague or trainee. The APA Ethical Code states: “A psychologist refrains from entering into a multiple relationship if the multiple relationship could reasonably be expected to impair the psychologist’s objectivity, competence or effectiveness in performing

his or her functions as a psychologist, or otherwise risks exploitation or harm to the person with whom the professional relationship exists” (see Standard 3.05 in the “Ethical Principles of Psychologists and Code of Conduct”; American Psychological Association [APA], 2017). While APA’s code does not explicitly forbid treating colleagues or professionals in training under this code, the onus would fall on the psychologist to prove that entering into such a relationship would be reasonable, which would be difficult to do. On the other hand, the American Medical Association’s (AMA) Code of Ethics stipulates that while one should exercise caution (for issues similar to those raised by the APA), “car[ing] for a fellow physician is a privilege” and that “physicians-in-training should not be required to provide medical care to fellow trainees, faculty members, or attending physicians if they are reluctant to do so.” Taking these guidelines in combination in a real-world setting, BHPs and PCPs may have very different opinions about the appropriateness of working with a patient with whom both providers have multiple relationship (e.g., colleagues, family members, relatives of current patients). Managing multiple relationships as a BHP is an important administrative consideration, as it will influence the referrals (and associated referral process) that a BHP will or will not accept. Here we will discuss the two types of multiple relationships BHPs will most often encounter and potential administrative solutions.

Working with Colleagues

Psychologists are trained communicators with expertise in emotions, relationships, and behavior. They are in an apt position to promote team development and a healthy culture in the workplace (McDaniel & Fogarty, 2009). In primary care, the psychologist may be called on to help staff deal with difficult and divisive patients, identify staff burnout, and improve workflows (McDaniel & deGruy, 2014). In some situations, clinic staff may ask the psychologist for help with a personal problem. In such scenarios, a BHP must adopt a policy(s) that will help them navigate these requests. These policies are likely to fall on a spectrum, ranging from a refusal to see any colleagues as patients to a policy in which the BHP will see anyone for an initial appointment. If a BHP adopts a policy refusing to see colleagues as patients, this is best communicated in the beginning when negotiating the terms of a practice. It is always easier to adopt this policy from the beginning, rather than attempt to implement it later on. This policy should then be clearly communicated to providers and staff. If a BHP adopts a policy on the other end of the spectrum, prior to intervening, it is recommended that the psychologist consider his or her working relationship with the staff member, the severity and sensitivity of the problem, and the likelihood the consultation will negatively impact the working relationship between staff member and BHP and the team (Reiter & Runyan, 2013). Again, if a BHP chooses such a policy, this should be communicated to providers and staff in a clear format. Regardless of the policies guiding a BHP’s practice, in the event of crises or problems with elevated risk to the staff member, the BHP may advise follow-up care such as counseling or hospitalization. Additionally, all services rendered to colleagues should be documented in their chart in a fashion similar to documenting other patients’ information.

Non-Colleague Patients

Issues of multiple relationships are also common when working with non-colleague patients. In primary care, it is not uncommon for members of the same family to receive care in the same clinic and even from the same provider (“whole family care”). PCPs may be skilled at navigating the challenges inherent in these multiple relationships, but such scenarios are far

less common in specialty mental health settings and may present ethical and administrative challenges to psychologists (Reiter & Runyan, 2013). Consider the plausible example of an elderly man referred to a BHP to address suicidal ideation and his adult daughter who is referred to the same BHP for a distinct but related issue, stress-induced gastritis. In this scenario, if the BHP were to follow the APA Standard 10.02 Therapy Involving Couples of Families stating “When psychologists agree to provide services to several persons who have a relationship (such as spouses, significant others, or parents and children), they take reasonable steps to clarify at the outset (1) which of the individuals are clients/patients and (2) relationship the psychologist will have with each person,” then the BHP would risk violating APA Standard 4.01 Maintaining Confidentiality, which instructs psychologists to “take precautions to protect confidential information.” “Whole family care” presents an ethical dilemma for the BHP as it is impossible to abide by one of these guidelines without violating the other (Reiter & Runyan, 2013). In this case, the BHP may not even be aware that father and daughter are related when they are referred. In the event that the BHP is made aware of this existing relationship, several different administrative actions can be taken. First, if other BHPs are available at the clinic, it would be advisable to refer one of the family members to see another BHP. If there is only one BHP, then they should assess the extent to which the father’s presenting issue and the daughter’s presenting issue are reasonably discrete and can each be conceptualized and treated without undue consideration of the other. Based on this assessment, the BHP will have to prioritize which standard will take precedence and be prepared to provide a rationale for this decision.

Thus, even when embedded within the primary care team, psychologists’ administrative challenges are unique from medical providers. Moreover, primary care psychologists’ practice management, reimbursement process, and understanding of confidentiality and multiple relationships differ from their colleagues working in traditional mental health settings.

Author Biographies

Esther N. Schwartz, PhD is a postdoctoral fellow in primary care psychology in the Department of Family and Community Medicine at the Texas Tech University Health Sciences Center. Dr. Schwartz received her PhD in counseling psychology from the Department of Psychological Sciences at Texas Tech University and completed an APA-accredited predoctoral internship in psychology at the Dallas VA Medical Center.

David R. M. Trotter, PhD is assistant professor and director of behavioral sciences in the Departments of Family and Community Medicine and Medical Education at the Texas Tech University Health Sciences Center. Dr. Trotter earned his PhD in clinical psychology at Texas Tech University and received postdoctoral training in primary care psychology at the UMass Medical School, Center for Integrated Primary Care (Department of Family Medicine). His areas of expertise include behavioral medicine, primary care behavioral health integration, and medical education.

References

- American Psychological Association. (2017). *Ethical principles of psychologists and code of conduct*. Retrieved from www.apa.org/ethics/code
- Blount, A., Schoenbaum, M., Kathol, R., Rollman, B., Thomas, M., O’Donohue, W., & Peek, C. J. (2007). The economics of behavioral health services in medical settings: A summary of the evidence. *Professional Psychology: Review and Practice*, 38, 290–297. doi:10.1037/0735-7028.38.3.290

- Bray, J. H. (2016). Primary care setting. In J. C. Norcross, G. R. VandenBos, & D. K. Freedheim (Eds.), *APA handbook of clinical psychology: Vol. 1 roots and branches* (pp. 463–475). Washington, DC: American Psychological Association. doi:10.1037/14772-026
- Centers for Disease Control. (2012). *National ambulatory medical care survey: 2012 summary tables 1.11.16*. Atlanta, GA. Retrieved from <https://www.cdc.gov/nchs/fastats/physician-visits.htm>
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196, 129–136. doi:10.1126/science.847460
- Gunn, W. B., & Blount, A. (2009). Primary care mental health: A new frontier for psychology. *Journal of Clinical Psychology*, 65, 235–252. doi:10.1002/jclp.20499
- Institute of Medicine. (1994). *Defining primary care: An interim report*. Washington, DC: National Academy Press.
- Kearney, L. K., Smith, C. A., & Pomerantz, A. (2015). Capturing psychologist' work in integrated care: Measuring and documenting administrative outcomes. *Journal of Clinical Psychology in Medical Settings*, 22, 232–242. doi:10.1007/s10880-015-9442-7
- McDaniel, S. H., & deGruy, F. V. (2014). An introduction to primary care and psychology. *American Psychologist*, 69(4), 325–331.
- McDaniel, S., & Fogarty, C. (2009). What primary care psychology has to offer the patient-centered medical home. *Professional Psychology: Research and Practice*, 40, 483–492. doi:10.1037/a0016751
- McDaniel, S. H., Grus, C. L., Cubic, B. A., Hunter, C., Kearney, L., Lisa, K., ... Johnson, S. B. (2014). Competencies for psychology practice in primary care. *American Psychologist*, 69, 409–429. doi:10.1037/a0036072
- Miller, B. F., Petterson, S., Burke, B. T., Phillips, R. L., Jr., & Green, L. A. (2014). Proximity of providers: Colocating behavioral health and primary care and the prospects for an integrated workforce. *American Psychologist*, 69, 443–451. doi:10.1037/a0036093
- Petterson, S. M., Phillips, B. L., Jr., Bazemore, A. W., Dadoo, M. S., Zhang, X., & Green, L. A. (2008). Why there must be room for mental health in the medical home. *American Family Physician*, 77, 757.
- Reiter, J., & Runyan, C. (2013). The ethics of complex relationships in primary care behavioral health. *Families, Systems & Health*, 31, 20–27. doi:10.1037/a0031855
- Robinson, P., & Reiter, J. T. (2016). *Behavioral consultation and primary care: A guide to integrating services* (2nd ed.). Basel, Switzerland: Springer.

Suggested Reading

- Bray, J. H. (2016). Primary care setting. In J. C. Norcross, G. R. VandenBos, & D. K. Freedheim (Eds.), *APA handbook of clinical psychology: Vol. 1 roots and branches* (pp. 463–475). Washington, DC: American Psychological Association. doi:10.1037/14772-026
- McDaniel, S. H., Grus, C. L., Cubic, B. A., Hunter, C., Kearney, L., Lisa, K., ... Johnson, S. B. (2014). Competencies for psychology practice in primary care. *American Psychologist*, 69, 409–429. doi:10.1037/a0036072
- Robinson, P., & Reiter, J. T. (2016). *Behavioral consultation and primary care: A guide to integrating services* (2nd ed.). Basel, Switzerland: Springer.

Affective Forecasting in Health Psychology

Shannon M. Martin¹, James I. Gerhart²,
Catherine Rochefort³, Laura Perry⁴, and Michael Hoerger⁴

¹*Department of Psychology, Central Michigan University, Mount Pleasant, MI, USA*

²*Department of Psychosocial Oncology, Cancer Integrative Medicine Program,
Rush University Medical Center, Chicago, IL, USA*

³*Department of Psychology, Southern Methodist University, Dallas, TX, USA*

⁴*Department of Psychology, Tulane University, New Orleans, LA, USA*

Affective forecasting, the process of predicting future emotional states, often drives decision making. Individuals routinely make decisions based on what they believe will optimize their happiness or general emotional well-being, such as whether to undergo or avoid a medical procedure that may cause some degree of distress. Unfortunately, affective forecasts are prone to a number of errors that may bias decision making. For example, in general people tend to overestimate the intensity and duration of future emotional states. Applied to healthcare behaviors, overestimating future negative emotionality surrounding proactive health behaviors may lead individuals to avoid medical treatments that may actually serve to improve their health and well-being. This entry reviews basic research on affective forecasting and discusses a number of important implications for health.

Affective forecasting research grew out of investigations of the disability paradox, or the tendency for individuals to overestimate the negative emotional impact of illness and disability (Albrecht & Devlieger, 1999). Although healthy individuals tend to predict catastrophic reductions in quality of life in response to disability, those actually living with disability often adapt to illness in a manner that affords a meaningful life. This work stemmed in part from late twentieth-century researchers such as Levine and Antonovsky who were interested in how quality of life could be obtained in the face of adversity. Around this time other researchers such as Albrecht and Higgins observed discrepancies between patient-rated well-being and their physical status. In the intervening years, numerous studies documented that healthy individuals overestimate the emotional impact of life-threatening illness such as cancer and noted a reasonable degree of well-being is possible at the end of life. The disability paradox

may arise from several social psychological processes including a tendency disregard the impact of coping and adaptation while maintaining a hyper-focus on isolated symptoms and subsequent impairment.

The interesting and curious findings from the disability paradox yielded further and more rigorously controlled studies. A number of social and cognitive psychologists attempted to better understand how and why people overpredict the intensity of future emotional states by utilizing basic research methods to build upon the important but sometimes anecdotal evidence for the disability paradox. Understanding affective forecasting errors is an important topic of study for these fields as predictions about how the future will unfold are directly relevant to decision making. That is, the degree to which people base immediate choices and actions on predictions about how they believe they will feel highlights the intersection of emotional forecasts and decision making. Further, if an individual's decision making is driven by current beliefs about future emotional states, these decisions may be based on inaccurate information. Not surprisingly, greater inaccuracy in future expectation leads to biased decision making across a range of day-to-day decisions, from how to spend free time to consequential decisions such as who to choose as a romantic partner or whether to pursue a particular medical treatment. For example, if an individual predicts that undergoing a particular medical procedure will lead to increased pain or prolonged stress, he or she is not likely to pursue that option, even if it might be better for their longer-term well-being or longevity.

In order to systematically study the influence of affective forecasts on decision making, researchers measure at least two major components of experience: predicted and experienced affect. First, participants make predictions about how they expect to feel in response to a future event, whether it be their favored sports team winning or losing a game, an important career event (e.g., a professor earning or being denied tenure), or a major health event (e.g., being diagnosed with HIV). These expectations are assessed in advance of the event, and then researchers measure experienced affect during the event or shortly afterward. The predicted and experienced affect ratings are then compared in order to quantify the relative accuracy of the forecasts. Additional correlates of accuracy such as personality traits are often measured, or situational variables may be modified to intentionally influence accuracy.

In general, people tend to overestimate the duration and intensity of future emotional states, regardless of whether the events are positive or negative in valence, a phenomenon called the impact bias. The impact bias is especially prevalent when individuals make forecasts about future negative life events. When experiencing negative events, individuals utilize numerous coping mechanisms (e.g., motivated reasoning, rationalization, self-justification, and other aspects of the "psychological immune system") that combat negative emotional reactions in order to help restore a sense of well-being. As people tend to disregard this active healing process when they are making predictions about their responses to a negative future event, they overestimate the effect of that event on their future emotions, a phenomenon called immune neglect (Gilbert, Pinel, Wilson, Blumberg, & Wheatley, 1998). For example, a study looked at voters' affective forecasts if their favored candidate lost an election, as well as their evaluations of the winning candidate before and after the election. Not only were voters' predictions about how they would feel if their candidate lost the election more extreme than how they felt when that event actually occurred, but they also evaluated the winning candidate more favorably after the election than they had before the election. Furthermore, the voters were unaware of this change in their views regarding the winning candidate from before the negative event to after it, which may have served as a coping mechanism to reduce their experience of negative emotions. These findings are especially important to consider when making choices regarding future potentially uncomfortable or stressful events, such as difficult medical

decisions. Although individuals have a tendency to fear the worst, many are later surprised that an unpleasant outcome was less intense or long lived as anticipated.

Additionally, when making an affective forecast, people tend to focus solely on the primary event in question, a common affective forecasting bias termed focalism (Wilson, Wheatley, Meyers, Gilbert, & Axson, 2000). By doing so, individuals fail to acknowledge other simultaneously occurring events that may serve to diminish the impact of any single event on their emotions. For example, when predicting how they would feel if their school's football team lost or won a game, college students may not consider other day-to-day occurrences that could affect their emotional well-being during the same time span and therefore overestimate their emotional reactions to the outcome of that football game. However, if individuals are asked to list activities they engage in during a typical day (e.g., going to class, eating meals, studying, socializing with friends) prior to making a forecast about the outcome of the game, the prediction tends to be less extreme. Simply stopping and thinking about life events other than the focal event mitigates the influence of focalism and leads people to make more accurate affective forecasts.

Researchers have identified additional errors in affective forecasts beyond immune neglect and focalism. For example, affective forecasts are based on one's present understanding of an event in question. Therefore, inaccurate conceptions of an event that one has never before experienced (misconstrual) or an inaccurate memory of emotional responses to an event that one has experienced before (inaccurate theories) may lead people to make errors in their affective forecasts.

A number of individual difference variables can also influence affective forecasts (Hoerger, Quirk, Lucas, & Carr, 2009). Researchers determined that factors such as personality traits, attachment style, or mood at the time of the forecast are important predictors of the intensity of forecasts and/or accuracy. For example, neuroticism, or the relatively enduring tendency to experience negative emotional states across situations, is typically associated with greater predicted and experienced negative affect. Similar findings have been found for those scoring higher in introversion. Recent research has suggested that personality factors may explain about 30% of the correspondence between predicted and experienced affect. Other traits, such as scoring higher in emotional intelligence, particularly in the domain of emotional management, is associated with making more realistic predictions about future emotional states. In addition, affective forecasting accuracy can be influenced by one's mood state. People who experience negative affect during the time of affective forecasting have been shown to overpredict the extent of their negative reactions to future events (dysphoric forecasting bias).

Although there is a large body of research on affective forecasting and impact bias in the general population, researchers know less about how affective forecasting relates to health-specific circumstances. However, understanding how these phenomena manifest in medical settings may be key to understanding how patients make important healthcare decisions that have serious consequences for their health and quality of life. For example, a large portion of the adult population may avoid colorectal cancer screenings, putting themselves at greater risk of developing colorectal cancer, because they overpredict the extent of embarrassment and pain they will experience during the procedure. They may not imagine the event accurately (misconstrual) compared to actual patient reports of the procedure. Additionally, men with prostate cancer may choose an unnecessary aggressive or surgical treatment instead of watchful waiting (a more conservative option appropriate in many cases) because they focus too much on, and thus overpredict, the negative effect of "living with cancer" on their emotional well-being and fail to consider the consequences these treatments may have on their day-to-day

functioning (focalism), such as incontinence and impotence. In both of these examples, patients' inaccurate affective forecasts may lead them to make less satisfying healthcare decisions. This is one reason why affective forecasting research needs to expand beyond the social cognitive perspective, which has historically focused on samples of White, healthy college students, and draw upon the health psychology perspective in order to understand and improve healthcare decision making.

Affective forecasting biases, such as focalism and immune neglect, play a significant role in many patients' health decision-making processes. Many health decisions can be understood as preference sensitive, meaning that the available options have comparable outcomes, unknown differences in outcomes, or outcomes that vary considerably across individuals. In absence of compelling evidence favoring one option over another, these decisions are often driven by affective forecasting. Next, we will discuss examples of preference-sensitive decision making across the health continuum, including decisions about health behaviors, illness screening, treatment, disease management, and end-of-life care.

One example of preference-sensitive decisions involves the decision to engage in health-related behaviors, such as exercise, alcohol consumption, or vaccinations. Using vaccination as an example, preference-sensitive decision making often influences people's decisions to get non-required vaccines, such as flu or HPV vaccines. Affective forecasting errors, such as expectation biases, can lead people to believe that post-vaccination pain will be greater or more bothersome than what it is in reality. Additionally, anticipating future worry or regret can lead patients to get vaccinations, especially if contracting the flu or HPV is forecasted to be of greater emotional impact. Furthermore, patients focusing on the anticipated regret of increasing their risk of cervical cancer may seek the HPV vaccination to avoid that future regret. Deciding against vaccinations based on forecasting errors can negatively influence a patient's health.

Other health-related examples include screening children for adult-onset illnesses and early screening for colorectal cancer. Regarding screening children for adult-onset illnesses, parents may prefer to delay screening due to immune neglect in which parents think that the child would not be able to cope with screening results. In regard to the age at which regular colorectal cancer screening should begin, patients may prefer to begin screening earlier than recommended due to forecasting errors in which they believe that the emotional impact of an abnormal screen will be greater than in reality. Conversely, patients might avoid colorectal cancer screenings altogether if overestimating the distress evoked by the screening process. Screening is an important aspect of preventative healthcare that can be significantly influenced by affective forecasting.

Affective forecasting also plays a role in preference-sensitive prevention and treatment decisions, such as starting preventative breast cancer medications and treating low-risk prostate cancer. Preventative breast cancer medications, such as tamoxifen, may reduce patient's risk of developing breast cancer, but patients often choose against these medications if they believe it will increase their health-related stress. Additionally, options for treating low-risk prostate cancer include (a) watchful waiting, in which patients and physicians monitor the cancer's progression; (b) surgery, after which urinary incontinence may occur; or (c) chemotherapy, in which debilitating side effects may occur. Some patients may choose watchful waiting because of difficulties recognizing their ability to cope with side effects or an overestimation of the intensity and duration of surgery or chemotherapy side effects. Other patients may choose surgery or chemotherapy because of difficulties recognizing their ability to adapt to a watchful waiting lifestyle and the potential anxiety of knowing cancer is present. In both of these scenarios, the pivotal factor is that challenges anticipating coping skills under varying

circumstances can lead people to make decisions they later regret. Clearly, more research is needed to determine how to help patients to make forecasts more accurately.

In chronic disease management, preference-sensitive decisions can also be influenced by affective forecasting errors. For example, patients with kidney problems may need to choose between dialysis and an organ transplant. One patient may choose dialysis due to an underestimation of their ability to adjust to stress and post-transplant protocols, such as taking immunosuppressant medications, but another patient may choose a kidney transplant because he or she overestimated the extent of future improvements to their health after a transplant. Forecasting errors in disease management can influence which decisions a patient believes are preferable, but very little is known about the relative accuracy of affective forecasts in disease contexts, let alone individual differences or situational factors that may augment accuracy.

Affective forecasting also influences preference-sensitive decisions at the end of life. Patients may decide against palliative care if they overestimate the distress of palliative care consultations that are frequently viewed as “giving up,” rather than empathic conversations aimed at improving quality of life. Conversely, some patients may decide against disease-directed therapy if they overestimate the emotional toll of aggressive end-of-life treatments. In either situation, patients may be underestimating their ability to cope with side effects and/or overestimating the extent to which they will have to adjust to palliative or disease-directed care. More attention and investigation is needed to help patients and their families understand and make these difficult decisions.

Notably, patients are not the only individuals whose decisions are influenced by affective forecasting errors. Focalism and immune neglect may also influence families of patients and physicians in ways that affect the patient’s decision making. For example, family members may underestimate a patient’s ability to adjust and overestimate the emotional duration of a side effect. In these situations, family members’ difficulties in affective forecasting could sway patients’ healthcare decisions.

In the context of medical decision making, individuals often heavily weigh the perceived emotional consequences as they consider between alternative treatment plans. To the extent to which individuals are biased toward overestimating negative emotional consequences, they may be less inclined to follow through with a potentially beneficial treatment. Therefore, basic and applied researchers have explored interventions to help mitigate decisional bias resulting from the disability paradox and affective forecasting errors. For instance, Ubel and colleagues have demonstrated that prompting individuals to reflect on adaptation may lead to increased estimates of quality of life with paraplegia. However, it is unclear how these processes may generalize to medical decision-making situations where patients must choose between various treatment options.

Emotional well-being plays a pivotal role in motivating health behavior. The perceived enjoyment of health behaviors tends to be more important predictors of future behavior compared with rational assessments of risk and benefit (e.g., continuing to smoke for enjoyment despite recognizing the risks of lung disease; Loewenstein, Weber, Hsee, & Welch, 2001). Women at high risk for breast cancer who perceive limited emotional benefit from chemoprevention are less inclined to opt for those therapies. Because emotional outcomes to novel events can be hard to predict, it is possible that patients may benefit from more explicit information on how they may feel following various treatment options. Basic research by Gilbert, Killingsworth, Eyre, and Wilson (1999) found that prediction of response to anticipated events could be enhanced by gathering information from others who had already experienced those events. Similar applied research by Dillard and colleagues suggests that forecasting errors regarding colorectal cancer screening can be reduced by providing individuals with

patient narratives about their experience with the screening process. The impact of these narratives on participants' attitudes toward screening was significantly more favorable compared with participants who did not receive the narratives.

Although anecdotal information and patient narratives may hold some influence over medical decision making, additional research is needed to determine how information could be derived and delivered. Consistent with the goals of the Patient-Centered Outcomes Research Institute (PCORI), clinical trials are needed to assess the differential impact of medical treatments on emotional outcomes so that this information can be shared with prospective patients and reduce uncertainty about emotional well-being. There is also a need to further explore how this information would be shared with patients in clinical settings. For instance, routine distress screening is now standard care, but healthcare professionals vary widely in their willingness and competence in addressing patient concerns. If comparative efficacy data on emotional well-being can be routinely tracked and providers can be coached to deliver this information with compassion and competence, patients may be guided to make better informed decisions.

While much of this discussion has focused on affective forecasts about specific life events, it is also important to emphasize that relatively generalized affective forecasts about the future—that life is getting better or worse—can have important implications for decision making, perhaps regardless of the accuracy of these expectations. Affective forecasting studies have often focused on predictions about the relatively immediate emotional consequences of events that are highly discrete, and often controllable, with the assumption that if people could forecast more accurately, they might make different and more satisfying life decisions. In the health arena, some events are relatively discrete (e.g., screening tests, influenza vaccinations, surgery). Other health events are significantly more complex such as the development and treatment of many chronic health conditions, which unfold gradually over time, are multidetermined by factors that vary in controllability, and are potentially influenced by decades of decisions. Thus, while people have specific affective forecasts relevant to relatively discrete events, they also have more generalized affect forecasts about the emotional consequences of aging, changing health trajectories, or the future, more globally. Along these lines, fuzzy trace theory (Reyna, 2008), for example, emphasizes the importance of people's relatively intuitive, gist-level understanding of life events (e.g., “the future is terrifying,” “I can't handle cancer and a divorce,” or “I'm lucky they caught it early”) for decision making. These generalized affective forecasts refer to the more nebulous and long-term expected trajectories in emotional well-being, rather than the relatively short-lived affective reactions often studied under the social cognitive paradigm. When people experience a generalized expectation of improved emotional well-being, this has often been called “hope,” which contrasts with the generalized negative expectancy, or “dread.” Regardless of accuracy, the valence of these generalized expectations—relative hope or dread about the future—could have serious implications for decision making.

The importance of generalized affective forecasts about the future—the experience of hope or dread—has known importance to health and is potentially a fruitful path for further investigation. As summarized by crisis decision theory, which has organized several useful frameworks for understanding emotion and health decision making, people routinely manage negative anticipatory emotions by mentally and behaviorally disengaging. Thus, when people feel more hopeful about the future, they may be more inclined to engage in proactive health behaviors, but when experiencing dread, worry, or hopelessness, people may be more inclined to avoid proactive health behaviors. Several studies have shown, for example, that people who are more hopeful are more likely to have better medication adherence, seek cancer screenings, engage in exercise, and maintain healthy diets. Patients who are more hopeful may also be

more likely to engage in “shared decision making,” a collaborative process by which patients actively participate in preference-sensitive decisions. These sorts of affective forecasts may also have important implications for suicide and other escape–avoidance behaviors. Whereas suicidal ideation and suicide attempts in young adults often receive considerable attention, older adults are at the greatest risk of completed suicide, with the highest rates among older White males, especially those recently diagnosed with a serious health condition and when in the context of other life stressors, such as divorce and unemployment. The experience of hopelessness is a leading predictor of completed suicide in older adults, suggesting the importance of generalized affective forecasts about changing health circumstances in accounting for suicidal behavior.

These findings help to illustrate an important unanswered question in affective forecasting research: what constitutes a “good” affective forecast? The social cognitive paradigm has traditionally focused on accuracy, but as emphasized in Funder’s (1995) realistic accuracy model of human judgment, there are other possibilities. One might define the relative goodness of an affective forecast not based on accuracy, but some other criterion, such as whether it is hopeful, benefits current emotional well-being, fosters particular decisions about health and health-care, or has a beneficial impact on longevity. The impact bias suggests that in light of declining health circumstances, people may be inaccurately negative in their perceptions of the future; thus, helping patients to forecast more accurately is essentially synonymous with helping them to be more positive and hopeful. In contrast, near the end of life, conversations about prognosis are often extremely overly optimistic, and this increased positivity could represent a departure from accuracy with potentially serious implications. For example, while overoptimism may have immediate emotional benefits, it has been hypothesized to be an important driver of overtreatment, lower quality of death, poorer family bereavement, and medical bankruptcies. Therefore, in the context of end-of-life care, there is an important debate over the relative importance of realistic versus optimistic communication, the relative benefits of accuracy versus positivity, and even the meaning of the construct “hope” itself. Many have advocated for approaches that, while difficult, seek to increase accurate perceptions of the future while simultaneously fostering a sense of hope and controllability (variously described as “realistic hope,” “deep hope,” and “blues-inflicted hope”). Ultimately, there are many important open questions about the ways in which affective forecasting drives decision making and health.

In conclusion, this section demonstrates the importance of basic research on affective forecasting for understanding decision making across the healthcare continuum. More research is needed to quantify the significance of affective forecasting in medical populations and examine interventions for improving affective forecasting, decision making, and healthcare outcomes.

Acknowledgments

This work was supported by U54GM104940 from the National Institute of General Medical Sciences (MH).

Author Biographies

Shannon M. Martin, PhD, is an assistant professor of psychology at Converse College in Spartanburg, South Carolina, USA. Her research interests include the measurement of maladaptive emotional response and its impact on everyday life.

James I. Gerhart, PhD, is an assistant professor of behavioral sciences and the director of the Psychosocial Oncology and Cancer Integrative Medicine Program at Rush University Medical Center, Chicago, Illinois, USA. He studies the impacts of anger and traumatic stress on medical decision making and patient–provider communication.

Catherine Rochefort, MS, is a clinical psychology doctoral student in the Health Behavior Lab at Southern Methodist University in Dallas, Texas, USA. She studies the influence of affect on health behavior change and adjustment to chronic illness.

Laura M. Perry, BS, is a health psychology doctoral student in the Psycho-Oncology Research Program at Tulane University in New Orleans, Louisiana, USA. Her research interests include how personality, affective, and social factors relate to cancer outcomes.

Michael Hoerger, PhD, MSCR, is an assistant professor of psychology, psychiatry, and oncology and director of the Psycho-Oncology Research Program at Tulane University in New Orleans, Louisiana, USA. His research aims to improve quality of life by anticipating, preventing, and alleviating the physical and emotional burden of cancer.

References

- Albrecht, G. L., & Devlieger, P. J. (1999). The disability paradox: High quality of life against all odds. *Social Science and Medicine*, 48, 977–988.
- Funder, D. C. (1995). On the accuracy of personality judgement: A realistic approach. *Psychological Review*, 102, 652–670.
- Gilbert, D. T., Killingsworth, M. A., Eyre, R. N., & Wilson, T. D. (1999). The surprising power of neighborly advice. *Science*, 323, 1617–1619.
- Gilbert, D. T., Pinel, E. C., Wilson, T. D., Blumberg, S. J., & Wheatley, T. P. (1998). Immune neglect: A source of durability bias in affective forecasting. *Journal of Personality and Social Psychology*, 75, 617–638.
- Hoerger, M., Quirk, S. W., Lucas, R. E., & Carr, T. H. (2009). Immune neglect in affective forecasting. *Journal of Research in Personality*, 43, 91–94.
- Loewenstein, G. F., Weber, E. U., Hsee, C. K., & Welch, N. (2001). Risk as feelings. *Psychological Bulletin*, 127, 267–286.
- Reyna, V. F. (2008). A theory of medical decision making and health: Fuzzy trace theory. *Medical Decision Making*, 28, 820–865.
- Wilson, T. D., Wheatley, T. P., Meyers, J. M., Gilbert, D. T., & Axson, D. (2000). Focalism: A source of durability bias in affective forecasting. *Journal of Personality and Social Psychology*, 78, 821–836.

Suggested Reading

- Halpern, J., & Arnold, R. M. (2008). Affective forecasting: An unrecognized challenge in making serious health decisions. *Journal of General Internal Medicine*, 23, 1708–1712.
- Hoerger, M., Scherer, L. D., & Fagerlin, A. (2016). Affective forecasting and medication decision making in breast cancer prevention. *Health Psychology*, 35, 594–603.
- Wilson, T. D., & Gilbert, D. T. (2005). Affective forecasting: Knowing what to want. *Current Directions in Psychological Science*, 14, 131–134.

Aging

Ann Rosenthal¹, Dara Mickschl¹, Edith Burns¹,
Elizabeth Neumann¹, Jay Urbain², Sergey Tarima¹,
and Steven Grindel¹

¹*Medical College of Wisconsin, Milwaukee, WI, USA*

²*Milwaukee School of Engineering, Milwaukee, WI, USA*

Background

Rotator cuff injuries are one of the most common joint ailments plaguing older adults today (e.g., Raymond, Sluka, McGowan, & American Academy of Orthopaedic Surgeons, 2011). This chapter describes the clinical epidemiology of shoulder impairment in older adults, standard approach to management, and the current ambiguity of the literature on the effectiveness of interventions for optimizing long-term outcomes. Preliminary data is presented grounded within a theoretical model, supporting the hypothesis that patients use “out-of-date” self-prototypes to establish expectations for outcomes, which has significant implications for long-term self-management behaviors and may explain lack of adherence to therapy. Finally, the authors describe feasibility work on theory-based interventions that have the potential to address and inform unrealistic patient expectations.

Epidemiology of Shoulder Impairment in Older Adults

The prevalence of symptomatic rotator cuff tendinopathy in the general population ranges from 2.8 to 7.6% (Milgrom, Schaffler, Gilbert, & van Holsbeeck, 1995; Reilly, Macleod, Macfarlane, Windley, & Emery, 2006; Senbursa, Baltaci, & Atay, 2011; Silverstein et al., 2006), while asymptomatic rotator cuff tendinopathy is found in 10–20% (Milgrom et al., 1995). Rotator cuff tears are even more common, with reported prevalence ranging from 20% in the general population (Yamamoto et al., 2010), 30% in cadaveric studies (Reilly et al., 2006; Silverstein et al., 2006), to 40% in adults over the age of 50 (Worland, Lee, Orozco,

SozaRex, & Keenan, 2003). Self-reported prevalence of shoulder pain among all adults is estimated to be between 16 and 26% and is the third most common cause of musculoskeletal consultation in primary care (Luime et al., 2004; Urwin et al., 1998). Symptomatic pathology is increasingly common with age, occurring in up to 80% of adults over the age of 80 (Milgrom et al., 1995). The resultant pain and reduced range of motion (ROM) can significantly impair functional status and independence and negatively affect quality of life (Chambers, Shea, & Carey, 2011; Cigolle, Langa, Kabeto, Tian, & Blaum, 2007; Leveille, Zhang, McMullen, Kelly-Hayes, & Felson, 2005; Sha et al., 2005). In a prior survey of older adults, the authors found a 31% prevalence of chronic, severe shoulder pain (pain lasting more than 3 months in the past year) and 48% prevalence of impaired ROM. Of those with pain and reduced ROM, 36% had impaired basic activities of upper body function (e.g., grooming) (Burner et al., 2014).

Approach to Management of Shoulder Impairment

The first-line management for shoulder impairment and pain typically includes referral to physical therapy (PT) and home exercise programs (HEP) (Hawkins & Dunlop, 1995; Lombardi, Magri, Fleury, Da Silva, & Natour, 2008; Ludewig & Borstad, 2003). Conservative management may also include injections, pain medications, and other modalities (e.g., heat, topical analgesia). There is little evidence on which to base optimum management, with clinical guidelines being based on mostly inconclusive evidence and expert consensus (Pedowitz, Hunter, Tracy, & Brennan, 2012; Rechartdt et al., 2010; Rookmoneea et al., 2010). Once patients undergo surgical repair, they are also referred to PT and instructed in HEP to maximize postoperative recovery.

There has been an increase in surgical treatment of rotator cuff trauma by 141% over the 10-year period from 1996 to 2006. When adjusting for population growth, this translates into a 115% increase in rotator cuff repairs over this 10-year period (Colvin, Egorova, Harrison, Moskowitz, & Flatow, 2012). The best operative results are seen when repairing smaller tears that are not associated with significant muscle atrophy. It remains standard practice to refer patients to PT after surgical repair to optimize recovery of ROM.

Efficacy of Management

Conservative treatments such as PT and HEP have been shown to contribute to significant improvement in function in small trials, though it may take many months or even years to reach full impact (Chambers et al., 2011; Fritz, Childs, Wainner, & Flynn, 2012). In usual clinical practice, many older adults have limited contact with PT for their shoulder impairment, and it is unknown how long they continue to maintain HEP. The average number of PT visits per patient for shoulder problems has been reported at about 9 over a period of 4 weeks (Fritz et al., 2012). In a longitudinal assessment of shoulder pain in general practice, 41% of patients with reported shoulder pain showed persistent symptoms after 12 months following conservative therapy (Luime et al., 2004). For those experiencing improvement in pain and function with PT, the degree of improvement, and how long improvements are sustained, is generally not specified (Chambers et al., 2011; Fritz et al., 2012). Surgery may lead to significant improvement in function, but pain and limited mobility are unlikely to resolve completely, and re-tearing is a common long-term complication (Van der Windt et al., 1996).

Several factors play into the paucity of high-quality literature on conservative management for rotator cuff impairment. Quantifying improvement remains a challenge, as no standardized treatment protocols or gold standard outcome measurements exist. Methodologic issues are prevalent, and published studies frequently are uncontrolled, and lack consistency in the outcome scales used to measure pain, ROM, and function (Chambers et al., 2011; Rookmoneea et al., 2010). Despite these limitations, there does seem to be improvement in rotator cuff symptoms without a full-thickness tear with PT (Bennell et al., 2010; Lombardi et al., 2008) or HEP (Ludewig & Borstad, 2003) and no difference comparing PT with HEP (Walther, Werner, Stahlschmidt, Woelfel, & Gohlke, 2004; Werner, Walther, Ilg, Stahlschmidt, & Gohlke, 2002). Adjunct therapies such as scapular stabilization (Baskurt, Başkurt, Gelecek, & Özkan, 2011) and manual therapy (Bang & Deyle, 2000; Senbursa et al., 2011) may provide additive benefit to PT. The improvements in pain range from 2 to 4 cm on the Visual Analogue Scale maintained at 8–10 weeks' follow-up (20–40% decrease in symptoms) (Bang & Deyle, 2000; Lombardi et al., 2008; Ludewig & Borstad, 2003). Improvements in pain, ROM, and function in these studies are maintained at 6–22 weeks of follow-up, though the magnitude of gain is vague (Baskurt et al., 2011; Bennell et al., 2010; Lombardi et al., 2008; Silverstein et al., 2006; Walther et al., 2004; Werner et al., 2002). Less evidence exists for the efficacy of PT and HEP for full rotator cuff thickness tears, consisting mainly of uncontrolled case series. Supervised PT (Ainsworth, 2006; Baydar et al., 2009; Hawkins & Dunlop, 1995) and HEP improved patients' function, with improvement maintained at 3 months to 3.8 years of follow-up (Bennell et al., 2010; Lombardi et al., 2008).

Thus, patients with rotator cuff issues are typically referred for PT, both with and without surgery and other treatment modalities. With any approach to treatment, it often takes months to years for full recovery, and though extended PT and HEP (self-management) may help to attain optimum outcomes, full recovery may never be achieved. If patients have the expectation that treatment should result in complete resolution of all symptoms and restoration of full function, especially in a short amount of time, they have a disincentive to maintain longer-term treatment. Because patients often focus on somatic symptoms (e.g., pain) and restricted movement as indicators of ineffectiveness of treatment, this may be particularly likely to happen with shoulder dysfunction and result in less than optimal self-management.

The Commonsense Model of Self-Regulation (CSM) as a Framework for Examining Management of Shoulder Impairment

CSM is based upon the patient's representations or mental models of the illness (e.g., rotator cuff injury), the procedures for treatment (e.g., surgery, PT), an action plan for implementing treatment, and timeline and expectations for outcomes against which they assess efficacy of a treatment. "Normal" ROM and strength is the underlying "self-prototype," which may be conscious and or automatic (e.g., "normal shoulder function will feel like it did when I was 25"). The creation of representations begins with the awareness, either implicit (minimally conscious) or explicit, of deviations from the normal, "healthy" self: pains (e.g., shoulder pain) and functional changes (trouble reaching, carrying). The location and properties of an experienced deviation (sharp pain when overextending the shoulder), its time frames (rapidity of onset, duration), perceived causes (a fall), and response to self or medical treatment (heat eased the pain, rate of "improvement") are concrete perceptual factors that "define" the deviations as a shoulder injury. The impact of the disorder on thought, physical activity, social exchanges, and work defines its perceived and anticipated consequences.

The five content areas of an “active” mental model are *identity* (symptoms and location), *timelines*, *cause*, *control*, and *consequences*. These areas are generated by the match of the deviations from the self-prototype to a belief structure or prototype of a specific illness. An active mental representation suggests a range of actions for ameliorating the perceived discrepancies that will be experienced as coherent with the illness/injury representation and repeated as needed. If benefits are temporary (timeline violated) or absent (no alleviation), or insufficient to permit important life activities (consequences), the individual is highly likely to talk to family and/or friends and eventually seek medical care (Cameron, Leventhal, & Leventhal, 1993).

Differences in the “changeability” of these processes are unfortunately little understood. Active mental models, that is, representations of an illness, a treatment, and their coherence (the treatment fits the problem, i.e., how it works), are potentially highly flexible, while the underlying prototypes are less so. The two least flexible prototypes are (a) the usual/healthy self (Self as Anchor) and (b) expected outcomes of action, that is, the fundamental “Expectancy that Actions Produce Immediate Outcomes” (EAPIO). The self is a powerful, underlying anchor, as the self is experienced implicitly and sometimes explicitly, every moment of the 24 hr in a day, every day of every year of life. Deviations and outcomes of actions (benefits) are perceived and measured against this anchor, and it shapes life expectancies (Mora, DiBonaventura, Idler, Leventhal, & Leventhal, 2008). The EAPIO is an anchor for action; the acquisition begins at birth, as the infant perceives environmental changes in response to action (the mobile moves when legs are shaken; grasping the object seen brings it to the mouth). When action fails to produce an immediate outcome and mom does not come in response to the cry, the cry morphs into distress, rage, and loss of hope. Violations of EAPIO are experienced daily, for example, when the effort needed to lift an apparently light or heavy object is discrepant with experience (small object is extremely heavy, large object unexpectedly light).

The Self as Anchor and EAPIO are critical factors for examining the rehabilitation process. The contrast between Self as Anchor and current function (e.g., rotator cuff injury) creates the representation of injury and the target and/or “hope” for treatment outcome. The model suggests that the representation of injury (perceived difference between pre- and post-injured self) often exceeds reality. The “psycho-logic” for this is simple; the sense of normal Self as Anchor is overlearned, and the “how I am now” versus “how I am (normally) supposed to be” is magnified. The best-case scenario for therapy is when the results are perceptible and match or exceed expectations. The worst case is that pain and distress following PT (negative feedback) overwhelms actual improved motion. Such immediate effects need to be seen and understood in terms of an extended time frame or pathway to reach recovery; otherwise maintenance of treatment is unlikely.

Self-Prototypes: The Case in Shoulder Impairment

Individual psychosocial factors such as passive coping style, fear of movement, and general psychological distress may impact the chronicity of symptoms. Patients also have explicit expectations for diagnosis, recovery, and self-management of their condition. Such “common-sense” perceptions may not accurately reflect the underlying biophysical and medical factors at play. There is often a difference between objective clinical and radiological evidence of musculoskeletal disease activity and a patient’s experience of pain and functional ability (Hanusch, Goodchild, Finn, & Rangan, 2009). If actions are taken, and expected results are not seen within the anticipated time frame then further assessment and intervention may be justified

(e.g., “I’ve gone to PT for my shoulder for 6 weeks and it doesn’t feel like it’s back to normal, so PT is obviously not working”). For patients, the greatest impact of their disease lies in the effect it has on their ability to continue with “normal” life. All the physician authors have had such perceptions communicated by their patients during the course of routine practice (SG, EB, AR).

A series of studies were conducted to explore patients’ underlying self-prototypes and expectations for outcomes of PT in relationship to impaired shoulder function. In tandem, feasibility data was gathered on a system that will provide patients with clear, objective feedback on changes in ROM of an injured rotator cuff, allowing them to see that PT and HEP do improve shoulder function. We utilized a prototype software application and Kinect™ system to generate and record a stick-figure image, or skeletal avatar, that may be stored and replayed to visualize progress in ROM over time. The feedback is designed to overcome the lack of awareness of the very gradual, slow changes generated by PT and HEP and maintain motivation for long-term adherence.

Semi-structured interviews were conducted with patients entering PT for shoulder pain for the first time ($N = 15$) and patients planning to undergo surgical rotator cuff repair ($N = 10$) to gather preliminary data on underlying self-prototypes (i.e., self “baseline” against which patients were comparing outcomes of treatment), expectations for outcomes of treatment, and anticipated timelines to reach successful outcomes. Mean age of the PT group was 65.7 ± 5.9 years, with an average of 6.3 chronic medical conditions (range 0–16). The presurgical group had an average age of 47.8 ± 11.2 years and a mean of 5.7 chronic conditions (range 1–21). About 90% of the entire group of patients reported shoulder pain lasting at least 3 months out of the last year, a decreased ability to complete basic activities of daily living (e.g., grooming, dressing), and that their sleep had been adversely affected by shoulder pain.

Self-Prototypes, Expectations, and Timelines for Outcomes

All patients were asked to give their age when their shoulders last felt “normal,” that is, without any pain or symptoms of impairment. PT patients who had never gone for PT before (“PT-naïve”) all gave ages at least 15 years younger as their self-anchor (range 15–40 years). Those who had previously been to PT for other problems, expected their shoulder to function as it had 1–2 years previously. All save one of the surgical patients expected their postoperative shoulder to feel and function as it had at least 15 years earlier. The only patient who varied from this pattern, giving his current age as “baseline normal,” had undergone rotator cuff repair of the opposite shoulder several years before and so had already experienced the procedure and its outcome.

When questioned about expectations for outcomes of treatment, most patients expected to regain 90% or more of normal function, expected steady improvement, and reported they would stop HEP if they experienced any pain or discomfort. The one patient with previous shoulder surgery estimated regaining only 50% of prior normal function. This individual had *objectively* regained about 90% function in the previously injured shoulder, but reported regaining only 75% of normal function, and expected a worse outcome for the current impairment. This exemplifies the CSM construct of an “overlearned” self-prototype, with exaggeration of a current deficit and projection of this onto future outcomes. All the participants had unrealistic expectations for timeline of recovery, estimating it would take 6 months or less. Forty percent of the entire group estimated it would take less than 4 months to regain full function, and none thought it would take more than a year.

The results of these interviews highlight that patient expectations about recovery of shoulder ROM may be unrealistic, both in terms of timeline and extent of recovery. The CSM predicts that patients use underlying prototypes or “anchors” of the “healthy self” as a comparison for desired outcomes. These anchors/prototypes may date back many years and not reflect that person’s actual pre-injury “baseline.” Other factors, such as lack of education on the typical course of musculoskeletal ailments, lack of willingness to experience pain in the context of healing, or unrealistic attributions of one’s injury, may all moderate adherence to treatment choices and self-management. Because all patients reported expectations for steady improvement and progress without setback (even the “surgically experienced” patient), the experience of pain or other perceived setback may negatively impact a patient’s adherence to PT due to perceived lack of efficacy. In general, patients failed to recognize that there might be overlap of discomfort from both PT and an underlying injury, or variability in symptoms.

Technology for Addressing Unrealistic Timelines and Expectations for Outcomes

The described surveys of patients entering PT for treatment of shoulder impairment found that the majority have unrealistic expectations for recovery, have difficulty in accurately assessing improvement, and express frustration about lack of positive feedback during PT. Technology such as the Kinect imaging sensor has the potential to address these misperceptions to improve adherence and outcomes from PT.

The best-case scenario for therapy is when the results, such as improved movement and less pain, match or exceed expectations. The worst case is that negative feedback such as pain and distress during or following PT overwhelms the perception of improved motion. Such immediate effects need to be seen and understood in terms of an extended time frame or pathway to recovery; otherwise adherence to a long-term therapy regimen is unlikely. Our Kinect application has the capability to record and store successive images of a patient’s ROM representing progress over time; viewing stored images alongside current ROM allows the patient to see how his/her movement has improved from “worst function” to the present, providing concrete evidence of efficacy that overrides momentary doubts about benefit and negative feedback (e.g., pain during movement). Such images also provide a dynamic record of ROM, instead of clinician recall or static measurements recorded during prior PT sessions.

The authors have designed a software application (JU) that takes the camera image and translates it into a skeleton or “stick avatar” of the patient that is displayed on a screen in real time. As the patient proceeds through an ROM exercise, the avatar shows this range on the screen along with fiducial limb ROM measurements for each exercise. The system is also capable of simultaneously displaying a prerecorded avatar, that is, prior patient images or exemplar patient. The patient (and clinician) thus receive immediate feedback on patient ROM because the application can record and store successive patient images for later review, allowing them to observe progress over time. Feasibility testing was completed in a proof-of-concept trial on the surgical patients at “baseline” (preoperatively) and at 6 weeks and 3 months’ postoperatively. At each visit shoulder ROM was measured by a clinician visually and with the Kinect. Motions assessed for all participants included forward elevation, external rotation with arm abducted and arm at side, and abduction.

The angle measurements as recorded by the Kinect showed a strong correlation to the clinician measurements ($R = 0.966$, 95% confidence interval 0.940–0.981), with no significant

difference between the two ways of measurement ($t = -0.23$, $p = 0.81$). As anticipated, ROM was impaired at baseline on the side of injury, was even more restricted at 6 weeks' postoperatively (about the time when patients could start moving their shoulder), and improved significantly at 3 months.

These data demonstrate the feasibility and validity of the Kinect system to record angle measurements that are in line with, and not statistically different from, that measured by a clinician. The observed ROM over time showed the pattern that would be expected over the course of surgical management of a rotator cuff injury: decreased ROM at baseline, followed by an even greater decrease after 6 weeks of immobilization following surgery, and improvements over baseline by 3 months. The benefits of this system include the ability of clinician and patient to track their improvements over time with concrete data and measurements to provide the patient with positive feedback to override temporary doubts and lapses in motivation. The system is currently being tested in a randomized trial. All participants are imaged prospectively over the course of PT, but half view their progressive images at each visit. This application will eventually be deployed in the home setting by migrating the application to a tablet or smartphone and will include the ability to send ROM data directly to the clinician for monitoring. This approach could limit unnecessary follow-up visits and cut healthcare costs while allowing the patient to continue their PT at home with proper visual feedback and clinician updates to instill motivation over time. Similar ROM images and measures are being collected on normal, healthy subjects to get a preliminary sense of interindividual variability. This data may be used to develop "ideal ROM" avatars that can be displayed with individual patient images as target goals for recovery.

Conclusion

Moving forward, it is hoped that providing patients with accurate feedback on progress over time (such as that provided by the Kinect imagery) will override subjective perceptions of lack of progress and thus improve long-term adherence to treatment. Another implication of this research is that clinicians may improve patient adherence by assessing their expectations for recovery as part of their initial evaluation and directly addressing unrealistic expectations. This study did not evaluate whether changing a patient's expectations leads to improved outcomes, but it does suggest that patients with shoulder pain are underinformed and unrealistic about the usual course of their disease.

Author Biographies

Ann Rosenthal, MD, is the Will and Cava Ross Professor of Medicine and Chief of Rheumatology at the Medical College of Wisconsin. Her major research interests are in calcium pyrophosphate deposition disease and other metabolic and age-related musculoskeletal syndrome.

Dara Mickschl, PA-C, is a physician assistant at the Medical College of Wisconsin. She focuses on orthopedic upper extremity of all ages. She has athletic training background. Her research topics include surgical interventions for upper extremity orthopedic conditions, demographic and health factors affecting outcomes, investigating techniques for accuracy and efficacy of steroid injections, and kinematic assessment during postsurgical rehabilitation.

Edith Burns, MD, is a professor of medicine at the Medical College of Wisconsin. Her primary research interests are self-management of chronic illness (e.g., diabetes, CHF, joint impairment), risks for hospital readmission after elective surgery, multimorbidity in geriatric populations, and assessing patient illness perceptions, expectations, and timelines for outcomes.

Elizabeth Neumann, MD, graduated from Medical College of Wisconsin in 2017. Her work with Dr. Burns, on expectations for outcomes of shoulder impairment, was supported by an NIA, T35 grant on aging and injury prevention. She is currently a first-year obstetrics–gynecology resident at the University of Florida, Sacred Heart System.

Jay Urbain, PhD, is a professor in the Electrical Engineering and Computer Science Department at the Milwaukee School of Engineering and codirector of Biomedical Informatics for CTSI of SE Wisconsin at the MCW. His research interests include machine learning, NLP, information retrieval and applications in biomedical informatics, intelligence, and real-time sensors.

Sergey Tarima, PhD, is an associate professor of biostatistics. He was appointed to the faculty at the Medical College of Wisconsin at completion of his doctorate in 2005. His research interests include use of additional information in statistical estimation and sample size estimation and reestimation in the presence of nuisance parameters.

Steven Grindel, MD, is professor of orthopedic surgery at the Medical College of Wisconsin. His practice and research interests focus on arthroscopic and reconstructive surgery of the shoulder, elbow, wrist, and hand, with extensive expertise in industrial problems affecting the upper extremity.

References

- Ainsworth, R. (2006). Physiotherapy rehabilitation in patients with massive, irreparable rotator cuff tears. *Musculoskeletal Care*, 4(3), 140–151.
- Bang, M. D., & Deyle, G. D. (2000). Comparison of supervised exercise with and without manual physical therapy for patients with shoulder impingement syndrome. *Journal of Orthopaedic and Sports Physical Therapy*, 30(3), 126–137.
- Baskurt, Z., Başkurt, F., Gelecek, N., & Özkan, M. H. (2011). The effectiveness of scapular stabilization exercise in the patients with subacromial impingement syndrome. *Journal of Back and Musculoskeletal Rehabilitation*, 24(3), 173–179.
- Baydar, M., Akalin, E., El, O., Gulbahar, S., Bircan, C., Akgul, O., ... Kizil, R. (2009). The efficacy of conservative treatment in patients with full-thickness rotator cuff tears. *Rheumatology International*, 29(6), 623–628.
- Bennell, K., Wee, E., Coburn, S., Green, S., Harris, A., Staples, M., ... Buchbinder, R. (2010). Efficacy of standardized manual therapy and home exercise programme for chronic rotator cuff disease: Randomized placebo controlled trial. *British Medical Journal*, 340, c2756.
- Burner, T., Abbott, D., Huber, K., Stout, M., Fleming, R., Wessel, B., ... Burns, E. (2014). Shoulder symptoms and function in geriatric patients. *Journal of Geriatric Physical Therapy*, 37(4), 154–158.
- Cameron, L., Leventhal, E. A., & Leventhal, H. (1993). Symptom representations and affect as determinants of care seeking in a community-dwelling, adult sample population. *Health Psychology*, 12(3), 171–179.
- Chambers, H. G., Shea, K. G., & Carey, J. L. (2011). American academy of orthopaedic surgeons clinical practice guideline: Diagnosis and treatment of osteochondritis dissecans. *Journal of the American Academy of Orthopaedic Surgeons*, 19(5), 307–309.
- Cigolle, C. T., Langa, K. M., Kabeto, M. U., Tian, Z., & Blaum, C. S. (2007). Geriatric conditions and disability: The Health and Retirement Study. *Annals of Internal Medicine*, 147(3), 156–164.

- Colvin, A. C., Egorova, N., Harrison, A. K., Moskowit, A., & Flatow, E. L. (2012). National trends in rotator cuff repair. *Journal of Bone and Joint Surgery, American Journal of Orthopedic Surgery*, 94(3), 227–233.
- Fritz, J. M., Childs, J. D., Wainner, R. S., & Flynn, T. W. (2012). Primary care referral of patients with low back pain to physical therapy: Impact on future health care utilization and costs. *Spine*, 37(25), 2114–2121.
- Hanusch, B. C., Goodchild, L., Finn, P., & Rangan, A. (2009). Large and massive tears of the rotator cuff; functional outcome and integrity of the repair after a mini-open procedure. *Journal of Bone and Joint Surgery*, 91-B, 201–205.
- Hawkins, R. H., & Dunlop, R. (1995). Nonoperative treatment of rotator cuff tears. *Clinical Orthopaedics and Related Research*, 321, 178–188.
- Leveille, S. G., Zhang, Y., McMullen, W., Kelly-Hayes, M., & Felson, D. T. (2005). Sex differences in musculoskeletal pain in older adults. *Pain*, 116(3), 332–338.
- Lombardi, I., Jr., Magri, A. G., Fleury, A. M., Da Silva, A. C., & Natour, J. (2008). Progressive resistance training in patients with shoulder impingement syndrome: A randomized controlled trial. *Arthritis and Rheumatism*, 59(5), 615–622.
- Ludewig, P. M., & Borstad, J. D. (2003). Effects of a home exercise programme on shoulder pain and functional status in construction workers. *Occupational and Environmental Medicine*, 60, 841–849.
- Luime, J., Koes, B. W., Hendriksen, I. J., Burdorf, A., Verhagen, A. P., Miedema, H. S., & Verhaar, J. A. (2004). Prevalence and incidence of shoulder pain in the general population; a systematic review. *Scandinavian Journal of Rheumatology*, 33, 73–81.
- Milgrom, C., Schaffler, M., Gilbert, S., & van Holsbeeck, M. (1995). Rotator-cuff changes in asymptomatic adults. The effect of age, hand dominance and gender. *Journal of Bone and Joint Surgery. British Volume*, 77(2), 296–298.
- Mora, P. A., DiBonaventura, M. D., Idler, E., Leventhal, E. A., & Leventhal, H. (2008). Psychological factors influencing self-assessments of health: Toward an understanding of the mechanisms underlying how people rate their own health. *Annals of Behavioral Medicine*, 36(3), 292–303.
- Pedowitz, R. A., Hunter, S. J., Tracy, D. M., & Brennan, G. P. (2012). Utilization and clinical outcomes of outpatient physical therapy for medicare beneficiaries with musculoskeletal conditions. *Physical Therapy*, 91(3), 330–345.
- Raymond, L., Sluka, P., McGowan, R., & American Academy of Orthopaedic Surgeons. (2011). Optimizing the management of rotator cuff problems. *Journal of the American Academy of Orthopaedic Surgery*, 19(6), 368–379.
- Rechardt, M., Shiri, R., Karppinen, J., Jula, A., Heliövaara, M., & Viikari-Juntura, E. (2010). Lifestyle and metabolic factors in relation to shoulder pain and rotator cuff tendinitis: A population based study. *BMC Musculoskeletal Disorders*, 20(11), 165.
- Reilly, P., Macleod, I., Macfarlane, R., Windley, J., & Emery, R. J. (2006). Dead men and radiologists don't lie: A review of cadaveric and radiological studies of rotator cuff tear prevalence. *Annals of the Royal College of Surgeons of England*, 88(2), 116–121.
- Rookmonee, M., Dennis, L., Brealey, S., Rangan, A., White, B., McDaid, C., & Harden, M. (2010). The effectiveness of interventions in the management of patients with primary frozen shoulder. *Journal of Bone and Joint Surgery. British Volume*, 92(9), 1267–1272.
- Senbursa, G., Baltaci, G., & Atay, O. A. (2011). The effectiveness of manual therapy in supraspinatus tendinopathy. *Acta Orthopaedica et Traumatologica Turcica*, 45(3), 162–167.
- Sha, M. C., Callahan, C. M., Counsell, S. R., Westmoreland, G. R., Stump, T. E., & Kroenke, K. (2005). Physical symptoms as a predictor of health care use and mortality among older adults. *American Journal of Medicine*, 118(3), 301–306.
- Silverstein, B. A., Viikari-Juntura, E., Fan, Z. J., Bonauto, D. K., Bao, S., & Smith, C. (2006). Natural course of nontraumatic rotator cuff tendinitis and shoulder symptoms in a working population. *Scandinavian Journal of Work, Environment & Health*, 32(2), 99–108.
- Urwin, M., Symmons, D., Allison, T., Brammah, T., Busby, H., Roxby, M., ... Williams, G. (1998). Estimating the burden of musculoskeletal disorders in the community: The comparative prevalence

- of symptoms at different anatomical sites, and the relation to social deprivation. *Annals of the Rheumatic Diseases*, 57, 649–655.
- Van der Windt, D. A., Koes, B. W., Boeke, A. J., Devillé, W., De Jong, B. A., & Bouter, L. M. (1996). Shoulder disorders in general practice: Prognostic indicators of outcome. *British Journal of General Practice*, 46, 519–523.
- Walther, M., Werner, A., Stahlschmidt, T., Woelfel, R., & Gohlke, F. (2004). The subacromial impingement syndrome of the shoulder treated by conventional physiotherapy, self-training, and a shoulder brace: Results of a prospective, randomized study. *Journal of Shoulder and Elbow Surgery*, 13(4), 417–423.
- Werner, A., Walther, M., Ilg, A., Stahlschmidt, T., & Gohlke, F. (2002). Self-training versus conventional physiotherapy in subacromial impingement syndrome. *Zeitschrift Fur Orthopadie Und Ihre Grenzgebiete.*, 140(4), 375–380.
- Worland, R. L., Lee, D., Orozco, C. G., SozaRex, F., & Keenan, J. (2003). Correlation of age, acromial morphology, and rotator cuff tear pathology diagnosed by ultrasound in asymptomatic patients. *Journal of the Southern Orthopaedic Association*, 12(1), 23–26.
- Yamamoto, A., Takagishi, K., Osawa, T., Yanagawa, T., Nakajima, D., Shitara, H., & Kobayashi, T. (2010). Prevalence and risk factors of a rotator cuff tear in the general population. *Journal of Shoulder and Elbow Surgery*, 19(1), 116–120.

Suggested Reading

- Greenberg, D. L. (2015). Evaluation and treatment of shoulder pain. *Medical Clinics of North America*, 98, 487–504.
- Leventhal, H., Phillips, L. A., & Burns, E. A. (2016). The Common-sense model of self-regulation (CSM): A dynamic framework for understanding illness self-management. *Journal of Behavioral Medicine*, 39(6), 935–946.
- Webster, D., & Celik, O. (2014). Systematic review of Kinect applications in elderly care and stroke rehabilitation. *Journal of Neuroengineering and Rehabilitation*, 11(108), 1–24.

Trends in Clinical Health Psychology and Behavioral Medicine

C. Steven Richards¹ and Lee M. Cohen²

¹*Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA*

²*Department of Psychology, College of Liberal Arts, The University of Mississippi, Oxford, MS, USA*

Clinical health psychology and behavioral medicine are interesting and important specialty areas where various healthcare fields intersect. In this entry, we briefly discuss seven trends, that we believe, are shaping these fields, including (a) methodological sophistication, (b) chronic diseases, (c) diversity and inclusiveness, (d) biological issues, (e) translation of health psychology knowledge, (f) chronic psychopathology that is comorbid with health problems and diseases, and (g) social justice. We conclude this entry with a brief discussion of recommendations regarding theory, research, and practice to consider for the future.

Methodological Sophistication

After a review of the research in clinical health psychology and behavioral medicine, it is clear that the intricacy of the studies being published has progressed significantly in multiple ways. For instance, many studies include large and diverse samples of participants. Sampling effectively is one of the cornerstones of sound research and certainly in any area involving human participants. An example of a recent study that includes a large and diverse sample was conducted by Polenick, Renn, and Birditt (2018), where they studied the dyadic effects of depression on medical morbidity among 992 middle-aged and older-adult couples. One of the major findings of this study was that depressive symptoms in this population have long-term associations with medical morbidity, such as chronic health conditions.

Another indication of the current methodological sophistication in this area is an increased use of multi-variable, multi-trait, and multi-method measures, with sound psychometric properties. Such methodology permits the collection of a more thorough picture of the

cognitive, emotional, behavioral, biological, and other health outcome variables relevant to the research questions being studied.

An interesting example is a study that explored the longitudinal pathways to alcohol use and abuse among 426 adolescents who had genetic risk factors for addiction, by Trucco, Villafuerte, Hussong, Burmeister, and Zucker (2018). These investigators predicted that the pathways under investigation would be moderated by depression and depressive relapse during the last wave of the study, which included adolescents aged 15–17 years. More specifically, it was expected that depression would be related to more substance use and would essentially facilitate the expression of the genetic risk factors. Trucco et al. (2018) also predicted that ineffective coping methods for stress would be a moderator, related to greater substance use. The investigators' predictions were supported: ongoing depression (or depressive relapse) over time, along with ineffective coping methods, appears to be a pathway that allows genetic risk factors to influence substance use and abuse. This study represents multiple research trends toward excellence in clinical health psychology and behavioral medicine, such as including a large sample ($N = 426$, plus a comparison sample), which was followed longitudinally over many years (ages 3–17, in 5 waves), with diverse measures (multi-method, multi-trait, and multi-source), and attention to both biological and psychological variables. Trucco et al. (2018) included measures of demographics, family history, temperament, coping, depression, aggression, substance use, and biological correlates such as gender, race, and genotyping. Data were obtained from multiple sources for the psychosocial variables. Moreover, this study provides a rich evidence base for future efforts at health promotion and intervention.

In summary, methodological sophistication is one of the important trends in research on clinical health psychology and behavioral medicine. Indeed, recent editorials by editors of some of the major journals in the field have boasted about this trend and called for it to continue (e.g., for discussions of methodological issues, see Appelbaum et al., 2018; Freedland, 2017; Lilienfeld, 2017).

Chronic Diseases

Another trend in clinical health psychology and behavioral medicine is an emphasis on psychosocial variables in the context of chronic diseases. There is tremendous interest and concern regarding highly prevalent chronic diseases such as heart disease, cancer, stroke, diabetes, arthritis, flu and other pandemic diseases, and so forth. These diseases are often listed in the top 5 or 10 causes of death from disease among adults across the industrialized world. Furthermore, psychosocial variables have been established as having important mediating, moderating, and health-promoting effects for the course and outcome of such diseases (e.g., for a readable review, see Taylor, 2018).

An example of an excellent study illustrating this trend is a recent study by Manne, Siegel, Heckman, and Kashy (2016). This study included 302 female patients with early-stage breast cancer, along with their spouses. For the most distressed patients, a couple-focused and supportive version of group therapy appeared to be the more-effective intervention for coping with psychosocial variables such as depression and anxiety. In contrast, for patients who were less distressed, a structured and skills-based version of group therapy appeared to be the most effective for reducing depression and anxiety, and in an associated manner, also improving health-promoting behaviors such as stronger adherence to medical regimens. This study successfully addressed some of the common research concerns among the psycho-oncology

research community, such as including a relatively large sample, a longitudinal and randomized controlled design, and use of a broad array of measures.

There are also numerous excellent examples of research examining combined psychosocial and medical interventions for post-heart-attack patients that are represented by the ENRICHD and CREATE randomized controlled trials (Berkman et al., 2003; Lespérance et al., 2007). These large trials (with beginning sample sizes of 2,481 and 284, respectively) randomized participants to combinations of SSRI antidepressant medications and either cognitive behavior therapy (ENRICHD) or interpersonal psychotherapy (CREATE) or usual care with brief supportive counseling from clinic and healthcare staff.

In addition to the standard goals of better heart health and medical improvement after a heart event, the treatment goals included improvement in depression and social support (Berkman et al., 2003; Lespérance et al., 2007). Numerous correlational studies have indicated clear relationships between these psychosocial variables and long-term heart health in cardiac disease patients. Although a comprehensive summary of the results from the ENRICHD and CREATE trials is beyond the scope of this brief entry, a major finding was that the medication plus psychosocial intervention conditions indicated improvement in depression and social support over time. However, these psychosocial improvements were not associated with significantly fewer heart events or cardiac fatalities over time. Therefore, while the improvements observed during follow-up visits on the psychosocial measures was encouraging, there was disappointment that improvements in depression and social support over time did not translate into improvement in physical cardiac health and avoidance of future heart attacks. Nonetheless, these heart disease studies stand as excellent examples of sophisticated research on psychosocial processes associated with disease.

In summary, there have been numerous excellent studies regarding clinical health psychology, behavioral medicine, and chronic disease. Chronic diseases are major killers, however, and we need to learn more. Fortunately, there appears to be movement in this direction, although the large randomized controlled trials like those noted above are expensive and thus vulnerable to the funding patterns of major national granting agencies and foundations/societies focused on health, such as the National Institutes of Health (United States), the National Health Service (United Kingdom), the American Heart Association, and the American Cancer Society.

Diversity and Inclusiveness

We live in a diverse world and the benefits of inclusiveness are obvious. Therefore, many areas within clinical health psychology and behavioral medicine, as with other fields that engage in theory, research, and practice relevant to human beings, have witnessed a large increase in studies and interventions that focus on diversity and inclusiveness. From our perspective, this is one of the most important trends in clinical health psychology and behavioral medicine (e.g., see the editorial by Davila, 2017; and the special section overview, on sexual and gender minority health, by Davila & Safren, 2017).

An example of a determined effort to recruit a diverse sample is a study examining physical activity and depressive symptoms after treatment for breast cancer (Brunet, O'Loughlin, Gunnell, & Sabiston, 2018). The early research in this area was often limited to samples of entirely Caucasian women, within a small age range, who were relatively well educated and mostly married or living with a partner. In contrast, this large study of 201 women with breast cancer reflected efforts to recruit a more diverse sample, including a wider range of age, race, civil status, and education. This study also evidenced considerable diversity regarding medical

variables such as stage of cancer, treatments received, and time since treatment completion. The investigators acknowledge, of course, that even more diverse samples will be helpful for future research. One of the major findings from this investigation is that more physical exercise was associated with less frequent depressive symptoms among participants after treatment for breast cancer; however, not all of the predicted relationships between depression and exercise were maintained longitudinally.

Another recent example of a study that included ambitious efforts to recruit a large and diverse sample is a study that examined the associations between pharmacotherapy for diabetes and immigrant status in the United States; this study was conducted by Hsueh et al. (2018). While a complete discussion regarding the many variables in this study that examined diversity is beyond the scope of this entry, if we focus solely on the race, ethnicity, and education variables used, there were numerous participants across several racial and ethnicity groups, including 28% non-Hispanic Black, 18% Mexican American, 8% other Hispanic, 6% multiracial, and 40% non-Hispanic White. Educational level completed was also quite diverse, ranging from ninth grade or lower to college graduate or above. One of the major findings included a clinically significant result that being foreign born was associated with significantly reduced odds of receiving insulin treatment (Hsueh et al., 2018). There were also some differential patterns of associations with treatment offerings and different racial/ethnic group membership. These results suggest that perhaps integrating diversity information about a patient's immigration status and racial/ethnic identity would allow for a more culturally sensitive care of diabetes.

In summary, we live in a diverse world, and additional research like the studies noted above is needed. It is essential that future studies make it a priority to recruit and assess diverse participants and consider relevant diversity variables.

Biological Issues

Traditional research on chronic health problems and diseases has characteristically paid considerable attention to biological factors. More recently, social scientists are paying increasingly more attention to such variables. Specifically, the fields of clinical health psychology and behavioral medicine appear to be moving toward a stronger biological focus (e.g., see editorials by Freedland, 2017; Lilienfeld, 2017). Furthermore, this increased focus on a biological emphasis is facilitated and enhanced by recent technology, such as MRI, PET, and CT scans. The sophistication and options available for biological assays of blood, urine, hormones, neurotransmitters, and so forth have expanded and become more accessible. Moreover, the ability to evaluate, store, and reexamine biological samples has grown tremendously (e.g., see discussions in Taylor, 2018).

An interesting example of a health psychology study that incorporates biological variables is the Trucco et al. study (2018), mentioned earlier in this entry. Trucco et al. (2018) included measures of genotyping, focusing on the polymorphisms from four genes, which were predicted to be relevant to the substance use variables being studied. A major finding was that depression and ineffective coping methods appear to be a pathway that allows genetic risk factors to influence substance use and abuse.

Another example of biological measures being utilized in recent clinical health psychology research is an investigation by Bakhshaie et al. (2018). The authors used a carbon monoxide analysis of breath samples as a method to verify the participants' smoking status. Similar biochemical verification methods have been used in numerous smoking cessation research

studies, along with other studies on nicotine and tobacco use. One of the investigators' primary findings was that teaching the participants strategies to more effectively manage symptoms of anxiety and depression appeared to be associated with fewer withdrawal symptoms, and thus better outcomes, in a smoking cessation study (Bakhshaie et al., 2018).

In summary, the inclusion of biological variables in clinical health psychology and behavioral medicine research is becoming more common, more sophisticated, and more central to the questions being asked.

Translation of Health Psychology Knowledge

Research studies conducted in laboratories, with carefully controlled variables, under almost perfect conditions are important. However, it is also critical that findings be applicable in real-world situations. These real-world situations often involve imperfect conditions, limited resources, overworked healthcare providers, underserved patients, and numerous other challenges. Practical and cost-effective translations have found encouraging effectiveness in real-world settings, in part, by using mobile and online technologies, public health education campaigns, social service agencies, charitable organizations, and religious organizations (e.g., see the journal editorial in *Translational Behavioral Medicine* by Miller-Halegoua, Bowen, Diefenbach, & Tercyak, 2016).

O'Hara and his colleagues have conducted several randomized controlled trials and more naturalistic, low-cost intervention trials in healthcare clinics with depressed postpartum women. Their findings indicate that interpersonal psychotherapy interventions in the acute phase of depression treatment, and/or during maintenance treatment, are helpful for depressive relapse prevention among women who often had numerous health challenges and who were also caring for young children (e.g., Nylén et al., 2010; O'Hara, Stuart, Gorman, & Wenzel, 2000; Serge, Brock, & O'Hara, 2015). Moreover, these studies showed a transition themselves, from efficacy-like studies as seen in O'Hara et al. (2000) to more translational studies as seen in Serge et al. (2015). Thus, even in economically challenged and underserved populations being treated in real-world healthcare settings with limited resources, interpersonal psychotherapy has potential as both an acute and maintenance treatment for depression and as a treatment that can be translated into real-world settings (e.g., see Serge et al., 2015 for the most translational study in this series of investigations).

Another excellent translational study explored how to improve adherence to cognitive behavior therapy for insomnia (CBT-I; Dolsen et al., 2017). In this randomized trial for 188 adults with persistent insomnia, the investigators found strong support for the hypothesis that effective sleep the night before and the night after a treatment session will improve patients' adherence to many aspects of the treatment regimen. Additionally, it was found that CBT-I interventions usually improve the patient's sleep before and after a treatment session. Such sleep improvement, in turn, will improve treatment adherence, based on their findings. Given that adherence to treatment is a huge challenge for most psychosocial and medical interventions, this study is an example of a methodologically sophisticated study that may be generalizable to many healthcare environments, help clinicians and patients to improve treatment adherence, and make a significant difference in public health.

In summary, research in the fields of clinical health psychology and behavioral medicine is continuing to improve upon translating findings into practical methods that have clear applications in the real world.

Chronic Psychopathology That Is Comorbid With Health Problems and Diseases

Patients suffering from comorbid chronic psychopathology and physical health concerns commonly present in healthcare settings. As such, gaining a better understanding of the relationships between physical and mental health is essential if treatments are to advance. One example of a study that has examined this complicated relationship was conducted by Stice, Gau, Rohde, and Shaw (2017). This research team (as well as their colleagues in numerous related studies) investigated the risk factors that predict the various *DSM-5* eating disorders (American Psychiatric Association, 2013). Stice et al. (2017) evaluated a rich array of variables that might predict the future onset of each *DSM-5* eating disorder. These disorders included anorexia nervosa, bulimia nervosa, binge eating disorder, and purging disorder. With a sample of 1,272 young women (*M* age of 18.5 years), the investigators conducted diagnostic interviews every year for 3 years of follow-up assessment, following a series of their own prevention trials. In addition, they collected a vast amount of self-report and weight, height, and health data. It was found that the strongest predictors leading to the development of eating disorders were related to negative affect, such as depressive symptoms, and interpersonal difficulties. As with much of the research literature on eating disorders, pursuit of a “thin ideal” and the associated body dissatisfaction that often accompanies this goal was strongly correlated with extreme dieting, unhealthy weight control behaviors, and the negative affect and interpersonal difficulties mentioned above. Therefore, the investigators note that incorporating interventions for depression and interpersonal discord into prevention efforts for eating disorders may be an effective strategy.

In summary, some types of chronic psychopathology, such as depression, are frequently observed with a large number of physical health concerns and diseases. The best research and clinical work accounts and plans for such comorbidity.

Social Justice

Given that health concerns (both physical and mental) can be influenced greatly by the distribution of wealth and opportunities for personal activity, it is our opinion that issues related to social justice should be of concern to everyone. Research and practice in the fields of clinical health psychology and behavioral medicine, therefore, should reflect these concerns. Fortunately, much of the recent work in these fields has begun to consider such concerns, and the emphasis is increasing enough to justify calling it a “trend.”

A good example is the work of Yang, Chen, and Park (2016), who examined perceived housing discrimination and self-reported health. With a sample of 9,842 adults in 830 neighborhoods across Philadelphia, PA, the investigators were interested in whether certain neighborhood features matter in terms of overall health. Specifically, associations of increased perceived housing discrimination, and more negative self-reported health, were strengthened in neighborhoods with relatively high housing values; however these associations were weakened in neighborhoods with a broader range of housing values and diversity (i.e., income, parenthood, and ethnic minority status). Another interesting finding of this large study was that the statistical models for illustrating a negative relationship between perceived housing discrimination and self-reported health generally underestimate the relationship, unless the models also incorporate neighborhood features such as economic level, family diversity, and ethnic minority status. This large, sophisticated study by Yang et al. (2016) is an excellent example of research on the intersection of clinical health psychology, social justice, and multicultural issues.

In summary, there has been important research on social justice, health psychology, behavioral medicine, and a broad range of multicultural and diversity issues. This research area is growing rapidly, and it should help guide our future efforts in assessment, intervention, and follow-up.

Discussion

The trends discussed in this entry (while not all inclusive) appear to be among the most prominent, consistent, and frequently cited developments in the fields of clinical health psychology and behavioral medicine. While staying close to the data and what is recently published, we offer a few cautious recommendations about these trends.

The importance of the trend of sophisticated methodology cannot be exaggerated. The history of science suggests that it is only as good as its methods. Therefore, we applaud improvements in methodology such as larger and more diverse samples, multi-method and multi-source measures, and longitudinal data collection. This kind of ambitious, large, and sophisticated research, along with translation to the real world, requires skill, resources, and determination. We hope that the research and practice infrastructures will continue to support this important work.

The trend of an emphasis on chronic diseases is expected and necessary, especially considering the significant impact behavioral and emotional factors play on overall health status. Some very important research and translational efforts have been accomplished in this area, and we hope that this important work continues.

Our world is diverse. Therefore, our research, clinical work, and translational efforts should reflect diversity, broadly defined. In our opinion, the fields of clinical health psychology and behavioral medicine have made a considerable amount of progress on this issue. However, we would like these efforts to continue and expand.

Biology has been strongly associated with how we behave (including our personality traits). Moreover, the explosion in biological technology allows for more sophisticated assessments of biological variables. As such, over the past couple of decades, it has become increasingly more common in clinical health psychology and behavioral medicine research to incorporate more biological variables and to include more biological measures in clinical and translational efforts. This trend toward a stronger biological focus can be expensive, and the technical demands can be daunting, but we believe the benefits make it worthwhile. We predict that the incorporation of biological variables in health psychology research will become expected in the foreseeable future, moving the emphasis on biology from a trend to a centerpiece of the field.

The translation of health psychology knowledge into the real world of healthcare settings is an important trend in clinical health psychology and behavioral medicine. After all, if the results we read about in academic journals do not apply to actual clinical populations in typical healthcare settings, then the utility of these findings holds less meaning. Clearly, researchers in these fields are interested in improving the health status of as many people as possible, not just the “ideal candidate.”

Chronic psychopathology is often comorbid with health problems and diseases. Depression is a good example, as it is associated with almost every health problem and disease on record. Depressive comorbidity is not helpful when it comes to our health, as, for example, the association of long-term depression with heart disease has many negative implications, including increased disability, disrupted close relationships, poorer adherence to medical and psychological regimens, increased problems at work and at home, further heart events, and increased rates of death from heart disease. Therefore, we can no longer afford to consider psychological and medical issues in isolation.

Last but not least, we discussed the importance of considering variables related to social justice. While progress has been made regarding the inclusion of variables relevant to social justice concerns as part of more traditional clinical health psychology and behavioral medicine studies, more emphasis regarding social justice variables clearly needs to be done. This work is exciting and important, and we need it if we are going to make progress for everyone.

Author Biographies

C. Steven Richards, PhD, is a professor of psychological sciences at Texas Tech University. His research interests include depression, clinical health psychology and behavioral medicine, comorbidity of health problems and psychopathology, self-control, and relapse prevention for depression. He has held 15 administrative positions during his faculty appointments at Texas Tech University, Syracuse University, and the University of Missouri–Columbia. Dr. Richards earned his PhD in clinical psychology at the State University of New York at Stony Brook (now Stony Brook University).

Lee M. Cohen, PhD, is dean of the College of Liberal Arts and professor in the Department of Psychology at the University of Mississippi. Dr. Cohen came to the University of Mississippi from Texas Tech University, where he served in a number of administrative roles including the director of the nationally accredited doctoral program in clinical psychology and the chair of the Department of Psychological Sciences. As a faculty member, he received several university-wide awards for his teaching and academic achievement. As a researcher, he received more than \$1.5 million from funding agencies, including the US Department of Health and Human Services, the National Science Foundation, and the National Institutes of Health/National Institute on Drug Abuse. His research program examines the behavioral and physiological mechanisms that contribute to nicotine use, and he worked to develop optimal smoking cessation treatments. Dr. Cohen is a Fellow of the American Psychological Association and the Society of Behavioral Medicine. He received his PhD in clinical psychology from Oklahoma State University.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed., DSM-5). Washington, DC: Author.
- Appelbaum, M., Cooper, H., Kline, R. B., Mayo-Wilson, E., Nezu, A. M., & Rao, S. M. (2018). Journal article reporting standards for quantitative research in psychology: The APA Publications and Communications Board Task Force Report. *American Psychologist*, 73, 3–25.
- Bakhshaie, J., Kulesz, P. A., Garey, L., Langdon, K. J., Businelle, M. S., Leventhal, A. M., ... Zvolensky, M. J. (2018). A prospective investigation of the synergistic effect of change in anxiety sensitivity and dysphoria on tobacco withdrawal. *Journal of Consulting and Clinical Psychology*, 86, 69–80.
- Berkman, L. F., Blumenthal, J., Burg, M., Carney, R. M., Catellier, D., Cowan, M. J., ... Schneiderman, N. (2003). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: The Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) randomized trial. *JAMA*, 289, 3106–3116.
- Brunet, J., O'Loughlin, J. L., Gunnell, K. E., & Sabiston, C. M. (2018). Physical activity and depressive symptoms after breast cancer: Cross-sectional and longitudinal relationships. *Health Psychology*, 37, 14–23.
- Davila, J. (2017). Editorial. *Journal of Consulting and Clinical Psychology*, 85, 1–4.

- Davila, J., & Safren, S. A. (Eds.) (2017). Introduction to the special section on sexual and gender minority health. *Journal of Consulting and Clinical Psychology*, 85, 1109–1110.
- Dolsen, M. R., Soehner, A. M., Morin, C. M., Belanger, L., Walker, M., & Harvey, A. G. (2017). Sleep the night before and after a treatment session: A critical ingredient for treatment adherence? *Journal of Consulting and Clinical Psychology*, 85, 647–652.
- Freedland, K. E. (2017). Editorial: A new era for *Health Psychology*. *Health Psychology*, 36, 1–4.
- Hsueh, L., Vraney, E. A., Patel, J. S., Hollingshead, N. A., Hirsh, A. T., de Groot, M., & Stewart, J. C. (2018). Associations between immigrant status and pharmacological treatments for diabetes in U.S. adults. *Health Psychology*, 37, 61–69.
- Lespérance, F., Frasure-Smith, N., Koszycki, D., Laliberté, M. A., van Zyl, L. T., Baker, B., ... the CREATE Investigators. (2007). Effects of citalopram and interpersonal psychotherapy on depression in patients with coronary artery disease: The Canadian Cardiac Randomized Evaluation of Antidepressant and Psychotherapy Efficacy (CREATE) trial. *JAMA*, 297, 367–379.
- Lilienfeld, S. O. (2017). Clinical psychological science: Then and now. *Clinical Psychological Science*, 5, 3–13.
- Manne, S. L., Siegel, S. D., Heckman, C. J., & Kashy, D. A. (2016). A randomized clinical trial of a supportive versus a skill-based couple-focused group intervention for breast cancer patients. *Journal of Consulting and Clinical Psychology*, 84, 668–681.
- Miller-Halegoua, S., Bowen, D. J., Diefenbach, M. A., & Tercyak, K. P. (2016). Editorial: Creating the future of *Translational Behavioral Medicine*. *Translational Behavioral Medicine*, 6, 167–168.
- Nylen, K. J., O'Hara, M. W., Brock, R., Moel, J., Gorman, L., ... Stuart, S. (2010). Predictors of the longitudinal course of post-partum depression following interpersonal psychotherapy. *Journal of Consulting and Clinical Psychology*, 78, 757–763.
- O'Hara, M. W., Stuart, S., Gorman, L. L., & Wenzel, A. (2000). Efficacy of interpersonal psychotherapy for postpartum depression. *Archives of General Psychiatry*, 57, 1039–1045.
- Polenick, C. A., Renn, B. N., & Birditt, K. S. (2018). Dyadic effects of depressive symptoms on medical morbidity in middle-aged and older couples. *Health Psychology*, 37, 28–36.
- Serge, L. S., Brock, R. L., & O'Hara, M. W. (2015). Depression treatment for impoverished mothers by point-of-care providers: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 83, 314–324.
- Stice, E., Gau, J. M., Rohde, P., & Shaw, H. (2017). Risk factors that predict future onset of each DSM-5 eating disorder: Predictive specificity in high-risk adolescent females. *Journal of Abnormal Psychology*, 126, 38–51.
- Taylor, S. E. (2018). *Health psychology* (10th ed.). New York, NY: McGraw Hill.
- Trucco, E. M., Villafuerte, S., Hussong, A., Burmeister, M., & Zucker, R. A. (2018). Biological underpinnings of an internalizing pathway to alcohol, cigarette, and marijuana use. *Journal of Abnormal Psychology*, 127, 79–91.
- Yang, T.-C., Chen, D., & Park, K. (2016). Perceived housing discrimination and self-reported health: How do neighborhood features matter? *Annals of Behavioral Medicine*, 50, 789–801.

Suggested Reading

- Berkman, L. F., Blumenthal, J., Burg, M., Carney, R. M., Catellier, D., Cowan, M. J., ... Schneiderman, N. (2003). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: The Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) randomized trial. *JAMA*, 289, 3106–3116.
- Polenick, C. A., Renn, B. N., & Birditt, K. S. (2018). Dyadic effects of depressive symptoms on medical morbidity in middle-aged and older couples. *Health Psychology*, 37, 28–36.
- Yang, T.-C., Chen, D., & Park, K. (2016). Perceived housing discrimination and self-reported health: How do neighborhood features matter? *Annals of Behavioral Medicine*, 50, 789–801.

Biopsychosocial Practice in Health Psychology

Timothy P. Melchert

Department of Psychology, Marquette University, Milwaukee, WI, USA

The biopsychosocial approach has been fundamental to the practice of health psychology across the history of the specialization (e.g., Engel, 1977). Indeed, this perspective became so dominant that Suls and Rothman in 2004 concluded that “The conceptual base for health psychologists in their roles as researchers, practitioners, and policymakers is the *biopsychosocial model*” (p. 119:1; italics in the original). Despite its rapid and widespread adoption, however, the conceptual nature and precise meaning of the biopsychosocial approach was unclear, and critics viewed it as encouraging an uncritical eclectic approach to practice (e.g., Ghaemi, 2010). But scientific knowledge of health and functioning has advanced dramatically in recent years, and the scientific foundations supporting the practice of health psychology are far stronger as a result. The nature of the biopsychosocial framework underlying health psychology practice has consequently also evolved.

This chapter will outline the evolution of the biopsychosocial approach for understanding health and healthcare from a general metatheoretical framework that was often used to support an eclectic approach to practice to a solidly science-based approach grounded firmly in current scientific knowledge. It will show how scientific explanations of human health and functioning have advanced dramatically such that we now have a much more thorough understanding of the causes of disease and the behaviors that contribute to illness and health. This knowledge is critical for developing more effective prevention and treatment interventions in health psychology and in healthcare generally.

Evolution of the Biopsychosocial Approach to Health Psychology

George Engel introduced the “biopsychosocial model” for understanding health and healthcare in 1977 to counter what he viewed as the overemphasis on biology in medicine. Engel argued

that healthcare needed to take a holistic, integrative approach to understand health, disease, and treatment. This perspective was an excellent fit for the new specialization of health psychology, which also focused on interactions between biological, psychological, and socio-cultural influences on health and functioning. Engel's model appeared the year before Division 38, Health Psychology became established as a specialization within the American Psychological Association in 1978.

Engel's biopsychosocial model soon became highly influential throughout healthcare and is now widely regarded as the appropriate framework through which to understand health and healthcare. McLaren (1998) pointed out, however, that Engel misnamed his approach because he did not propose a *model* in the scientific sense of using observations, rules, and scientific laws to explain a class of phenomena, but instead used the term in its colloquial sense referring to a perspective or framework. Engel's approach technically refers to a metatheoretical framework that points to the range of factors that need to be considered to understand theory and research in medicine, but it does not refer to a scientific model of a falsifiable theory that attempts to explain a particular class of phenomena (Melchert, 2015). Engel's very general biopsychosocial framework has also been criticized for encouraging an uncritical eclecticism in behavioral healthcare because it can encompass a wide range of biological, psychological, and social interventions without requiring that they be linked to known mechanisms or processes (Ghaemi, 2010). It takes the same general approach as the various integrative and eclectic frameworks that were introduced in psychology about the same time and point to the range of factors that need to be considered to understand human psychology (e.g., Bronfenbrenner's, 1979 ecological model; Lazarus, 1976 *BASIC-ID* framework). Though these frameworks can be very useful in the early stages of researching a topic, they are not scientific models or theories that explain the behavior of particular mechanisms or systems.

There are several reasons why psychology relied on general metatheoretical frameworks to understand human development and functioning at the time the biopsychosocial model was introduced and health psychology became established as a specialization. Perhaps the most important underlying reason was that human development and functioning are so staggeringly complex that science had not yet progressed far enough to explain many of the processes involved (Melchert, 2015). Evolutionary theory still had not developed far enough to explain social behavior until the 1970s, functional magnetic resonance imaging (fMRI) was not used to observe brain function until the 1990s, and the human genome was not sequenced until the early 2000s. Given the limitations of scientific knowledge and technology at the time, all that was really possible was a metatheoretical perspective that focused attention on what appeared to be the correct range of variables that should be considered when attempting to understand many aspects of human behavior and functioning.

This situation has fundamentally changed, however, as a result of dramatic scientific progress in recent years. Replicated findings from experimental tests of falsifiable hypotheses have resulted in explanations of a steadily increasing number of biopsychosocial processes. Of course, knowledge of many biopsychosocial processes is still very limited, and many experimental findings have not yet been sufficiently tested and verified. As a result, many findings remain tentative and sometimes controversial, which is true at the frontiers of knowledge in any scientific discipline. But the overall trajectory is clear. Over the past couple decades, the behavioral, neurological, and biological sciences have advanced dramatically, and we now have verified explanations of many aspects of human psychology and biopsychosocial functioning.

Instead of referring to a general metatheoretical framework, the biopsychosocial approach is now understood to refer to the integrated body of scientific knowledge that explains human development and functioning in a manner entirely consistent with the rest of the natural

sciences (Melchert, 2015). This approach does not rely on the customary practice of choosing one (or more) of the traditional theoretical orientations for conceptualizing cases in psychology (e.g., psychodynamic, behavioral, humanistic, cognitive), but instead relies on the growing body of scientific knowledge that explains biopsychosocial mechanisms and processes. The “biopsychosocial” term is not necessarily superior to alternative terms (such as Bronfenbrenner’s “ecological” model) though it does have two distinct advantages. First, it is widely known and accepted throughout healthcare; indeed, it is accepted in many healthcare professions at levels similar to the way it is in health psychology. Second, and more importantly, it refers to the levels of natural organization that are integral to the understanding of human behavior and biopsychosocial functioning. All organic life is organized in a hierarchy of levels inherent in the natural world (i.e., from the subatomic to the level of atoms, molecules, organelles, cells, tissues, organs and organ systems, individual organisms, and, in the case of humans and many other social animals, families, communities, culture, and, for all organic life, the biosphere). The term “biopsychosocial” effectively captures the three broad, interacting levels of natural organization that are necessary for understanding human development and functioning.

The biopsychosocial framework is necessary for understanding physical as well as behavioral health and social functioning. Human health and behavior are fundamentally dependent on underlying biological structures and processes that interact with psychological factors, which in turn interact with social, cultural, and environmental processes. Appreciating the inextricably intertwined BPS domains of functioning is necessary for understanding all aspects of human functioning from the level of the particular mechanisms that control behavior (i.e., *proximate* explanations of behavior) to the level of explaining why humans are designed the way we are (i.e., the origins and functions of behavior, or *ultimate* explanations of behavior; Tinbergen, 1963).

Illustration: Current Biopsychosocial Perspective on the Primary Causes of Disease and Death

The impact of the steadily growing body of scientific knowledge regarding human health and disease can be illustrated by considering the current understanding of the primary causes of disease and death in the United States. The primary causes of morbidity and mortality are of course a main concern for health psychology research and practice. Detailed knowledge of many aspects of these topics, however, were unavailable when health psychology was founded four decades ago, but proximate and ultimate explanations for an increasing number of diseases and conditions are now available. A brief overview of this literature highlights the importance of recent scientific findings to education, practice, research, and prevention in health psychology.

Patterns of morbidity and mortality have changed dramatically in the United States and other parts of the industrialized world over the past century. At the end of the nineteenth century, the primary causes of illness and death began shifting from acute and infectious disease to chronic diseases associated with lifestyle and behavior, a shift that has been called the *epidemiological transition* (Gribble & Preston, 1993). The death rate from infectious disease was approximately 800 per 100,000 individuals in 1900 and fell to roughly 50 per 100,000 by 1950, where it has stayed since (Centers for Disease Control [CDC], 1999). Most of these deaths now involve influenza in elderly individuals, while other infectious diseases have been nearly or completely eliminated. Public health measures, vaccines, antiseptic practices in medicine, and antibiotics became so effective over the course of the twentieth century that the majority of Americans now suffer and die from chronic rather than acute infectious diseases.

The top five leading causes of death in the United States are now heart disease, cancer, chronic lower respiratory diseases, stroke, and accidents (CDC, 2013). Many of these conditions share a small number of underlying causes. At the very general level, the Institute of Medicine (2004) concluded that roughly 50% of overall morbidity and mortality in the United States is caused by behavior and lifestyle. In terms of more specific factors, smoking is the leading cause, accounting for 18.1% of all deaths (CDC, 2004); obesity is the second leading cause, accounting for 16.6% of all deaths (Mokdad, Marks, Stroup, & Gerberding, 2004); and alcohol consumption is the next most important factor, accounting for 3.5% of deaths. The World Health Organization (2008) arrived at similar conclusions regarding the global population and estimated that up to 80% of heart disease, stroke, and type 2 diabetes and more than one-third of cancers worldwide could be prevented by eliminating the shared risk factors of tobacco use, unhealthy diet, physical inactivity, and excessive alcohol use.

More detailed knowledge regarding both proximate and ultimate explanations of these diseases is also now available. Research from a range of biological, social science, and medical disciplines has clarified that the long evolution of hominins over the past 6 million years has resulted in capacities and vulnerabilities that are poorly suited to modern diets and behavioral patterns (Lieberman, 2013). Before the development of agriculture (starting 12,000 years ago in the Middle East and more recently in other areas of the world), humans were hunter-gatherers who ate primarily nuts, fruits, tubers, and some meat. Most of their food contained large amounts of fiber—in fact, chewing these foods actually consumed a large amount of time and energy; a typical meal might require thousands of chews (Lieberman, 2011). Before hybridization, the fruits they ate contained far less sugar than modern fruits (e.g., wild crab apples are similar to carrots in sweetness), the only really sweet food that could be consumed was honey, and the meat they ate from wild animals contained only a fraction of the fat found in modern farm-raised meat, fish, and poultry. Salt was not consumed in amounts larger than what was naturally present in plants and animals. Hunting and gathering (and even chewing) required large amounts of activity, and people were very lean (Lieberman, 2013).

Humans evolved to crave sweets and fats so that any excess calorie intake was quickly and easily converted into fat and people could survive periods of insufficient food intake. In contrast, since the agricultural revolution and especially since the industrial revolution, people typically eat large amounts of refined grains that contain little fiber, large amounts of sugar, and large amounts of fatty meats (Cordain et al., 2005). Humans' bodies evolved over millions of years to consume very different foods and be far more active than modern Americans. This has caused widespread problems related to high cholesterol, high blood pressure, obesity, and conditions that are caused by disuse such as back pain, osteoporosis, and dental problems, all of which are rare in hunter-gatherers (Lieberman, 2013).

Translating this growing knowledge regarding the causes of many of the most common modern medical conditions into effective treatments and prevention strategies has been challenging. The reasons for this are complex, but changing behaviors that contribute to the main causes of disease and death clearly can be difficult. Psychologists have long known that individuals can readily engage in a wide variety of behaviors that are unhealthy, self-defeating, and addictive even when they are aware of the consequences of their behavior.

Knowledge of the psychological mechanisms and processes underlying unhealthy behavior has also grown in recent years. Many issues are involved, but one prominent area of research illustrates the progress being made. It has long been known that automatic, subconscious cognitive processes operate in conjunction with reflective, conscious processes, and together they frequently produce seemingly irrational thoughts and behaviors. These two types of cognitive processing are often referred to as *System 1* and *System 2* thinking (Kahneman, 2012).

System 1 thinking is fast, automatic, largely unconscious, intuitive, reliant on affect, and conditioned emotional associations and produces a “quick and dirty” draft of reality that is often highly useful for responding to threats and opportunities. System 2 thinking, on the other hand, evolved much later, is slow and deliberate, and requires conscious effort to reason analytically and explicitly about the world. This system allows us to reason deductively and is less influenced by past events and feelings. It allows us, for example, to have a craving or a feeling such as anger or depression but not act on it due to awareness of long-term consequences or because it does not fit with one’s values. System 2 thinking is lazy, however, and often accepts the version of events provided by System 1 (Kahneman, 2012). Clearly, changing unhealthy behavior patterns in the context of these two systems can be complicated.

Implications

Suls’ and Rothman’s (2004) statement regarding the conceptual base for health psychology is still accurate if “the biopsychosocial model” is replaced with “the biopsychosocial approach.” When health psychology is approached as a clinical science grounded firmly in scientific knowledge of human health and functioning, then the term “model” needs to be used in its scientific sense. From this perspective, the biopsychosocial approach is a general metatheoretical framework that points to the levels of natural organization involved in human health and behavior and the corresponding body of scientific knowledge that provides proximate and ultimate explanations of health, disease, development, and functioning. This steadily growing body of knowledge provides a solid foundation for practicing evidence-based health psychology and will almost certainly lead to increasingly effective prevention and treatment interventions that can improve the health and well-being of the public.

When psychologists in the past entered medical units in hospitals to consult regarding psychological aspects of a case, they were sometimes apologetic and hesitant in their interactions with patients and other healthcare professionals. This attitude likely resulted in part from the equivocal support for the validity of the traditional theoretical orientations in psychology and the eclecticism associated with the biopsychosocial approach. Compared with the biological basis for much of medicine, the evidence supporting the biopsychosocial approach seemed weak. This is no longer the case. The scientific basis for understanding behavioral health and biopsychosocial functioning is now solidly based on verified research from across the biological, neurological, and behavioral sciences, and research suggests that psychosocial treatments are highly effective and sometimes more effective than many medical interventions (American Psychological Association [APA], 2012). There is certainly still much work to be done, but the scientific basis underlying health psychology is now solid. Clinicians can now interact with patients and other healthcare professionals with confidence that comes from having firm scientific foundations for practice that are entirely consistent with and supported by the rest of the natural sciences.

Author Biography

Timothy P. Melchert is a professor in the Department of Counselor Education and Counseling Psychology at Marquette University. He writes on the need to resolve theoretical and conceptual conflicts and confusion in professional psychology by adopting a unified science-based biopsychosocial framework for understanding human development, functioning, and behavior change.

References

- American Psychological Association. (2012). *Resolution of psychotherapy effectiveness*. Retrieved from www.apa.org
- Bronfenbrenner, U. (1979). *The ecology of human development*. Cambridge, MA: Harvard University Press.
- Centers for Disease Control. (1999). U.S. Department of Health and Human Services, Atlanta, Georgia, USA.
- Centers for Disease Control. (2004). *The health consequences of smoking: A report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services.
- Centers for Disease Control and Prevention. (2013). Deaths: Final data for 2013. *National Vital Statistics Report*, 64(2). Retrieved from www.cdc.gov/nchs/data_access/Vitalstatsonline.htm
- Cordain, L., Eaton, S. B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B. A., ... Brand-Miller, J. (2005). Origins and evolution of the Western diet: Health implications for the 21st century. *American Journal of Clinical Nutrition*, 81(2), 341–354.
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196, 129–136. doi:10.1126/science.847460
- Ghaemi, S. N. (2010). *The rise and fall of the biopsychosocial model: Reconciling art and science in psychiatry*. Baltimore, MD: Johns Hopkins University Press.
- Gribble, J. H., & Preston, S. H. (Eds.) (1993). *The epidemiological transition: Policy and planning implications for developing countries*. Washington, DC: National Academies Press.
- Institute of Medicine. (2004). *Improving medical education: Enhancing the behavioral and social science content of medical school curricula*. Washington, DC: National Academies Press.
- Kahneman, D. (2012). *Thinking, fast and slow*. New York, NY: Farrar, Straus & Giroux.
- Lazarus, A. A. (1976). *Multimodal behavior therapy*. New York, NY: Springer.
- Lieberman, D. E. (2011). *The evolution of the human head*. Cambridge, MA: Harvard University Press.
- Lieberman, D. E. (2013). *The story of the human body: Evolution, health, and disease*. New York, NY: Pantheon.
- McLaren, N. (1998). A critical review of the biopsychosocial model. *Australian and New Zealand Journal of Psychiatry*, 32, 86–92.
- Melchert, T. P. (2015). *Biopsychosocial practice: A science-based framework for behavioral health care*. Washington, DC: American Psychological Association.
- Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *Journal of the American Medical Association*, 291, 1238–1245. doi:10.1001/jama.291.10.1238
- Suls, J., & Rothman, A. (2004). Evolution of the biopsychosocial model: Prospects and challenges for health psychology. *Health Psychology*, 23, 119–125. doi:10.1037/0278-6133.23.2.119
- Tinbergen, N. (1963). On the aims and methods of ethology. *Zeitschrift für Tierpsychologie*, 20, 410–433.
- World Health Organization. (2008). *Primary health care: Now more than ever*. Geneva, Switzerland: Author.

Child and Family Health

Joaquin Borrego, Jr., Tabitha Fleming,
Elizabeth Ortiz-Gonzalez, and Amber Morrow

Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

The health of a child can have a significant impact on the family. When a child has a medical illness (e.g., type 2 diabetes) or experiences a negative event (e.g., child maltreatment), the family, as well as other systems (e.g., communities), can be negatively impacted. The family is inevitably impacted whether it be an acute or chronic condition. This negative impact is especially significant for family members and others close to the child (e.g., friends, extended family members). Given this, it is imperative that inclusion of the family and other systems be taken into account when discussing children's health.

Some major public health concerns impacting children and families include, but not limited to, nutrition (an increase in obesity rates for both pediatric and adult populations, food insecurity for families living in poverty), chronic illness (e.g., asthma, cardiovascular disease, high blood pressure), pediatric and adult cancer, poor sleep hygiene, and child maltreatment (the child as victim and adult as the perpetrator). These, and other conditions, are very common in our society and are associated with additional health complications later in life.

Ecological Framework

A useful framework to help understand the complexity of child, family, and social relationships as they relate to health is the ecological framework. This conceptual framework is the work conducted by Urie Bronfenbrenner (1979) on the ecology of child development. Briefly, this conceptual model posits that the child and family operate in systems that are interrelated and reciprocal. Most immediate to the child is what is known as the *microsystem*. This system is the most immediate and includes those that are closely connected to the child such as siblings, parents, family, peers, and schools. Within this microsystem, there are subsystems that focus on relationships that the child has with peers, siblings, and the parents. The *mesosystem* involves the interactions and relationships that are developed between the different components of the

microsystem. As examples, the mesosystem helps explain interactions and relationships formed between the family and the school, siblings and peers, etc. The *exosystem* involves the linkage between two settings that may directly or indirectly involve and impact the child. This can include extended family members, neighborhood, etc. The *macrosystem* is the most distant from the child but still has an influence on the child. Examples include social and cultural changes at a societal level that will inherently impact the child and family. A family's practices, values, beliefs, attitudes, and behaviors are shaped over time.

Bronfenbrenner's model has been applied to explain the complexities of the child and family within the context of health (Kazak, Alderfer, & Rader, 2017). The *social ecology model* helps identify the systems that are involved related to a child's health such as the family, hospitals, and school personnel (Kazak et al., 2017). As noted above, this is a useful conceptualization as it helps explain the intricate systems involved and the relationship between these systems. Examining these systems carefully can help identify contextual factors that may decrease the risk for continued health problems and increase resilience and adjustment for the child and family.

Impact of Children's Health on the Family

Children with medical illnesses can be very stressful for the family, especially siblings and parents or other caregivers. Illnesses in children can be stressful and even distressing for caregivers whether it be parents, guardians, or other caregivers such as a grandparent or close family friend (Cousino & Hazen, 2013). A child who is ill will miss school, and, depending on available family resources, some parents or guardians may need to miss work with or without pay. The literature from pediatric psychology suggests that a child with an illness can have a negative impact on siblings (Vermaes, van Susante, & van Bakel, 2012) and caregivers (Cousino & Hazen, 2013). There is also research to suggest that children with long, chronic illness experience difficulties across different domains in their life including academic and social problems (Pinquart & Teubert, 2012). In addition, children cannot manage their medical illness themselves, so they have to rely on family members for medical care. The level of parent or family involvement can range from being under-involved in the management of the child's illness to over-involvement such as overprotection of the child (Martire & Helgeson, 2017).

From an ecological framework, the child's negative health status has an impact within and across different systems. Within the family, a child's illness can impact his/her relationship with siblings and the parent-child relationship. These close family relationships have an impact on health (Chen, Brody, & Miller, 2017). In addition, the child's health status can also impact the relationship between parents or other caregivers. As noted above, a child with an illness can contribute to parenting stress, and, in turn, this can lead to discord in the marriage or the relationship. This discord can have a negative impact on relationship satisfaction. Parents and caregivers can become stressed and frustrated with the child or their partner in the relationship. A child missing significant amount of time from school or other events (e.g., sports-related activities) can also have a negative impact on peer relationships.

Factors Relevant to Healthcare

Health disparities can be defined as quantifiable differences in incidence, prevalence, mortality, and health indicators in certain populations or groups (e.g., Latinos, African Americans;

U.S. Surgeon General, 2001). Health disparities also include differences in access to healthcare in groups based on ethnicity/race, socioeconomic status (SES), and area of residence (Flores, 2010). This next section discusses factors that may contribute to health disparities.

Socioeconomic Status

Poverty can have a long-term deleterious impact on a child's psychological, emotional, physical, and behavioral development (Bradley & Corwyn, 2002; Yoshikawa, Aber, & Beardsee, 2012). In addition, years of research have identified profound health disparities associated with poverty (Gallo & Matthews, 2003). Particularly, families with means may provide their children with access to health services, education, and social connections that might not be available or less available to lower SES families (Lescano & Rahill, 2017). Research shows that there is a relationship between SES (an exosystemic factor) and health as SES can impact health outcomes even before birth. Growing evidence suggests that children from low SES families are at risk of suffering from neurodevelopmental abnormalities in utero, premature birth, and low birth weight (Bradley & Corwyn, 2002).

Unfortunately, low SES families are less likely to consistently follow an immunization schedule and have adequate access to healthcare and proper nutrition (Bradley & Corwyn, 2002). Nutrition has been identified as an important link between SES and psychological and physical well-being. Additionally, poor nutrition during childhood can negatively impact cognitive and behavioral development and contribute to an increase in morbidity in children. As previously mentioned, SES can contribute to biological and physiological changes in utero, during infancy, and during childhood. These disturbances create vulnerabilities that result in an overall decrease in health status and health problems that continue or manifest in adulthood. However, the degree of impact caused by low SES varies depending on how many years the child has spent in poverty and how young the child is when the family is living in poverty (Bradley & Corwyn).

Access to Healthcare

Lower SES families often have limited access to medical care, which often translates into deficient prenatal care during pregnancy and inadequate preventive care during childhood. Children from low SES families often do not have adequate immunizations, may not receive appropriate medical care when experiencing acute or chronic diseases, or may only take their children to the doctor when the disease is at an advanced stage (Bradley & Corwyn, 2002). Many of these families do not have financial means to purchase insurances and tend to rely on emergency rooms more or might avoid visiting the doctor altogether.

Ethnic Minorities and Healthcare

A disproportionate amount of families living at or below national poverty levels in the United States are ethnic and racial minorities. According to 2016 data, 22% of Black/African Americans and 19.4% of Hispanics (all races) live in poverty (U.S. Census Bureau, 2017). In contrast, 10.1% of those who identify as Asian and 8.8% of individuals who identify as White, not Hispanic, live in poverty (U.S. Census Bureau, 2017). Social disparities, low educational attainment, low income, and deficient housing contribute to the inequalities experienced by ethnic minority families (Sanders-Phillips, Settles-Reaves, Walker, & Brownlow, 2009).

Furthermore, even when adjusting for factors such as income and insurance coverage, ethnic minorities in the United States receive medical services that are of lower quality than European Americans (U.S. Surgeon General, 2001).

It is estimated that by 2020, ethnic minority children will outnumber European American children in the United States (U.S. Census Bureau, 2014). However, even when ethnic minority children are close to representing the numerical majority of the child population, profound disparities in access to healthcare remain. Research suggests that ethnic minorities are less likely to seek medical care (U.S. Surgeon General, 2001). This phenomenon can be explained by differences in beliefs and attitudes toward healthcare. Ethnic minority parents may not trust medical providers, and they may place a different emphasis on health. For example, Latino and African American parents often indicate that their doctors do not understand their children's needs (Flores, 2010). However, the intersectionality between ethnic minority status, low SES, and limited insurance coverage is a key factor that contributes to the underutilization of health services.

Racial disparities are evident when examining mortality rates in the United States. For example, childhood mortality rates tend to be significantly higher for African American children compared with European American children (Flores, 2010). African American children have the lowest immunization rates and present with high rates of obesity. The risk for obesity continues throughout adolescence, with African American female adolescents reporting a higher prevalence of obesity and needing but not getting medical care (Flores). Important disparities can be found in obesity rates, physical activity, and nutrition in Latino children as well. Research shows that Latino children have the highest rate of obesity when compared with other ethnic/racial groups (Flores).

The Influence of Culture

Psychologists and medical professionals alike are recognizing the importance and influence that culture has on different aspects of an individuals' life. Culture can shape attitudes, behaviors, practices, and values, to name a few. Culture can also influence how health distress is expressed in families, how families adjust, and how family relationships can impact health (Campos & Kim, 2017; Clay, 2017). In addition, culture has an influence on how individuals view the concept of the family. As an example, for some, the concept of who family and who is a caregiver only involves immediate family members (e.g., parents and siblings), and for others, the concept of family or caregiver is not restricted to the immediate family. As an example, in African American culture, it may be that a long-standing neighbor or friend is known as family and is not distinguished between other immediate family relatives. This is important to consider as it highlights the flexibility within which the different systems function for the child. From a cultural lens perspective, these systems are best conceptualized as flexible in parameters from one family to another.

Disparities in Access to Healthcare

Research suggests that lack of health insurance is a barrier to accessing quality healthcare (U.S. Surgeon General, 2001). This is particularly relevant for ethnic minority populations as being uninsured is a barrier for Latinos and African Americans attempting to access quality healthcare. For example, African American families are more likely to be uninsured and/or sporadically insured. Uninsured families typically experience delays and problems in accessing healthcare. Latino and African American children have an increased likelihood of not having

access to a usual source of care or primary care physician (Flores, 2010). Moreover, Latino and African American children are less likely to be referred to see a medical specialist. Research indicates that higher hospitalization rates for ambulatory care and emergency department visits for African American children and African American and Latino children have a higher rate of avoidable hospital admissions (Flores). Overall, African American and Latino children experience disparities in the use of healthcare including less visits to the doctor (spending a year or more since the last physician visit) and less calls to the doctor's office.

Language

Other disparities in access that warrant attention include difficulties in communication. Research shows that Latino families are more likely to report difficulties with doctor-patient communication with their provider (Flores, 2010). This may be due to a language barrier between the family being more proficient and comfortable speaking Spanish and medical personnel not being proficient in Spanish. Children of Spanish-speaking parents are often placed in a position to interpret for their parents or provide their own information. Developmentally, depending on the child's age, the child may not be able to provide accurate information, may be embarrassed to convey personal information, or may provide distorted information to medical personnel. This can also be the case for any family whose preferred language is not English.

Discrimination and Health Disparities

Some ethnic minority children are exposed from a young age to prejudicial attitudes and racial discrimination, and contact with discrimination may contribute to feelings of hopelessness and lead to a poor self-concept (Sanders-Phillips et al., 2009). Moreover, an awareness of minority status, which is typically achieved in adolescence, might lead to an internalized devalued group membership and identity. Adversity and negative social experiences experienced by ethnic minority children may have a long-standing effect in physiological functioning. Moreover, research shows that chronic exposure to racial discrimination correlates with biological and psychological changes that may persist and might impact health outcomes in childhood. Fortunately, the family and other systems such as churches and schools are in a position to help children cope with prejudice and racism through teaching cultural pride and racial socialization. Research suggests that having a sense of ethnic/racial identity may reduce the risk against developing of internalizing behavior problems (Smith & Trimble, 2016).

Protective and Risk Factors

As with protective and risk factors associated with psychological disorders, a host of these factors are also correlated with medical disorders and negative health-related outcomes (Mash & Barkley, 2014). A risk factor can be defined as "a characteristic at the biological, psychological, family, community, or cultural level that precedes and is associated with a higher likelihood of problem outcomes," while a protective factor can be defined as "a characteristic at the biological, psychological, family, or community (including peers and culture) level that is associated with a lower likelihood of problem outcomes or that reduces the negative impact of a risk factor on problem outcomes" (National Research Council and Institute of Medicine, 2009, pp. xxvii-xxviii). Across the lifespan, protective and risk factors tend to vary according to developmental stage beginning in early childhood all the way through middle childhood,

adolescence, and adulthood. However, some protective or risk factors are present in several stages over the course of development. These factors play different roles in shaping a child, and, in turn the family, thus it is important to be cognizant of each and their role in the child's health as well as in the outcomes associated.

In early childhood, secure attachment is crucial for healthy development that has implications later in life for emotion regulation, family relationships, and prosocial behavior (National Research Council and Institute of Medicine, 2009). Through building a foundation of consistent and positive and parent-child interactions, a secure relationship can be formed and solidified early, promoting healthy child development. A positive home environment can also contribute to development of executive functioning skills (e.g., problem-solving skills, language acquisition), encourage early childhood and lifelong positive psychological, emotional, and behavioral health (National Research Council and Institute of Medicine, 2009).

In middle childhood, it is increasingly important for children to successfully achieve appropriate developmental milestones such as academic achievement, following rules for appropriate behavior and positive peer relations. As noted earlier, academic and social problems can develop for children with chronic illnesses. A child's ability to master academic skills, along with making friends with peers, can contribute to protecting against poor health or overall negative outcomes. Additionally, the concept of resilience is a key component eliciting positive developmental outcomes in this stage of development (National Research Council and Institute of Medicine, 2009). The resilient ability to persevere despite adversity protects children in middle childhood from potentially problematic outcomes.

In adolescence, essential protective factors leading to positive development and child health include engagement in positive physical health habits, intellectual development, physiological and emotional development, and social development (National Research Council and Institute of Medicine, 2009). On an individualized level, adolescents who exhibit high sense of self-worth, emotional regulation, and good coping and problem-solving skills are more equip to avoid or minimize the impact of negative outcomes (National Research Council and Institute of Medicine, 2009). From a family and school perspective, protective factors in adolescence also include receiving extended family support and engaging in positive peer relationships. Further, different systems can each play a role in helping reduce health-compromising behaviors and increasing health-promoting behaviors in youth (Wilson, Coulon, & Huffman, 2017).

These protective factors associated with outcomes of children across the development are important to consider in contrast with the risk factors also associated. Across the lifespan, there are many risk factors associated with negative health outcomes that can be detrimental regardless of developmental stage. Such risk factors including child maltreatment, family dysfunction (e.g., poor family communication, avoidance of negative emotions) community and school risk factors (e.g., violence), and poverty can contribute to a host of problematic outcomes in children of all ages (National Research Council and Institute of Medicine, 2009).

Further, family dysfunction and disruption including divorce, a poor parent-child relationship, insecure attachment, and parental psychopathology can also elevate risk for negative outcomes in children across all developmental stages (National Research Council and Institute of Medicine, 2009). Additionally, community and school risk factors including victimization, bullying, academic failure, and association with deviant peers contribute to potential negative health outcomes in children (National Research Council and Institute of Medicine, 2009).

Conclusion

The health of the child and family has such an important and profound impact on each other. This reciprocal system, in turn, impacts other systems in the child's life such as development of peer relationships and academic performance in schools. We have a sufficient scientific knowledge base regarding prevention and early intervention programs that can assist in raising children with prosocial skills and having close family relationships (Biglan, 2015). In addition, we can now point to numerous social determinants of health that can have a negative impact on the child and family (Black, 2017). Having close family relationships can ameliorate some of the negative impact that acute and chronic medical conditions can have on the child, family, and other systems such as peers, schools, and communities. Practitioners and researchers alike are encouraged to closely examine the quality of the parent–child relationship as well as the quality of the sibling relationship and the caregiver's relationship. In addition to examining family cohesion, the degree to which the child and family have a positive and supportive social network should be taken into account. This can include the child's peer relationships and the family's network extended family members and friends through work, the child's school, and the community. At a broader level, schools and the healthcare system need to be examined in the context of child and family health.

Author Biographies

Joaquin Borrego, Jr., PhD, is an associate professor in the Department of Psychological Sciences at Texas Tech University. He is a member of the clinical psychology doctoral program and directs the Parent-Child Interaction Therapy (PCIT) research lab. His research interests include parent–child relationships, discipline practices, culture, and prevention and early intervention of child physical abuse. He moved to Department of Psychology, Pacific University, Forest Grove, Oregon, USA, in summer 2018, after this chapter was initially written.

Tabitha Fleming, MA, is a doctoral student in the Texas Tech University Clinical Psychology doctoral program and a member of the Parent-Child Interaction Therapy (PCIT) research lab. Her research interests include parent management, parental discipline practices, and prevention of child maltreatment.

Elizabeth Ortiz-Gonzalez, MA, is a doctoral student in the Texas Tech University Clinical Psychology doctoral program and a member of the Parent-Child Interaction Therapy (PCIT) research lab. Her research interests include parenting practices with ethnic minority families, particularly Latino families.

Amber Morrow, BA, is a doctoral student in the Texas Tech University Clinical Psychology doctoral program and a member of the Parent-Child Interaction Therapy (PCIT) research lab. Her research interests include discipline practices and the prevention of the use of physical punishment and child maltreatment.

References

- Biglan, A. (2015). *The nurture effect: How the science of human behavior can improve our lives and our world*. Oakland, CA: New Harbinger.
- Black, M. (2017). Prevention: A multilevel, biobehavioral, lifespan perspective. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (pp. 538–549). New York, NY: Guilford Press.

- Bradley, R., & Corwyn, R. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, 53, 371–399.
- Bronfenbrenner, U. (1979). *The ecology of human development*. Cambridge, MA: Harvard University Press.
- Campos, B., & Kim, H. S. (2017). Incorporating the cultural diversity of family and close relationships into the study of health. *American Psychologist*, 72, 543–554.
- Chen, E., Brody, G. H., & Miller, G. E. (2017). Childhood close family relationships and health. *American Psychologist*, 72, 555–566.
- Clay, D. L. (2017). Culture and diversity in research and practice. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (pp. 81–91). New York, NY: Guilford Press.
- Cousino, M. K., & Hazen, R. A. (2013). Parenting stress among caregivers of children with chronic illness: A systematic review. *Journal of Pediatric Psychology*, 38, 809–828.
- Flores, G. (2010). Technical report: Racial and ethnic disparities in the health and health care of children. *Pediatrics*, 125(4), 979–1020.
- Gallo, L., & Matthews, K. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin*, 129, 10–51.
- Kazak, A. E., Alderfer, M. A., & Rader, S. K. (2017). Families and other systems in pediatric psychology. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (pp. 566–579). New York, NY: Guilford Press.
- Lescano, C. M., & Rahill, G. J. (2017). Racial and ethnic health disparities. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (pp. 499–508). New York, NY: Guilford Press.
- Martire, L. M., & Helgeson, V. S. (2017). Close relationships and the management of chronic illness: Associations and interventions. *American Psychologist*, 72, 601–612.
- Mash, E. J., & Barkley, R. A. (2014). *Child psychopathology* (3rd ed.). New York, NY: Guilford Press.
- National Research Council and Institute of Medicine. (2009). *Preventing mental, emotional, and behavioral disorders among young people progress and possibilities*. Washington, DC: National Academic Press.
- Pinquart, M., & Teubert, D. (2012). Academic, physical, and social functioning of children and adolescents with chronic physical illness: A meta-analysis. *Journal of Pediatric Psychology*, 37, 376–389.
- Sanders-Phillips, K., Settles-Reaves, B., Walker, D., & Brownlow, J. (2009). Social inequality and racial discrimination: Risk factors for health disparities in children of color. *Pediatrics*, 124(3), 176–186.
- Smith, T. B., & Trimble, J. E. (2016). *Foundations of multicultural psychology: Research to inform effective practice*. Washington, DC: American Psychological Association.
- U.S. Census Bureau. (2014). *Projections and size of the U.S. population: 2014–2060*. Washington, DC: Author.
- U.S. Census Bureau. (2017). *Income and poverty in the United States: 2016*. Washington, DC: Author.
- U.S. Surgeon General. (2001). *Mental health: Culture, race, and ethnicity. A supplement to mental health: A report of the Surgeon General*. Rockville, MD: Author.
- Vermaes, I. P. R., van Susante, A. M. J., & van Bakel, H. J. A. (2012). Psychological functioning of siblings in families of children with chronic health conditions: A meta-analysis. *Journal of Pediatric Psychology*, 37, 166–177.
- Wilson, D. K., Coulon, S. M., & Huffman, L. E. (2017). Health promotion in children and adolescents: An integration of the biopsychosocial model and ecological approaches to behavior change. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (pp. 566–579). New York, NY: Guilford Press.
- Yoshikawa, H., Aber, J. L., & Beardsee, W. R. (2012). The effects of poverty on the mental, emotional, and behavioral health of children and youth. *American Psychologist*, 67, 272–284.

Suggested Reading

- Biglan, A. (2015). *The nurture effect: How the science of human behavior can improve our lives and our world*. Oakland, CA: New Harbinger.
- Roberts, M. C., & Steele, R. G. (2017). *Handbook of pediatric psychology*. New York, NY: Guilford Press.

Childhood Cancer

Dianna Boone, Savannah Davidson, Jordan Degelia, and
Jason Van Allen

*Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University,
Lubbock, TX, USA*

Introduction

Pediatric cancers are newly diagnosed in approximately 14,000 youth in the United States each year (Howlader et al., 2013) and are the leading cause of death by disease for children (Heron et al., 2010). Acute lymphoblastic leukemia (ALL) is the most common form of pediatric cancer and is responsible for one-fourth of all childhood cancers and 75% of all cases of childhood leukemia (Belson, Kingsley, & Holmes, 2007). In 2014, nearly 16,000 children and adolescents were diagnosed with cancer in the United States. In addition, more than 80% of those diagnosed with pediatric cancer will survive at least 5 years after their diagnosis (Wessler, 2015). This chapter provides an overview of the short- and long-term effects of cancer on children and their families, with special attention paid to psychosocial outcomes and intervention efforts. The importance of medication adherence and the health challenges associated with survivorship are also discussed.

Psychosocial Functioning in Pediatric Cancer

Many studies have examined the impact of pediatric cancer on the psychological and emotional functioning of children, adolescents, and their families (Germann et al., 2015). The psychological effects of cancer therapy include increased levels of anxiety, depression, and posttraumatic stress (Dietz & Mulrooney, 2011). Studies have also indicated that children diagnosed with cancer have significantly higher internalizing symptoms (Sawyer, Antoniou, Toogood, & Rice, 1997) and a potentially higher risk of suicidal ideation than the general population (Recklitis et al., 2010). Research has illustrated that these psychosocial effects are typically experienced near the time of diagnosis and early in the treatment process (Compas et al., 2014; Kazak,

Boeving, Alderfer, Hwang, & Reilly, 2005). However, some studies have also illustrated that children with cancer demonstrate better emotional functioning than comparison groups (Van Schoors, Caes, Verhofstadt, Goubert, & Alderfer, 2015) and have not shown higher rates of depression or posttraumatic stress than population norms (Germann et al., 2015).

Health-Related Quality of Life

A component of psychosocial functioning that may be affected by cancer is health related quality of life. Health-related quality of life (e.g., a broad multidimensional concept that usually includes self-reported measures of physical and mental health; Centers for Disease Control and Prevention, 2012) has also been found to be lower in children undergoing treatment for pediatric cancer when compared with the general population, particularly in the areas of physical complaints, motor functioning, and positive emotional functioning (Fakhry et al., 2013). It has also been found that among children with ALL, females, older children, and those with multiple medical complications were at a higher risk of poor quality of life (Fakhry et al., 2013). Demographic factors such as age, gender, type of cancer, treatment modality/intensity, and progression of illness have all been significantly related to health-related quality of life (Fakhry et al., 2013).

Depression and Anxiety

In addition to lower health-related quality of life, research has shown that increased levels of depression and anxiety can result from cancer treatment as well (Dietz & Mulrooney, 2011). Myers et al. (2014) examined the emotional and behavioral functioning of a large sample of children with ALL. Both child and parent reports were obtained on measures related to the child's behavior, anxiety, hyperactivity, aggression, and depression. These measures were given at three different time points (e.g., 1, 6, and 12 months after being given a diagnosis). The results revealed that on average, all of the child outcome variables were similar to population norms; however, more children scored in the at-risk range for depression at 1, 6, and 12 months (Myers et al., 2014). Children scored in the at-risk range for anxiety at 1 month only (Myers et al., 2014).

Similarly, Compas et al. (2014) examined anxiety and depression in a sample of children recently diagnosed with cancer. They found that based on reports from children and their caregivers, children in the sample experienced mild to moderate symptoms of anxiety and depression (Compas et al., 2014). Both of these studies reveal that anxiety and depression may be a significant problem for children after receiving a diagnosis of pediatric cancer (Myers et al., 2014).

Resiliency

Although pediatric cancer can result in negative psychosocial outcomes (e.g., increased depression and anxiety and lower quality of life), research has demonstrated that children can show healthy developmental trajectories despite a cancer diagnosis. This positive adaption, often termed resiliency (e.g., achieving positive outcomes despite exposure to significant levels of adversity), has been found to be significantly related to better adjustment (Germann et al., 2015). Germann et al. (2015) conducted a study to determine the pattern of resiliency and adjustment (measured by hope, anxiety, depression, and quality of life) in children between ages 8 and 18 as well as to examine the relationships between these variables. Overall, results

demonstrated that pediatric cancer patients are resilient and that one's level of hope has the potential to maintain and promote adjustment and well-being in children with cancer (Germann et al., 2015). A systematic review was also conducted to examine family resiliency in the context of pediatric cancer. Van Schoors et al. (2015) found that although a diagnosis of pediatric cancer disrupts family functioning, most families are resilient and adapt well to receiving a pediatric cancer diagnosis.

Findings from these studies suggest that pediatric cancer patients may experience anxiety and depression, particularly near the time of diagnosis and early in the treatment process (Kazak et al., 2005; Pinquart & Shen, 2011). However, research also indicates that families demonstrate resiliency when facing pediatric cancer (Van Schoors et al., 2015). Thus, it may be important for researchers to develop interventions that promote positive psychosocial outcomes such as resiliency in children undergoing cancer treatment.

Family Adjustment to a Pediatric Cancer Diagnosis

Families of children with cancer are also at risk for poor adjustment outcomes, including posttraumatic stress symptoms (Rodriguez et al., 2012) and anxious and depressive symptoms (Pai et al., 2007). Research has demonstrated that the way a child's parents adjust after their child is diagnosed with cancer can be crucial to their child's adjustment following diagnosis (Rodriguez et al., 2016). Thus, interventions targeting parental adjustment can result in positive outcomes for their children (Fedele et al., 2013; Phipps et al., 2015). Fedele et al. (2013) conducted a study to determine if maternal distress predicted child adjustment outcomes and if a parent-focused interdisciplinary intervention positively affected children's adjustment. It was found that maternal distress predicted child internalizing symptoms, which suggests that interventions aiming to improve child adjustment outcomes should consider incorporating parent factors (Fedele et al., 2013). This study also found that the parent-focused intervention significantly reduced child internalizing symptoms, which indicates that psychological interventions for mothers of children newly diagnosed with cancer can positively influence child adjustment outcomes (Fedele et al., 2013).

In addition, research has demonstrated that certain sociodemographic factors can be associated with psychological distress in families facing pediatric cancer (Bemis et al., 2015). Bemis et al. (2015) found that factors representing sociodemographic disadvantage (i.e., single parenthood, lower family income, lower parental education, non-White race) may influence general perceived stress, cancer-related stressors, and psychological distress in families experiencing pediatric cancer (Bemis et al., 2015). The results of this study are important, as researchers will be able to develop effective interventions for families who are more likely to experience psychological distress (Bemis et al., 2015).

Overall, it is essential to understand the ways that children and families cope with the effects of cancer to develop optimal interventions for improving coping skills in children (Compas et al., 2014). It is important that interventions continue to focus on not only child psychosocial outcomes but family functioning as well.

Medication Adherence

Pediatric psychologists often collaborate with multidisciplinary teams in oncology to improve medication adherence. Despite the importance of taking medications as prescribed, some

studies have shown that as many as 50% of pediatric cancer patients do not adhere to their maintenance treatment regimen (Kondryn, Edmondson, Hill, & Eden, 2011). Nonadherence can take many forms (e.g., not filling prescriptions, skipping or missing doses) and can be intentional (e.g., deciding not to take a medication because of side effects) or unintentional (e.g., forgetting; Hommel & Baldassano, 2010). Poorer adherence has been documented for regimens that are more time consuming and complex, and rates of nonadherence in pediatric chronic illness populations, especially among adolescents, are even higher than those in adult populations (Rapoff, 2010). Nonadherence to a medication regimen, especially for pediatric cancer patients, can potentially interfere with the efficacy of drug regimens, resulting in difficulties meeting treatment goals, more frequent visits to the emergency room, or increased hospitalizations.

Barriers to Medication Adherence

Medication adherence, particularly in adolescents with cancer, can be more challenging because adolescents are generally diagnosed with higher risk cancers than younger children; thus, adolescents have more complicated treatment plans (Hullmann, Brumley, & Schwartz, 2015). Hullmann et al. (2015) investigated parent and adolescent reported rates of, and reasons for, nonadherence, as well as the demographic, treatment and disease-related, and psychosocial correlates of nonadherence. They found that adolescent- and parent-reported adherence were significantly correlated, with approximately half of the sample reporting perfect adherence (Hullmann et al., 2015). The most common reason for missed medication reported by adolescents and caregivers was “just forgot” (Hullmann et al., 2015). Interestingly, for adolescents who endorsed perfect adherence, they reported having greater social support from their families (Hullmann et al., 2015). These results indicate that children receiving family support are more likely to adhere to their medication regimens (Hullmann et al., 2015).

Interventions That Aim to Promote Adherence

Due to the importance of medication adherence to positive health outcomes for pediatric cancer patients, interventions promoting medication adherence are necessary. Pai and McGrady (2015) recently conducted a literature review examining interventions that aimed to improve medication adherence in pediatric cancer patients. The results indicate that a multifaceted approach is needed to promote medication adherence in this population (Pai & McGrady, 2015). More specifically, these researchers suggest that multidisciplinary teams should combine “adherence-related assessments, education, anticipatory guidance, and documentation in standard clinical care” (Pai & McGrady, 2015). It was also concluded that medication adherence should be consistently monitored throughout the course of treatment (Pai & McGrady, 2015). Interventions targeting family support and psychosocial functioning with an educational component have also been shown to be more effective in improving medication adherence compared with control conditions (Pai & McGrady, 2015).

In sum, proper assessment of medication adherence in pediatric cancer patients is crucial. Nonadherence increases one’s risk of developing complications and can lead to treatment goals not being met. Although there are numerous barriers to medication adherence (e.g., side effects of medication, cost of treatment, poor planning), research has demonstrated that improving family involvement is helpful in encouraging medication adherence. Research on interventions to improve adherence in this population has demonstrated that adherence should be promoted through a multifaceted approach, include an educational component, and be

monitored routinely and throughout the course of one's treatment (Pai & McGrady, 2015; Rohan et al., 2015).

Health Challenges Associated With Survivorship

After surviving pediatric cancer, the patient may experience other chronic health issues such as delayed growth, endocrinological problems, and neurocognitive impairment (Bhatia, 2005). Research has demonstrated that childhood cancer survivors are at a significantly greater risk of developing secondary cancers, cardiovascular disease, osteoporosis, and diabetes (Greving & Santacroce, 2005). Comorbid conditions can result from cancer treatment, genetic predisposition, or various lifestyle factors (Demark-Wahnefried, Aziz, Rowland, & Pinto, 2005).

Neurological Functioning After Cancer Treatment

Survivors of cancer are also at greater risk for adverse neurocognitive outcomes (Patel et al., 2014). Certain methods of treatment such as cranial radiation therapy put children at risk for "late effects" that emerge after cessation of treatment (Ness & Gurney, 2007). Long-term effects of treatment can include malignant brain tumors and neurocognitive deficits such as deficits in attention, executive functioning, memory, and processing speed (Butler & Mulhern, 2005; Patel et al., 2014).

However, interventions have demonstrated efficacy in treating these adverse neurocognitive effects resulting from cancer treatment (Patel et al., 2014). For example, Butler et al. (2008) designed a cognitive remediation intervention for pediatric cancer survivors with documented attention disturbance. Results indicated that the children showed small to modest improvements in academic achievement and parent-reported behavioral functioning (Butler et al., 2008). Similarly, Patel et al. (2014) conducted a study to evaluate the efficacy of a similar intervention directed toward parents of childhood cancer survivors with neurocognitive late effects, to improve targeted parenting skills, and therefore to improve their children's educational performance. Results revealed that children who participated in the intervention significantly increased their use of study strategies (Patel et al., 2014). Additionally, caregivers' self-efficacy to promote their child's school success increased after the intervention (Patel et al., 2014). These studies show that interventions can potentially improve cognitive functioning in child cancer survivors.

Increased Risk for Obesity

There is also an increased risk for weight gain and decreased physical activity following cancer treatment (Stern et al., 2015). For example, 5 years' posttreatment, 21% of all pediatric cancer survivors are classified as obese (BMI $\geq$ 95th percentile for age and gender) and 20% as overweight (BMI $\geq$ 85th percentile and $\leq$ 94th percentile for age and gender; Chow, Pihoker, Hunt, Wilkinson, & Friedman, 2007). Children with certain types of cancers (e.g., ALL and specific sarcomas) are at an even greater risk for obesity (Chow et al., 2007). Research has illustrated that the association between cancer treatment and future obesity may be due to cranial radiation and exposure to corticosteroids (Chow et al., 2007). Additionally, consumption of high fat foods and limited physical activity could influence posttreatment weight gain.

However, researchers have developed interventions that aim to promote healthy lifestyles for children after cancer treatment. Family-based interventions that treat pediatric obesity by

promoting healthy lifestyle changes, physical activity, and dietary behaviors are effective, and it is likely that these types of interventions are also effective for child cancer survivors (Stern et al., 2015).

Conclusion

In sum, pediatric cancer is a prevalent and potentially life-threatening illness that affects the entire family (Long & Marsland, 2011; Ward, DeSantis, Robbins, Kohler, & Jemal, 2014). Although many families facing pediatric cancer show resiliency (Van Schoors et al., 2015), research indicates that these children often experience psychosocial problems including anxiety, depression, and posttraumatic stress (Dietz & Mulrooney, 2011; Seitz et al., 2010). In addition to psychosocial problems, children and adolescents may also experience late effects of cancer including neurocognitive deficits, such as with attention and concentration (Butler & Mulhern, 2005). Research in pediatric cancer indicates that there are often significant health challenges associated with survivorship, which include increased risk for obesity, delayed growth, endocrinological problems and neurocognitive impairment (Krivoy, Jenney, Mahajan, & Peretz Nahum, 2012), and other chronic illnesses (Bottomley & Kassner, 2003; Greving & Santacroce, 2005). Pediatric psychologists will continue to address and develop solutions to these challenges to promote resiliency and quality of life in pediatric cancer patients.

Author Biographies

Dianna Boone, MA. Dianna is a second year student in the clinical psychology program at Texas Tech University, under the mentorship of Dr. Jason Van Allen. She received her MA in Rehabilitation and Mental Health Counseling from the University of South Florida. Her research interests include pediatric psychology and childhood obesity.

Savannah Davidson, BA. Savannah received her bachelor's degree in psychology from Texas Tech University and is currently a graduate student in the School Psychology Program at the University of Texas at Austin.

Jordan Degelia, BA. Jordan received her bachelor's degree in psychology from Texas Tech University and is currently a graduate student in the Social Work Program at Texas Tech.

Jason Van Allen, PhD. Jason is an associate professor in the Clinical Psychology Program at Texas Tech University. He received his PhD in Clinical Child Psychology from the University of Kansas, and his research focuses on pediatric psychology and childhood obesity.

References

- Belson, M., Kingsley, B., & Holmes, A. (2007). Risk factors for acute leukemia in children: A review. *Environmental Health Perspectives*, 115(1), 138–145. doi:10.1289/ehp.9023
- Bemis, H., Yarboi, J., Gerhardt, C. A., Vannatta, K., Desjardins, L., Murphy, L. K., ... Compas, B. E. (2015). Childhood cancer in context: Sociodemographic factors, stress, and psychological distress among mothers and children. *Journal of Pediatric Psychology*, 40(8), 733–743. doi:10.1093/jpepsy/jsv024
- Bhatia, S. (2005). Cancer survivorship—Pediatric issues. *ASH Education Program Book*, 2005(1), 507–515. doi:10.1182/asheducation-2005.1.507

- Bottomley, S. J., & Kassner, E. (2003). *Journal of Pediatric Nursing*, 18(2), 126–133.
- Butler, R. W., Copeland, D. R., Fairclough, D. L., Mulhern, R. K., Katz, E. R., Kazak, A. E., ... Sahler, O. J. Z. (2008). A multicenter, randomized clinical trial of a cognitive remediation program for childhood survivors of a pediatric malignancy. *Journal of Consulting and Clinical Psychology*, 76(3), 367. doi:10.1037/0022-006X.76.3.367
- Butler, R. W., & Mulhern, R. K. (2005). Neurocognitive interventions for children and adolescents surviving cancer. *Journal of Pediatric Psychology*, 30(1), 65–78. doi:10.1093/jpepsy/jsi017
- Centers for Disease Control and Prevention. (2012). Retrieved from <http://www.cdc.gov/hrqol/>
- Chow, E. J., Pihoker, C., Hunt, K., Wilkinson, K., & Friedman, D. L. (2007). Obesity and hypertension among children after treatment for acute lymphoblastic leukemia. *Cancer*, 110(10), 2313–2320. doi:10.1002/cncr.23050
- Compas, B. E., Desjardins, L., Vannatta, K., Young-Saleme, T., Rodriguez, E. M., Dunn, M., ... Gerhardt, C. A. (2014). Children and adolescents coping with cancer: Self- and parent reports of coping and anxiety/depression. *Health Psychology*, 33, 853–861. doi:10.1037/hea0000083
- Demark-Wahnefried, W., Aziz, N. M., Rowland, J. H., & Pinto, B. M. (2005). Riding the crest of the teachable moment: Promoting long-term health after the diagnosis of cancer. *Journal of Clinical Oncology*, 23(24), 5814–5830. doi:10.1200/JCO.2005.01.230
- Dietz, A. C., & Mulrooney, D. A. (2011). Life beyond the disease: Relationships, parenting, and quality of life among survivors of childhood cancer. *Haematologica*, 96(5), 643–645. doi:10.3324/haematol.2011.042606
- Fakhry, H., Goldenberg, M., Sayer, G., Aye, S. S., Bagot, K., Pi, S., ... IsHak, W. W. (2013). Health-related quality of life in childhood cancer. *Journal of Developmental and Behavioral Pediatrics*, 34(6), 419–440. doi:10.1097/DBP.0b013e31828c5fa6
- Fedele, D. A., Hullmann, S. E., Chaffin, M., Kenner, C., Fisher, M. J., Kirk, K., ... Mullins, L. L. (2013). Impact of a parent-based interdisciplinary intervention for mothers on adjustment in children newly diagnosed with cancer. *Journal of Pediatric Psychology*, 38(5), 531–540. doi:10.1093/jpepsy/jst010
- Germann, J. N., Leonard, D., Stuenzi, T. J., Pop, R. B., Stewart, S. M., & Leavey, P. J. (2015). Hoping is coping: A guiding theoretical framework for promoting coping and adjustment following pediatric cancer diagnosis. *Journal of Pediatric Psychology*, 40(9), 846–855. doi:10.1093/jpepsy/jsv027
- Greving, D. M., & Santacroce, S. J. (2005). Cardiovascular late effects. *Journal of Pediatric Nursing Oncology*, 22(1), 38–47. doi:10.1177/1043454204272531
- Heron, M., Sutton, P. D., Xu, J., Venture, S. J., Strobino, D. M., & Guyer, B. (2010). Annual review of vital statistics: 2007. *Pediatrics*, 125(1), 4–15. doi:10.1542/peds.2009-2416
- Hommel, K. A., & Baldassano, R. N. (2010). Brief report: Barriers to treatment adherence in pediatric inflammatory bowel disease. *Journal of Pediatric Psychology*, 35(9), 1005–1010. doi:10.1093/jpepsy/jsp126
- Howlader, N., Noone, A. M., Krapcho, M., Garshell, J., Neyman, N., Altekruse, S. F., & Cho, H. (2013). *SEER cancer statistics review, 1975–2010*. [Based on the November 2012 SEER data submission, posted to the SEER web site, April 2013.]. Bethesda, MD: *National Cancer Institute*.
- Hullmann, S., Brumley, L., & Schwartz, L. (2015). Medical and psychosocial associates of nonadherence in adolescents with cancer. *Journal of Pediatric Oncology Nursing*, 32(2), 103–113. doi:10.1177/1043454214553707
- Kazak, A., Boeving, C., Alderfer, M., Hwang, W., & Reilly, A. (2005). Posttraumatic stress symptoms during treatment in parents of children with cancer. *Journal of Clinical Oncology*, 23(30), 7405–7410. doi:10.1200/JCO.2005.09.110
- Kondryn, H. J., Edmondson, C. L., Hill, J., & Eden, T. O. (2011). Treatment non-adherence in teenage and young adult patients with cancer. *The Lancet Oncology*, 12(1), 100–108. doi:10.1016/S1470-2045(10)70069-3
- Krivoy, E., Jenney, M. E. M., Mahajan, A. M., & Peretz Nahum, M. (2012). Survivorship in childhood cancer. In S. Kreitler, M. W. Ben Arush, & A. Martin (Eds.), *Pediatric psycho-oncology*. Oxford, UK: Wiley-Blackwell.

- Long, K. A., & Marsland, A. L. (2011). Family adjustment to childhood cancer: A systematic review. *Clinical Child and Family Psychology Review*, 14(1), 57–88. doi:10.1007/s10567-010-0082-z
- Myers, R. M., Balsamo, L., Lu, X., Devidas, M., Hunger, S. P., Carroll, W. L., ... Kadan-Lottick, N. S. (2014). A prospective study of anxiety, depression, and behavioral changes in the first year after a diagnosis of childhood acute lymphoblastic leukemia. *Cancer*, 120(9), 1417–1425. doi:10.1002/cncr.28578
- Ness, K. K., & Gurney, J. G. (2007). Adverse late effects of childhood cancer and its treatment on health and performance. *Annual Review of Public Health*, 28, 279–302. doi:10.1146/annurev.publhealth.28.021406.144049
- Pai, A. L., Greenley, R. N., Lewandowski, A., Drotar, D., Youngstrom, E., & Peterson, C. C. (2007). A meta-analytic review of the influence of pediatric cancer on parent and family functioning. *Journal of Family Psychology*, 21(3), 407–415. doi:10.1037/0893-3200.21.3.407
- Pai, A. L., & McGrady, M. E. (2015). Assessing medication adherence as a standard of care in pediatric oncology. *Pediatric Blood & Cancer*, 62(S5), S696–S706. doi:10.1002/pbc.25795
- Patel, S. K., Ross, P., Cuevas, M., Turk, A., Kim, H., Lo, T. T., & Bhatia, S. (2014). Parent-directed intervention for children with cancer-related neurobehavioral late effects: A randomized pilot study. *Journal of Pediatric Psychology*, 39(9), 1013–1027. doi:10.1093/jpepsy/jsu045
- Phipps, S., Long, A., Willard, V. W., Okado, Y., Hudson, M., Huang, Q., & Noll, R. (2015). Parents of children with cancer: At-risk or resilient? *Journal of Pediatric Psychology*, 40(9), 914–925. doi:10.1093/jpepsy/jsv047
- Pinquart, M., & Shen, Y. (2011). Behavior problems in children and adolescents with chronic physical illness: A meta-analysis. *Journal of Pediatric Psychology*, 36(9), 1003–1016. doi:10.1093/jpepsy/jsr042
- Rapoff, M. (2010). Editorial: Assessing and enhancing clinical significance/social validity of intervention research in pediatric psychology. *Journal of Pediatric Psychology*, 35(2), 114–119. doi:10.1093/jpepsy/jsp102
- Recklitis, C., Diller, L., Li, X., Najita, J., Robison, L., & Zeltzer, L. (2010). Suicide ideation in adult survivors of childhood cancer: A report from the Childhood Cancer Survivor Study. *Journal of Clinical Oncology*, 28(4), 655–661. doi:10.1200/JCO.2009.22.8635
- Rodriguez, E. M., Dunn, M. J., Zuckerman, T., Vannatta, K., Gerhardt, C. A., & Compas, B. E. (2012). Cancer-related sources of stress for children with cancer and their parents. *Journal of Pediatric Psychology*, 37(2), 185–197. doi:10.1093/jpepsy/jsr054
- Rodriguez, E. M., Murphy, L., Vannatta, K., Gerhardt, C. A., Young-Saleme, T., Saylor, M., ... Compas, B. E. (2016). Maternal coping and depressive symptoms as predictors of mother–child communication about a child’s cancer. *Journal of Pediatric Psychology*, 41(3), 329–339. doi:10.1093/jpepsy/jsv106
- Rohan, J., Drotar, D., Alderfer, M., Donewar, C., Ewing, L., Katz, E., & Muriel, A. (2015). Electronic monitoring of medication adherence in early maintenance phase treatment for pediatric leukemia and lymphoma: Identifying patterns of nonadherence. *Journal of Pediatric Psychology*, 40(1), 75–84. doi:10.1093/jpepsy/jst093
- Sawyer, M., Antoniou, G., Toogood, I., & Rice, M. (1997). Childhood cancer: A two-year prospective study of the psychological adjustment of children and parents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(12), 1736–1743. doi:10.1097/00004583-199712000-00022
- Seitz, D. C., Besier, T., Debatin, K. M., Grabow, D., Dieluweit, U., Hinz, A., ... Goldbeck, L. (2010). Posttraumatic stress, depression and anxiety among adult long-term survivors of cancer in adolescence. *European Journal of Cancer*, 46(9), 1596–15606. doi:10.1016/j.ejca.2010.03.001
- Stern, M., Ewing, L., Davila, E., Thompson, A. L., Hale, G., & Mazzeo, S. (2015). Design and rationale for NOURISH-T: A randomized control trial targeting parents of overweight children off cancer treatment. *Contemporary Clinical Trials*, 41, 227–237. doi:10.1016/j.cct.2014.12.018
- Van Schoors, M., Caes, L., Verhofstadt, L. L., Goubert, L., & Alderfer, M. A. (2015). Systematic review: Family resilience after pediatric cancer diagnosis. *Journal of Pediatric Psychology*, 40(9), 856–868. doi:10.1093/jpepsy/jsv055

- Ward, E., DeSantis, C., Robbins, A., Kohler, B., & Jemal, A. (2014). Childhood and adolescent cancer statistics, 2014. *CA: A Cancer Journal for Clinicians*, 64(2), 83–103. doi:10.3322/caac.21219
- Wessler, J. M. (2015). Leukemia in children getting back to school—Part 1. *NASN School Nurse*, 30(2), 116–118. doi:10.1177/1942602X15570256

Suggested Reading

- Compas, B. E., Desjardins, L., Vannatta, K., Young-Saleme, T., Rodriguez, E. M., Dunn, M., ... Gerhardt, C. A. (2014). Children and adolescents coping with cancer: Self- and parent reports of coping and anxiety/depression. *Health Psychology*, 33, 853–861. doi:10.1037/hea0000083
- Germann, J. N., Leonard, D., Stuenzi, T. J., Pop, R. B., Stewart, S. M., & Leavey, P. J. (2015). Hoping is coping: A guiding theoretical framework for promoting coping and adjustment following pediatric cancer diagnosis. *Journal of Pediatric Psychology*, 40(9), 846–855. doi:10.1093/jpepsy/jsv027
- Hullmann, S., Brumley, L., & Schwartz, L. (2015). Medical and psychosocial associates of nonadherence in adolescents with cancer. *Journal of Pediatric Oncology Nursing*, 32(2), 103–113. doi:10.1177/1043454214553707

Ethical Issues for Psychologists in Medical Settings

Gerald P. Koocher¹ and Jeanne S. Hoffman^{2,*}

¹*Department of Psychology, DePaul University, Chicago, IL, USA*

²*US Department of the Army, Washington, DC, USA*

Over the past several decades, the numbers of psychologists practicing in health delivery systems have expanded as have the types of sites and roles of psychologists in these settings. This result follows the progress of research and practice in health psychology, behavioral medicine, and pediatric psychology but also tracks the evolution of the US healthcare system and specialty care settings. Ethical regulation of the practice of psychology began with the first code of ethics published in 1953 and has evolved as the scope of psychology has changed with the most recent revision adopted in 2010 (American Psychological Association [APA], 2010). The inclusion of psychology as a healthcare profession has resulted in new ethical challenges to practitioners. In response APA developed “Guidelines for Psychological Practices in Health Care Delivery Systems” (APA, 2013). These guidelines are aspirational rather than enforceable standards of care and are “intended to facilitate the continued systematic development of the profession and to help ensure a high level of professional practice” (APA, 2013). They also serve to highlight challenges to practitioners in this specific, but varied, specialty.

Working in the Medical Culture

Practicing medical psychology requires intercultural skills. Just as we expected psychologists to become knowledgeable about and sensitive to the culture of their patients, they must also acquire knowledge and sensitivity to the culture of the healthcare system. Medical culture has a different lexicon; requires an expanded knowledge of physical illnesses, their symptoms, and

*The views expressed in this manuscript are those of the authors and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

treatments; has a powerful hierarchy, often with a physician as leader; requires flexibility with tolerance for multitasking, “stat” consultations, and interventions (from the Latin *statum*, meaning immediately); and may not understand or recognize the distinct capabilities, services, or ethical responsibilities of psychologists.

A degree of willingness to adapt and skill working across professional lines are required for any mental health professional planning to work in such settings. One must, for example, acquire a new lexicon of terminology that may seem paradoxical (e.g., a “progressive disease” is one that gets worse, and “positive findings” are a bad sign when discovered during a physical examination). Familiarity with the symptoms, course, and treatment of physical illnesses as well as an understanding of how medical hospitals and outpatient centers work (as distinct from mental hospitals, community mental health centers, or college counseling services) will prove very important.

Mental health practitioners in medical settings also must remain keenly aware of their skills and limitations in the context of healthcare regulations. These include interdisciplinary collaboration in outpatient settings, maintaining competencies, and attending to issues in hospital practice related to confidentiality and the Health Insurance Portability and Accountability Act (HIPAA). Many medical conditions can present in ways that suggest psychopathology, and having a medical degree does not ensure against diagnostic errors. Some physicians seem too willing at times to see physical complaints as psychological, and some mental health practitioners seem all too eager to go along with them. Similarly, some health-care practitioners can become so focused of detecting or fighting a serious illness that the psychological needs of the patient get lost in the process.

Professional Integrity

Typically, mental health professionals working in medical settings will be employed under the supervision of physicians, historically hired in departments of psychiatry or pediatrics, but increasingly in subspecialty practices such as cardiology, endocrinology, gastroenterology, genetics, nephrology, oncology, or organ transplant services. At other times, they may be administratively organized in separate departments labeled behavioral health, medical psychology, or family services in an effort to avoid the stigma some associate with psychiatry and mental illness. They may provide assessment and intervention services to treat psychopathology but may also be involved in addressing medical nonadherence, symptom control (e.g., pain treatment or anxiety about pending medical procedures), screening for qualification as candidates for complex procedures (e.g., organ transplantation or gastric bypass), genetic counseling, or end-of-life care. Wherever they work, psychologists must take care not to surrender their professional integrity or standards (APA, 2013, *Guideline 1*) within this milieu. The *Guidelines* acknowledge that this may be challenging due to “constraints or pressures posed by other professions or systemic factors” (APA, 2013).

Consider the medical practice in which the office medical clerk asks every patient to complete brief depression and anxiety measures, scores them, and hand the scores over to a psychologist who writes a few sentences as an “assessment” of patients they have not personally interviewed. The patients’ insurance is billed for the assessment screening that creates an unnecessary expense with no clear benefit for most patients. Consider the hospital outpatient clinic the administrator routinely overbooks all practitioners to allow for DNK (did not keep) or no-show appointments. On days when every patient shows up, they may be told, “The doctor

is running late,” with no mention of overbooking as the cause. This type of scheduling may not qualify as unreasonable in some circumstances where the practitioner’s time with each patient is brief, but would certainly qualify as unreasonable for psychotherapy appointments. These examples may seem like good business practices aimed at increasing billing volume, but they do not meet the ethical test of holding the best interests of the patient paramount.

One should not assume that other professionals understand the training, services, standards, or ethics of psychologists. We can and should take steps to expand colleagues’ knowledge of their potential contributions within these settings in accord with acceptable practice. Of course this also requires our profession’s understanding the roles of the myriad professionals in complex medical systems. For example, do social workers perform psychotherapy or case management, do speech therapists provide behavioral treatment for feeding disorders, do pediatricians evaluate ADHD, do pharmacists address adherence issues, and who makes final decisions on how professional services are coded and billed?

Competence and Credentialing in Healthcare Delivery

Our ethics require us to practice only within the limits of our training and competence. As psychology has matured as a specialty in medical settings, specific core competencies have evolved. An example of such core competencies was generated by the Interorganizational Work Group on Competencies for Primary Care Practice (McDaniel et al., 2014) and could be considered as a model for core competencies in other medical settings: science, systems, professionalism, interprofessional relationships, application of care, and teaching. At the same time, many of the roles we perform in medical settings have evolved with the rapid pace of change in medical care, requiring continuing learning and creative adaptation by experienced clinicians. Intellectual laziness and conceptual rigidity will lead to rapid failure in medical settings.

Healthcare delivery systems must, by law (42 U.S.C.233(h)(2)), require documented credentialing and privileging of its providers. Credentialing in this context involves “the process of assessing and confirming the qualifications of a licensed or certified health care provider” (Health Resources and Services Administration, 2007). Clinical privileges define the scope of services the individual is deemed qualified to provide within the organization. Staff appointments define the extent of participation in the governing bodies of medical institutions. The type of appointment for psychologists (e.g., full medical staff, allied health professional, consultant) and particular privileges (e.g., admitting, consulting, treating, or conducting research) varies between institutions.

Board certification of physicians has become a well-established standard, often required for their credentialing in medical settings. While the majority of psychologists have not sought board certification, those practicing in healthcare are more likely to have board certification (21.7%) (Robiner, Dixon, Miner, & Hong, 2012). The American Board of Professional Psychology (ABPP), founded in 1949, has become the body predominantly responsible for the granting of board certification with its specialties in clinical psychology, clinical health psychology, clinical child and adolescent psychology, clinical neuropsychology, and rehabilitation psychology the most relevant in healthcare practice. As psychologists increasingly become integrated team members in the evolving healthcare system, and with mounting emphasis on competence and safety within these institutions, the necessity for board certification as a commensurate credential with our colleagues is in the future of psychology.

Confidentiality

Confidentiality stands as the cornerstone of the therapeutic relationship, but the duty of confidentiality becomes nuanced and complex within medical systems. We owe preservation of confidentiality to individual patients, but medical settings include a community of providers that requires a network of confidentiality (Condie, Grossman, Robinson, & Condie, 2014). The psychologist should recognize that within these networks both formal and informal exchanges of information occur. In addition, differing views of confidentiality may be held by the patient, the family, the psychologist, the various other care providers, and also the medical institution. In certain areas such as pediatrics, the primary care provider has an expectation that all information will be shared. Families often have similar expectations even with adult patients.

Limits of confidentiality in medical settings are controlled by HIPAA, state law, or other government regulations that create conflicting demands at times. HIPAA allows providers to share information necessary for treatment and payment operations, but there may be circumstances when patients may rightly request that sensitive information be compartmentalized. For example, suppose that family members agree to screening as possible bone marrow donors for a child and genetic testing reveals that the person who believes he is the child's biological father turns out to be genetically unrelated. This incidental parentage discovery unrelated to the primary question (i.e., Is the putative father a good donor match?) could cause considerable emotional distress, if revealed. Similarly, consulting psychologist will routinely learn of sensitive emotional issues unrelated to immediate medical treatment needs of the patient. In the community of caregivers, some information should be shared on a need-to-know basis.

Electronic health records often make better integrated care possible, but it is important to understand and make appropriate use of role segregation. Once again, need-to-know becomes the key principle, but the psychologist needs to understand who will have access to what information and understand the potential consequences. For example, the patient registration clerk and the proctologist most likely should not have access to sensitive psychotherapy notes. Patients also have a right to know and consent to the sharing of their medical and mental health records. At an institutional level this typically occurs at the time of clinic registration or hospital admission, when the patient signs a standard HIPAA information document. However, many people do not read, understand, or remember the details. Therefore, the psychologist should repeat the necessary HIPAA cautions to patients when we begin to serve them in these settings.

Informed Consent

This process supports the basic ethical principle of autonomy and promotes trust between provider and patient. It also addresses some of the conundrums posed within a multidisciplinary care setting. Informed consent not only provides a patient with the risks, benefits, and alternatives to specific treatments but also delineates with whom a patient's information will be shared and the nature of the community of providers involved. We must remain mindful that consent is a process, rather than a single conversation or signature on a form. We must refresh the patient's understanding as changes that might affect their decisions occur.

The most recent APA Ethics Code (APA, 2010) acknowledges the need to address these complexities when Standards 3.07 "Third-Party Requests for Services" and 3.11 "Psychological

Services Delivered To Or Through Organizations” were modified to include the concept of client definition (Behnke, 2014). When psychologists work with children, families, or couples, this becomes most salient (Behnke, 2014; Koocher & Keith-Spiegel, 2016; Rae, Brunquell, & Sullivan, 2009) and is addressed in Standard 10.2, which mandates that the provider “take reasonable steps at the outset (1) which of the individuals are clients/patients and (2) the relationship the psychologist will have with each person” (APA, 2010).

Consent, assent, permission, and capacity are different concepts that demand parsing.

Consent implies three separate aspects—knowledge, voluntariness, and capacity (both legal and cognitive). The person seeking the consent must provide sufficient information for the person granting consent to understand fully what is being asked of them. It is not necessary to disclose every potential aspect of the situation, only those facts a reasonable person might need to formulate a decision. Voluntariness refers to the absence of coercion, duress, misrepresentation, or undue inducement. People can only give consent for themselves. Capacity refers to legal competence to give consent as well as the cognitive ability. Although all adults are deemed competent to give consent unless they are found to be incompetent, children are assumed incompetent to grant consent (Koocher & Keith-Spiegel, 2016).

The concept of capacity involves weighing the right to autonomy of the patient vs. beneficence and non-maleficence. For persons who are legally incapable of giving informed consent, such as children, adolescents, or adults lacking capacity, psychologists nevertheless must seek assent. This means that they must provide an appropriate explanation of the treatment or assessment, seek the individual’s assent and consider the person’s preferences or best interests, and seek appropriate permission from a legally authorized person, if such substitute consent is permitted or required by law. Children and adolescents are presumed to lack competence to consent, but the provider should determine the minor’s ability to understand information offered about the nature and potential consequences of the pending decision (i.e., the risks and benefits of a specific treatment or action) and invite a clear preference based on the information presented.

The terms competency and capacity are often used interchangeably, but competence is a legal concept determined by a court or defined by law as in the case of minors. Capacity is determined by a qualified psychologist or physician based on the circumstances and jurisdiction. Questions of capacity arise in medical settings most often arise with refusals or discontinuation of treatment. If an articulate child, medical providers, or the parent/guardian disagrees on the necessity of medical treatment, a judicial decision may be required.

Boundaries

Psychologists are trained to maintain boundaries, but working in medical settings, especially acute care settings, will present many unexpected challenges. The challenges might be categorized as boundary crossings versus boundary violations. The former is in service of the therapy and must be a considered action taking into account the general ethical principle of beneficence while a boundary violation results in harm to the patient. The context in which boundary crossings occur must be considered (Zur, 2017). The medical context is unique and may impose varying degrees of beneficent exigency. In other settings a psychologist does not make physical contact with a patient, but what if a patient in the final stages of life requests that the psychologist hold her hand? What if a patient requires oral suctioning to continue the therapeutic interaction? What if the therapist is assisting the patient remain calm during a procedure in which the patient’s body is exposed? Does this violate the patient’s right to dignity or

promote coping? Context, contingency, and patient needs and rights must all be considered in the decision to deviate from standard operating procedures in psychotherapeutic practice.

Challenging Healthcare Contexts

In this brief chapter we cannot address all possible complicating contexts that will challenge psychologists in healthcare setting, but we have chosen a few that illustrate the range of ethical challenges that can arise. These include providing services in military medical settings, care of patients seeking organ transplantation, children with gender dysphoria, and patients with cystic fibrosis (CF). We selected this sampling because of the significant ethical issues that come up in providing care with these patients and setting contexts.

Military medical care provides some thought-provoking examples. A recent report by the Defense Health Board acknowledges such ethical challenges in its introduction (Defense Health Board, 2015). Clinical health psychologists practicing in military medical settings also face conflicts between their professional ethics, the challenges of working in a medical setting, and the requirement of following military policies and orders. Military medical settings range from very large medical institutions such as the Walter Reed National Military Medical Center to small embedded units in combat zones where the psychologist is the sole mental health provider. Depending on the site and role, the clinician may be a civilian employee or military personnel caring for active duty personnel or their dependents. While psychologists are required to avoid most multiple relationships, this is not always possible in military medical settings (Dobmeyer, 2013; Johnson, Ralph, & Johnson, 2005; Koocher & Keith-Spiegel, 2016; Staal & King, 2000). Examples include a psychologist serving on an aircraft carrier who was required to provide a urine sample for random drug testing only to find the only female observer available was one of her patients (Johnson et al., 2005) or a psychologist working in a family medicine clinic who received his medical care from a physician who was also his client (Dobmeyer, 2013).

In the military active duty psychologists are also officers and bound by law and policy “to hold subordinates accountable, to promote order and discipline and to place the mission above the interests of the individual service member” (Kennedy & Moore, 2008). What is a clinician to do when in the course of therapy another officer reveals that he is having an extra-marital affair, an offense under the *Uniform Code of Military Justice* (UCMJ), or a pilot reports that he is taking stimulant medication for ADHD that is prohibited and considered a safety risk? Confidentiality also becomes much more complex issue in a system where commanders are entitled to information about a service member’s medical or psychological condition and how its treatment may effect fitness for duty or deployability and all treatment is documented in a system-wide electronic medical record.

Cultural competency also comes into acute focus, as psychologists in military medical settings work within both the medical culture and the military culture. The latter may seem quite alien to civilian psychologists. The military culture is highly structured, hierarchical, with specific norms of behavior, specific belief systems, and even unique forms of language. The military subscribes to core values that have in common: duty, honor, and country. Families of service members live within this culture, and veterans often subscribe to military values long after their active service ends. Military psychologists understand this culture, but civilians must seek training or mentoring to ethically practice in these settings.

Organ transplantation is another arena in which psychologists provide invaluable services as members of transplant teams by evaluating potential recipients and their support systems,

making recommendations to these teams, providing both clinical interventions and support to recipients and their families, and evaluation of candidates as living tissue or marrow donors. Each of these potential services comes with a myriad of ethical challenges. Confidentiality issues become very complex as information must be shared, especially the results of assessment of psychological or behavioral factors that could adversely affect the success of the transplant. Will the psychological status of the patient compromise success? How likely is the recipient to adhere to the complex post-transplant regimen? Of course, informed consent must be obtained for psychological evaluation or treatment, but can the patient truly refuse? Does consent qualify as voluntary if the alternative is death? Can the patient's support system refuse assessment since this will jeopardize the patient being accepted for transplant? What happens if a screened donor qualifies as a "good match" but has intense ambivalence about donating? How can we balance the ethical principles of beneficence and non-maleficence in transplant decisions?

Conflicting loyalties may lead to ethical dilemmas that force the psychologist to sort through the question of to whom one owes a duty of loyalty and in what hierarchy: the patient, the family, the donor, the medical teams, and the transplant center? Certainly issues of justice are a topic of great debate in transplant medicine. What if the potential recipient has cognitive or emotional impairments that might compromise success? Dual relationships are common. Can the psychologist who provides therapy and support also objectively evaluate the patient? Are other psychologists with the requisite skills and knowledge available to consult? The donor status can trigger other ethical issues. Is this to be a living donor? Are pressures being applied to donate? Is there a conflict between the possible harm to the donor and possible good to the recipient? Can a minor consent to donation of an organ in any event? What if the best donor for a parent is their child? In nonliving donation other issues arise: pressure applied to donor families when time is of the essence in harvesting the organ and psychological impact on families when donors are kept alive by mechanical means to facilitate harvest. Issues can even arise related to how death of the donor is determined: brain death or circulatory death.

Minors With Gender Dysphoria/Transgender Youth

Psychology has a unique and essential contribution to the treatment of this population both because of our knowledge set but also in our awareness of the ethical challenges embedded in this area of practice. While adults with these conditions have a more direct route to medical and surgical interventions, psychological assessment is still required. Children also require psychological evaluation, but ethical challenges become prominent when they present for treatment. The core of these challenges arises from the fact that gender identity and gender expression may change over time (Tishelman et al., 2015) and the percentage of gender diverse children decreases with age due to return to conformity with natal sex, or a determination they are gay, lesbian, or bisexual (APA, 2015). Ethical principles of beneficence and non-maleficence as well as autonomy must be addressed as well as the ethical standards of informed consent, assent, and capacity.

The typical course of treatment guided by psychosocial development principles suggests these differences. Young children expressing gender dysphoria are candidates for exploratory psychotherapy for the child, but often, primarily for parents. Prepubertal children are candidates for psychotherapy, assessment, and consideration of puberty suppression to give time for the child to mature cognitively in order to more fully understand the consequences of their decision. Pubertal children may be candidates for addition of use of cross-sex hormones to the treatment course to begin the transition to another sex. Adults may be prepared to consider

sexual reassignment surgery. Assent becomes a particularly thorny issue with minors as they may not have the capacity to fully understand the course of treatment, irreversible consequences, or psychosocial challenges that they might face. But an additional issue involves the fact rates of autistic spectrum disorders are higher in the population of minors who express gender dysphoria, raising the fundamental question whether these youth with impaired social skills can fully understand the implications of gender modification. The APA guidelines on practice with transgender patients also caution practitioners to remain mindful of the cognitive processes of adolescents that might focus on immediate gratification to the detriment of a considered decision (APA, 2015). Again, we must adhere to the standards of competence and informed consent in order to prevent harm to these youth (non-maleficence) even as we strive to help them optimize adjustment as part of the treatment team.

Cystic Fibrosis

CF provides an excellent example of a life-threatening genetically transmitted (autosomal recessive) disease illness previously associated with childhood death. With new treatments CF has become a chronic disease with mean survival into the sixth decade of life. Successful treatment of CF involves a complex and unrelenting treatment regimen that imposes a significant burden on caretakers and patients alike. A burgeoning body of research on CF has begun to identify factors related to prognosis. As one might expect, adherence is an important factor that itself impacts other variables such as lung function and body weight that, in turn, influence long-term survival (Sawicki & Goss, 2015; Smith, Georgiopoulos, & Quittner, 2016). A recent, and growing, body of literature has found that rates of depression in individuals with CF and their caretakers rank well above comparison samples (Quittner et al., 2014). These higher rates of depression and anxiety link to decreased adherence as well as poorer lung function and lower body weight. As a result, the Cystic Fibrosis Foundation (CFF) Committee on Mental Health has recommended that individuals with CF ages 12 to adulthood as well as caretakers of all children and adolescents with CF be screened annually for depression and anxiety. For all children, adolescents, and adults with CF, storing of such results in their medical records would be expected, although with the same ethical consideration given to all mental health test and screening results. But what of the results of caretaker screenings? Should they be recorded in the caretaker's medical record or CF patient's? This becomes an ethical question that is being currently discussed on the CFF Mental Health Group Listserve and at the most recent CFF Conference. In addition, the prevention of CF via genetic screening and pregnancy termination raises sensitive questions for family members who may carry the gene.

Emerging Challenges

We have simply scratched the surface of the range of issues and contexts that raise ethical challenges for psychologists working in medical settings. Reflecting on the special challenges discussed here, it is clear that most have arisen as medical science has advanced. Inexorably with future advances, new ethical challenges to psychologists will emerge extend beyond psychologists' current knowledge and thereby increase responsibility to acquire working knowledge of these advances. Neuroimaging is one such area as is genetic modification and enhancement technologies. To these and to as yet unimagined technologies, psychologists must apply our basic ethical tenets but also strive to anticipate quandaries that might arise.

Solutions to Ethical Challenges

Just as working in medical settings is a team sport, solutions to ethical dilemmas require a team approach. Collegial consultation is key. Developing familiarity and routine discussions with colleagues across disciplines, consulting with experts including those involved in direct care, and third-party operations will be essential. National and state psychological associations will be a resource, but monitoring regulatory changes by licensing boards will also prove critical. Hospitals are required by Joint Commission to have ethics committees that exist to provide consultation. Perfection is not required, but knowledge of ethical principles, thoughtfulness, and efforts to avoid harm are.

Author Biographies

Gerald P. Koocher, PhD, ABPP, received his doctorate in clinical psychology from the University of Missouri. He is professor of psychology and dean of the College of Science and Health at DePaul University. He previously served as chief psychologist at Boston Children's Hospital and the Judge Baker Children's Center.

Jeanne S. Hoffman, PhD, ABPP, received her doctorate in developmental psychology and clinical respecialization from the University of Hawaii with a fellowship in pediatric psychology from Boston Children's Hospital and Judge Baker Children's Center. She currently is chief of the pediatric psychology clinic at Tripler Army Medical Center, Honolulu, Hawaii.

References

- American Psychological Association. (2010). 2010 Amendments to the 2002 "ethical principles of psychologists and code of conduct". *American Psychologist*, 65, 493. <http://dx.doi.org/10.1037/a0020168>
- American Psychological Association. (2013). Guidelines for psychological practice in health care delivery systems. *American Psychologist*, 69(1), 1–6.
- American Psychological Association. (2015). Guidelines for psychological practice with transgender and gender nonconforming people. *American Psychologist*, 30(9), 832–864.
- Behnke, S. (2014). American Psychological Association. Informed consent and the APA Ethics Code. Washington, DC, USA.
- Condie, L. O., Grossman, L., Robinson, J. D., & Condie, D. B. (2014). Ethics and the law. In C. Hunter, C. L. Hunter, & R. Kessler (Eds.), *Handbook of clinical psychology in health settings* (pp. 99–124). New York, NY: Springer.
- Defense Health Board. (2015, March 3). *Ethical guidelines and practices for U.S. medical professionals*. Retrieved from www.health.mil.
- Dobmeyer, A. C. (2013). Primary care behavioral health: Ethical issues in military settings. *Families, Systems & Health*, 31(1), 60–68.
- Health Resources and Services Administration. (2007). U.S. Department of Health and Human Services, Bethesda, MD, USA.
- Johnson, W. B., Ralph, J., & Johnson, S. J. (2005). Managing multiple roles in embedded environments: The case of aircraft carrier psychology. *Professional Psychology*, 36, 73–81. <http://dx.doi.org/10.1037/0735-7028.36.1.73>
- Kennedy, C. H., & Moore, B. A. (2008). Evolution of military psychology ethics. *Military Psychology*, 20(1), 1–6.
- Koocher, G. P., & Keith-Spiegel, P. C. (2016). *Ethics in psychology and the mental health professions: Professional standards and cases* (4th ed.). New York, NY: Oxford University Press.

- McDaniel, S. H., Grus, C. L., Cubic, B. A., Hunter, C. L., Kearney, L. K., Schuman, C. C., ... Johnson, S. B. (2014). Competencies for psychological practice in primary care. *American Psychologist*, 69, 409–429.
- Quittner, A. L., Goldbeck, L., Abbott, J., Duff, A., Lambrecht, P., Solé, A., ... Barker, D. (2014). Prevalence of depression and anxiety in patients with cystic fibrosis and parent caregivers. *Thorax*, 69, 1090–1097.
- Rae, W. A., Brunquell, D., & Sullivan, J. R. (2009). Ethical and legal issues in pediatric psychology. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (4th Eded. ed., pp. 19–34). New York, NY: The Guilford Press.
- Robiner, W. N., Dixon, K. E., Miner, J. L., & Hong, B. A. (2012). Board certification in psychology: Insights from medicine and hospital psychology. *Journal of Clinical Psychology in Medical Settings*, 19, 30–40.
- Sawicki, G. S., & Goss, C. H. (2015). Tackling the increasing complexity of CF care. *Pediatric Pulmonology*, 50, S74–S79.
- Smith, B. A., Georgiopoulos, A. M., & Quittner, A. L. (2016). Maintaining mental health and function for the long run in cystic fibrosis. *Pediatric Pulmonology*, 51, S71–S78.
- Staal, M. A., & King, R. E. (2000). Managing a multiple relationship environment: The ethics of military psychology. *Professional Psychology: Research and Practice*, 31(6), 698–705.
- Tishelman, A. C., Kaufman, R., Edwards-Leeper, L., Mandel, F. H., Shumer, D. E., & Spack, N. P. (2015). Serving transgender youth: Challenges, dilemmas, and clinical examples. *Professional Psychology: Research and Practice*, 46(1), 37–45.
- Zur, O. (2017). *Multiple relationships in psychotherapy and counseling: Unavoidable, common, and mandatory dual relations in therapy*. New York, NY: Rutledge.

Suggested Reading

- Johnson, W. B., & Koocher, G. P. (Eds.) (2011). *Ethical conundrums, quandaries, and predicaments in mental health practice*. New York, NY: Oxford University Press.
- Koocher, G. P., & Keith-Spiegel, P. C. (2016). *Ethics in psychology and the mental health professions: Professional standards and cases* (4th ed.). New York, NY: Oxford University Press.
- Linton, J. C. (2010). 2020 foresight: Practicing ethically while doing things that don't yet exist. *Journal of Clinical Psychology in Medical Settings*, 17, 278–284.

History of Clinical Health Psychology

Rachel Postupack and Ronald H. Rozensky

*Department of Clinical and Health Psychology, College of Public Health and Health Professions,
University of Florida, Gainesville, FL, USA*

Disease is not the accident of the individual, nor even of the generation, but of life itself. In some form, and to some degree or other, it is one of the permanent conditions of life.

Henry David Thoreau

Health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity (World Health Organization, 1948). This formulation of health, adopted globally immediately after World War II, predates Engel's (1977) "new" "biopsychosocial" model of healthcare by decades and, as pointed out by Rozensky (2015), is as "new" as 190 CE when Galen postulated the integration of mind and body in medicine during the Roman Empire. Every textbook and article that discusses the history of clinical health psychology (Baum, Perry, & Tarbell, 2004; Belar, Deardorff, & Kelly, 1987; Friedman & Adler, 2007; Stone, Cohen, & Adler, 1979; Sweet, Rozensky, & Tavian, 1991) notes that "psychology has long been in the forefront of the scientific inquiry into the understanding of health" (Resnick & Rozensky, 1996, p. 1). While psychology clearly is a scientifically based discipline that both carries out research concerning health, disease, and treatment and provides direct clinical services to those who seek help with disease prevention or treatment for managing a wide range of medical diagnoses and problems, it was not until 2001 that "the American Psychological Association (APA) amended its mission statement to include the term 'health.'" The American Psychological Association (APA) stated that psychology's focus is to "advance as a science and a profession, and as a means to promoting health and human welfare" (Rozensky, Johnson, Goodheart, & Hammond, 2004, p. *xix*).

First recognized formally as specialty in 1997 by the APA (2015a), "Clinical Health Psychology applies scientific knowledge of the interrelationships among behavioral, emotional, cognitive, social and biological components in health and disease to: the promotion and maintenance of health; the prevention, treatment and rehabilitation of illness and disability; and the improvement of the health care system. The distinct focus of Clinical Health Psychology

(also referred to as behavioral medicine, medical psychology and psychosomatic medicine) is at the juncture of physical and emotional illness, understanding and treating the overlapping challenges.” This definition was built, in turn, on Matarazzo’s (1980) definition as adopted by the APA Division of Health Psychology: “the aggregate of the specific educational, scientific, and professional contributions of the discipline of psychology to the promotion and maintenance of health, the prevention and treatment of illness, and the identification of etiologic and diagnostic correlated of health, illness, and related dysfunction” (p. 815).

Given these definitions, Belar (2008, p. 230) described both the venues in which health psychology services take place and the range of clinical issues addressed:

Clinical health psychology services may be integrated with primary care, urgent care, tertiary care, and dental care, and occur in clinic, hospital-based, nursing care, rehabilitation, work site, and hospice settings.

Belar listed the nine problem areas addressed by clinical health psychologists:

- 1 Psychological factors secondary to disease, injury, or disability—which includes both normal adjustment reactions to posttraumatic stress disorders and all psychological diagnoses in the patient or family members.
- 2 Somatic presentations of psychological issues, which can include such issues as chest pain with panic attack.
- 3 Psychophysiological problems (e.g., pain, headache).
- 4 Physical symptoms or medical conditions responsive to psychological and behavioral health interventions (e.g., urinary and fecal incontinence, anticipatory nausea, asthma).
- 5 Somatic complications associated with behavioral issues such as poor adherence to health-care regimens.
- 6 Psychological presentation of organic depression with hypothyroidism.
- 7 Psychological and behavioral aspects secondary to stressful medical procedures such as self-infections, dental procedures, and burn debridement.
- 8 Behavioral risk factors for disease, injury, or disability related to problematic health behaviors such as smoking, overeating, low exercise, and risk-taking.
- 9 Problems encountered by healthcare providers and healthcare systems such as provider–patient relationship issues, staff “burnout,” assuring universal precautions, healthcare team behavior and cooperation, clinical pathway development, and quality assurance and program evaluation activities.

Psychology and Health

Paralleling the history of health psychology is the development of the US federal government’s *Healthy People* initiative (Department of Health and Human Services [US], 1979) that establishes goals for improving the quality of the nation’s health with objectives focused on health promotion and disease prevention (Healthy People 2020, <http://www.healthypeople.gov/>). Rozensky (2012a) stated that along with psychology’s traditions of efficacy, effectiveness, and community-based research and treatment, the profession has engaged in this population-based approach to the scientific study and treatment of the human condition. Further, Rozensky (2012a) reviewed societal and healthcare trends impacting health service psychology including competency-based education, interprofessionalism, evidence-based care, and

team-based, patient-centered care and changes in population demographics including a more diverse population that is aging with more chronic illness. As one of the recognized specialties under the broader umbrella of health service psychology, clinical health psychology has been a leader, throughout its history, in anticipating the various scenarios, or interactions of these trends, in preparing each new generation of health service psychologists to successfully function in an evolving research and healthcare environment.

With definitions in hand, trends in mind, and current clinical activities and services described, the history of clinical health psychology will be presented from the perspective of how the profession itself has advanced. This will include the scientific and clinical changes in the field as reflected in the evolution of philosophies and organizations that have promulgated a biopsychosocial understanding of health and illness.

Defining Health Psychology and the Formation of Professional Organizations

The evolution of clinical health psychology in becoming a recognized specialty is rooted in the development of definitions that include the interaction of cognitive, behavioral, emotional, and psychological processes and physical health and the formation of professional organizations that focus on science and practice in those domains. The ancient Greeks provided the philosophical basis for modern Western medicine by shifting the focus from supernatural causes to recognition of the importance of actual bodily factors in health. They theorized that illness was caused by an imbalance of the four bodily humors: blood, phlegm, choler (yellow bile), and melancholy (black bile). Health could be restored then by bringing balance to these fluids (Friedman & Adler, 2007). Galen, a Greek physician in the Roman Empire, posited that the dominant humor in an individual's balance determined temperament and vulnerability to illness (Maher & Maher, 1994). This early integration of mind and body was later de-emphasized in favor of focusing on primarily bodily or physical processes in the biomedical model.

In the early twentieth century, the split between mind and body was reflected in the distinction of psychiatry as a medical specialization focused on the influence of the mind to explain symptoms for which there was no biological basis. Interest in the contribution of mental disturbance to disease became the domain of the emerging field of psychosomatic medicine (Dunbar, 1943). Flanders Dunbar began the journal of *Psychosomatic Medicine* in 1938–1939, which is the official journal of the American Psychosomatic Society founded in 1942. Today their mission is to “promote and advance the scientific understanding and multidisciplinary integration of biological, psychological, behavioral, and social factors in human health and disease, and to foster the dissemination and application of this understanding in education and health care” (www.psychosomatic.org).

Moving into the 1960s and 1970s, growing insight into biological mechanisms prompted a philosophical shift in psychosomatic medicine to consider the bidirectional interactions between physiological and emotional states. George F. Solomon and Moos (1964) introduced the term *psychoneuroimmunology* (PNI) to refer to the theoretical connections between emotions, immunity, and disease. Research on stress and disease expanded and was based on the “fight or flight response” (Cannon, 1932) and understanding of physiological reactions to stress (Selye, 1956). Engel's (1977) descriptions of the biopsychosocial model contributed to the ongoing integration of the mind and body in contemporary healthcare science and practice. Herbert Benson demonstrated the utility of meditation or relaxation to counteract both acute and chronic stress while impacting health status in conditions like hypertension

(Benson, 1983). These discoveries among others provided an evidence base for the introduction of behavioral treatments into medical settings. Emphasis on the integration of mind and body became known as *behavioral medicine* (Birk, 1973). Interdisciplinary from its inception, contributors to the science and practice of behavioral medicine included professionals from a range of professions including physicians, psychologists, bio-behaviorists, and others. The Society of Behavioral Medicine and *Journal of Behavioral Medicine* were founded in 1978 to promote a “field concerned with the development of behavioral science knowledge and techniques relevant to the understanding of physical health and illness and the application of this knowledge and these techniques to prevention, diagnosis, treatment, and rehabilitation” (Schwartz & Weiss, 1978, p. 7).

In the 1980s *behavioral health* was distinguished from *behavioral medicine* as “an interdisciplinary field dedicated to promoting a philosophy of health that stresses *individual responsibility* in the application of behavioral and biomedical science knowledge and techniques to the *maintenance* of health and the *prevention* of illness and dysfunction by a variety of self-initiated individual or shared activities” (Matarazzo, 1980). It is within this focus on the individual and a personal ability to affect change that impacts health that psychologists found a professional niche to more specifically define their scientific and clinical competencies to encourage health promotion as well as address challenges associated with illness and its treatment. The field of *health psychology* was defined as promoting the integration of psychological knowledge and biomedical information as it applies to health promotion, prevention, diagnosis, treatment, and rehabilitation (APA, 2015b; Matarazzo, 1980). To assist in the development of a cohesive field of specialization then, George Stone established the *Journal of Health Psychology* in 1982. Then in 1994, the *Journal of Clinical Psychology in Medical Settings* (JCPMS) was founded by Ronald Rozensky to further the role of health service psychologists as scientists and practitioners in medical settings as they addressed diseases and medical problems (Rozensky, 2006).

Established in 1892 the APA advances the creation, communication, and application of psychological knowledge on the national level (APA, 2015c). Today APA is home to 54 divisions, which are special interest groups that represent topic areas germane to the range of clinical, scientific, educational, and public interest foci of psychologists. Movement toward the foundation of the division of health psychology began in 1973 when APA’s Board of Scientific Affairs directed that health research should be reviewed and disseminated at scientific meetings. This decision was influenced by William Schofield’s (1969) paper describing psychologists’ roles in health service delivery. A section of interest was added to Division 18, Psychologists in Public Service, in 1975 specifically to increase the application of psychological principles to health research and patient care as well as disseminate the results of this work. Steven Weiss and Joseph Matarazzo advocated for the foundation of an APA division devoted to health psychology. Their efforts were successful when Division 38 Health Psychology was established in 1978. The division continues its mission to promote and disseminate health-related research through *The Health Psychologist* newsletter begun in 1979 and the *Health Psychology* journal that published its first issue 1980–1981 (Wallston, 1997). In August 2015 the Division of Health Psychology voted to change its name the Society for Health Psychology. It continues to provide an intellectual home to health psychologists and other professionals devoted to the intersection of psychological processes and physical health.

One venue that illustrates the robust growth of psychologists engaged in health and health-care research, education, and clinical practice is number of psychologists in academic health centers and hospitals. Noting that environment as “a true healthcare home” for psychologists (p. 353), Rozensky (2012b) reviewed a series of published surveys from the early 1950s through the first decade of the twenty-first century carried out by such notables as Matarazzo

and Mensh. The number of psychologist in medical school was less five per school in the 1950s to 20.7 per school in the late 1970s with at least 3,894 psychologists identified in academic medical settings at the end of the twentieth century (Williams & Wedding, 1999). Rozensky noted that the most recent data finds more than half (54.5%) of psychologists work in institutional settings with 12.4% in hospitals and 4.6% in medical school alone (APA, 2009).

In 1991, the American Board of Professional Psychology (ABPP) formally recognized clinical health psychology as a specialty in professional psychology and began granting board certification in the field based on peer evaluation (Belar & Jeffrey, 1995). In 1997 the APA (1997) archived a formal definition of the specialty of clinical health psychology, and it remains a recognized specialty to date. Today, contemporary clinical health psychologists are seen as working interprofessionally in clinical practice. They learn side by side with other healthcare professionals during their education and training in the context of the changes in healthcare both predating and promulgated by the implementation of the *Patient Protection and Affordable Care Act* (Public Law No: 111–148, 2010, March 23). As part of the evolution of health psychology's place in the healthcare system, the growing acceptance of the use of Health and Behavior (billing) Codes reflects both the system's integration of mind and body and the integration of psychologists on the healthcare team. Allowing clinical health psychologists to directly bill these Health and Behavior Codes for treating and helping patients manage specific medical problems reflects, financially, the recognition of both psychologists' healthcare competencies and systemic integration (Kessler, 2008) as not just mental health providers but members of the integrated healthcare team. Along with their clinical services and educational activities, health psychologists also participate in team science to ensure research integrates mind and body and ensures interprofessionalism as part of science (Rozensky, 2014).

Education and Training

Clinical health psychologists conduct research and practice at the intersection of physical and mental health in a variety of settings addressing diversified patient populations and problems. The formulation of education and training guidelines has been an essential component in the development and maintenance of professional competencies for both current and future psychologists (Fouad et al., 2009; Health Service Psychology Education Collaborative, 2013; Rodolfa et al., 2005).

In 1983 the Arden House National Working Conference on Education and Training established the first set of education and training guidelines for the field of clinical health psychology (Stone, 1983). The conference recommended that training at the doctoral level remain “broad and general” to develop an integration of theory, research, and practice based on knowledge and understanding of the biopsychosocial model. Predoctoral clinical internships completed in settings where both psychological and physical healthcare services take place were encouraged by the Arden House participants as a part of each student's professional development.

Competencies

In general, doctoral programs in health service psychology have maintained their commitment to the integration of science- and evidence-based practice (Belar & Perry, 1992) where students acquire foundations in scientific psychology, research, ethics, diversity, theory, and clinical practice (Kaslow, Graves, & Smith, 2012) as core competencies in their broad and general education and training. In that manner, curricula for programs with a specific major area of study in clinical health psychology include knowledge of biological, affective, cognitive, social,

and cultural bases of health and illness. Additional education may be provided on special topics such as pathophysiology, psychoneuroimmunology, and pharmacology (Belar, 2008). Ideally training is conducted in clinical settings conducive to the development of competencies to conduct health-related clinical assessments and interventions, health-oriented research, and the competencies to function as a part of healthcare delivery system including consultation services and engagement in interprofessional, team-based collaboration (Belar, 2008; France et al., 2008). It was recommended that accreditation *for* health psychology training programs begin at the postdoctoral level (France et al., 2008), but it is clear that many accredited doctoral programs in health service psychology offer major areas of study in clinical health psychology.

France et al. (2008) provided a competency model for clinical health psychology that details knowledge and applied skills in assessment, intervention, consultation, research, training in supervision, and management administration. Kaslow, Dunn, and Smith (2008) detail the core foundational and functional competencies for health service psychologists practicing in academic health centers, while Kerns, Berry, Franstve, and Linton (2009) extend the description of competencies to issues related to developing lifelong competencies in clinical health psychology. Across competency models, the importance of ensuring clinical health psychologists possess the interprofessional competencies to function successfully within a healthcare team (Fouad & Grus, 2014) and understand the characteristics of a truly competent team (Interprofessional Education Collaborative, 2011) is key to the ongoing evolution of the specialty of clinical health psychology.

Specialization

A specialty is defined as an area of psychological practice that requires advanced knowledge and skills acquired through an organized sequence of education and training (Commission for the Recognition of Specialties and Proficiencies in Professional Psychology, 2015; CRSPPP). Specialization allows professionals to focus on content that they must learn, refresh, and maintain to retain competence in a field as it continues to develop (Kaslow et al., 2012; Rozensky & Kaslow, 2012). The Commission for the Recognition of Specialties and Proficiencies in Professional Psychology (CRSPPP) establishes policies and procedures that are used by APA to review and recognize health service specialties with clinical health psychology being one of those recognized specialties. To identify individual specialists, individuals competent to deliver quality health psychology services, the ABPP (www.abpp.org) offers board certification for practitioners at the postdoctoral level who pass a peer-review, competency-based exam. Achieving board certification is particularly helpful for clinical health psychologists who work in integrated medical settings because board certification is considered the standard of practice for our colleagues in medicine and related health professions (Rozensky & Kaslow, 2012).

Clinical health psychologists face the ongoing challenge of learning and maintaining a current, relevant, evidence-based perspective to meet the ethical requirements of competent practice within their specialty. To help students and professionals describe their level of education and training in clinical health psychology, the Council of Clinical Health Psychology Training Programs (CCHPTP) developed the Clinical Health Psychology Specialty Taxonomy (<http://cospp.org/guidelines>) based on the APA policy designed to ensure consistent descriptions of the levels of education and training within specialties across the sequence of learning from graduate school, to internship, and through post-licensure, lifelong learning (Rozensky, Grus, Nutt, Carlson, Eisman, & Nelson, 2015). That CCHPTP taxonomy lists the expectation for a major area of study in clinical health psychology at each stage of training. Recognizing that each year there is a continuing influx of new scientific and clinical information and that there is a decreasing half-life of professional knowledge, each psychologist must establish a

commitment to lifelong learning on the part of the competencies required of being a specialist (Wise et al., 2010). Incorporating the lifelong learning process into the broader context of the competency movement involves the evaluation of professional skills, practices, and outcomes (Neimeyer & Taylor, 2011). In addition to formal structures for continuing education and competency-based assessments, a self-study/self-assessment can be used to develop learning plans for specialization throughout a professional's career (Belar et al., 2001). Clinical health psychology has embraced a competency-based model of education and training from the early days of the Arden House Conference through professional psychology's evolution to a "culture of competence" (Roberts, Borden, Christiansen, & Lopez, 2005, p. 359).

Clinical Health Psychology, the 2010s, and Beyond

Each step in the history of clinical health psychology has been motivated by the spirit of that time—spirit based on politics within and external to the profession, scientific developments in psychology and medicine, technological evolution, vicissitudes in contemporary healthcare and healthcare financing, and changes in educational philosophy. The future of clinical health psychology will be based on contemporary *zeitgeist* composed of those same motivating forces today and tomorrow.

Contemporary clinical health psychology exists within the broader context of healthcare reform as detailed in the *Patient Protection and Affordable Care Act* (Public Law No: 111-148, 2010, March 23) and its ongoing implementation. That law recognizes the importance of evidence-based healthcare, quality care including credentialing and specialization, team based, and interprofessionalism in both education and practice (Rozenky, 2011, 2013). And clearly, education and training in all branches of health service psychology, including clinical health psychology, must address these issues in its curricula and practical training (Rozenky, 2014).

In order to understand where clinical health psychologists will be employed in the future, a clear national workforce analysis philosophy must be operationalized, and data collected and studied in order to inform educators and practitioners alike as to societal need and employment opportunities (Rozenky, Grus, Belar, Nelson, & Kohout, 2007). Questions like how many psychologists will be needed in primary care, in cardiac care, in rehabilitation psychology, in psycho-oncology, etc. will help the field have a picture of the future and provide direction to students and early career psychologists planning their education and practice focus.

Many scholars have described the history and successful evolution of the specialty of clinical health psychology and highlighted the various individuals and organizations that have helped define the education, training, science, and practice of clinical health psychologists. This history reflects a robust science whose application is key to helping those who wish to learn to prevent disease, improve their health, or seek to manage their illness or their reaction to that illness. Clinical health psychologists clearly are dedicated to improving the human condition, and each recognizes, in their day-to-day work, what Shakespeare said some 400 years ago, "What wound does not heal but by degree?"

Author Biographies

Rachel Postupack, MS, completed her graduate education in the Department of Clinical and Health Psychology at the University of Florida, where she is currently completing her predoctoral internship. She is focusing her clinical experiences on providing services to

underserved medical populations. Her research has focused on pain, distress, cytokines, coping, and quality of life for patients with cancer.

Ronald H. Rozensky, PhD, ABPP, is a professor in the Department of Clinical and Health Psychology in the College of Public Health and Health Professions at the University of Florida where he served as department chair and associate dean for International Programs. He is the founding editor of the *Journal of Clinical Psychology in Medical Settings* and board certified in both clinical and clinical health psychology.

References

- American Psychological Association. (1997, August). *Archival description of clinical health psychology as a specialty in professional psychology*. Minutes of the meeting of the American Psychological Association Council of Representatives.
- American Psychological Association. (2009). *2008 APA survey of psychology health service providers*. Retrieved from <http://www.apa.org/workforce/publications/08-hsp/index.aspx>.
- American Psychological Association. (2015a). *Clinical health psychology*. Retrieved from <http://www.apa.org/ed/graduate/specialize/health.aspx>
- American Psychological Association. (2015b). Our mission. *About Division*, 38.
- American Psychological Association. (2015c). *About APA*. Retrieved from <http://www.apa.org/about/index.aspx>
- Baum, A., Perry, N. W., & Tarbell, S. (2004). The development of psychology as a health science. In R. G. Frank, J. L. Wallander, & A. Baum (Eds.), *Handbook of clinical health psychology* (pp. 9–28). Washington, DC: American Psychological Association.
- Belar, C. D. (2008). Clinical health psychology: A health care specialty in professional psychology. *Professional Psychology: Research and Practice*, 39, 229–233.
- Belar, C. D., Brown, R. A., Hersch, L. E., Hornyak, L. M., Rozensky, R. H., Sheridan, E. P., ... Reed, G. W. (2001). Self-assessment in clinical health psychology: A model for ethical expansion of practice. *Professional Psychology: Research and Practice*, 32, 135–141.
- Belar, C. D., Deardorff, W. W., & Kelly, K. E. (1987). *The practice of clinical health psychology*. Elmsford, NY: Pergamon Press.
- Belar, C. D., & Jeffrey, T. B. (1995). Board certification in health psychology. *Journal of Clinical Psychology*, 2, 129–132.
- Belar, C. D., & Perry, N. W. (1992). The National Conference on scientist-practitioner education and training for the professional practice of psychology. *American Psychologist*, 47, 71–75.
- Benson, H. (1983). The relaxation response: Its subjective and objective historical precedents and physiology. *Trends in Neurosciences*, 6, 281–284.
- Birk, L. (1973). *Biofeedback: Behavioral medicine*. New York, NY: Grune & Stratton.
- Cannon, W. B. (1932). *The wisdom of the body*. New York, NY: Norton.
- Commission for the Recognition of Specialties and Proficiencies in Professional Psychology. (2015). Retrieved from <http://www.apa.org/ed/graduate/specialize/index.aspx>.
- Department of Health and Human Services (US). (1979). *Healthy people: A report of the Surgeon General*. Washington, DC: Author.
- Dunbar, F. (1943). *Psychosomatic diagnosis*. New York, NY: Hoeber.
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196, 129–136.
- Fouad, N. A., & Grus, C. L. (2014). Competency-based education and training in professional psychology. In W. B. Johnson & N. J. Kaslow (Eds.), *The Oxford handbook of education and training in professional psychology* (pp. 105–119). New York, NY: Oxford University Press.
- Fouad, N. A., Grus, C. L., Hatcher, R. L., Kaslow, N. J., Hutchings, P. S., Madison, M. B., ... Crossman, R. E. (2009). Competency benchmarks, a model for understanding and measuring competency in

- professional psychology across training levels. *Training and Education in Professional Psychology*, 3, S5–S26.
- France, C. R., Masters, K. S., Belar, C. D., Kerns, R. D., Klonoff, E. A., Larkin, K. T., ... Thorn, B. E. (2008). Application of the competency model to clinical health psychology. *Professional Psychology: Research and Practice*, 39(6), 573.
- Friedman, H. S., & Adler, N. E. (2007). The history and background of health psychology. In H. S. Friedman & R. Cohen Silver (Eds.), *Health psychology*. New York, NY: Oxford University Press.
- Health Service Psychology Education Collaborative. (2013). Professional psychology in health care services: A blueprint for education and training. *American Psychologist*, 68, 411–426.
- Interprofessional Education Collaborative. (2011, February 16 and 17). *Proceedings from team-based competencies: Building a shared foundation for education and clinical practice*. Washington, DC: Author.
- Kaslow, N. J., Dunn, S. E., & Smith, C. O. (2008). Competencies for psychologists in academic health centers. *Journal of Clinical Psychology in Medical Settings*, 15, 18–27.
- Kaslow, N. J., Graves, C. C., & Smith, C. O. (2012). Specialization in psychology and health care reform. *Journal of Clinical Psychology in Medical Settings*, 19(1), 12–21.
- Kerns, R. D., Berry, S., Franstve, L. M. E., & Linton, J. C. (2009). Life-long competency development in clinical health psychology. *Training and Education in Professional Psychology*, 3, 212–217.
- Kessler, R. (2008). Integration of care is about money too: The Health and Behavior Codes as an element of a new financial paradigm. *Families, Systems & Health*, 26, 207–216.
- Maher, B. A., & Maher, W. B. (1994). Personality and psychopathology: A historical perspective. *Journal of Abnormal Psychology*, 103(1), 72–77.
- Matarazzo, J. D. (1980). Behavioral health and behavioral medicine. *American Psychologist*, 35, 807–817.
- Neimeyer, G. J., & Taylor, J. M. (2011). Continuing education in psychology. In J. C. Norcross, G. R. Vandeboos, & D. K. Freedheim (Eds.), *History of psychotherapy: Continuity and change*. Washington, DC: American Psychological Association.
- Public Law No: 111–148, 111th Congress: Patient Protection and Affordable Care Act. (2010). *124 STAT*, p. 119. Retrieved from www.gpo.gov/fdsys/pkg/PLAW-111publ148/pdf/PLAW-111publ148.pdf.
- Resnick, R. J., & Rozensky, R. H. (Eds.) (1996). *Health psychology through the life span: Practice and research opportunities*. Washington, DC: American Psychological Association.
- Roberts, M. C., Borden, K. A., Christiansen, M. D., & Lopez, S. J. (2005). Fostering a culture shift: Assessment of competence in the education and careers of professional psychologists. *Professional Psychology: Research and Practice*, 36, 355–361.
- Rodolfa, E., Bent, R., Eisman, E., Nelson, P., Rehm, L., & Ritchie, P. (2005). A cube model for competency development: Implications for psychology educators and regulators. *Professional Psychology: Research and Practice*, 36(4), 347.
- Rozensky, R. H. (2006). Clinical psychology in medical settings: Celebrating our past, enjoying the present, building our future. *Journal of Clinical Psychology in Medical Settings*, 13(4), 343–352.
- Rozensky, R. H. (2011). The institution of the institutional practice of psychology: Health care reform and psychology's future workforce. *American Psychologist*, 66, 794–808.
- Rozensky, R. H. (2012a). Health care reform: Preparing the psychology workforce. *Journal of Clinical Psychology in Medical Settings*, 19, 5–11.
- Rozensky, R. H. (2012b). Psychology in academic health centers: A true healthcare home. *Journal of Clinical Psychology in Medical Settings*, 19, 353–363.
- Rozensky, R. H. (2013). Quality education in professional psychology: Flowers blooming, Flexner, and the future. *American Psychologist*, 68, 701–716.
- Rozensky, R. H. (2014). Implications of the Affordable Care Act for education and training in professional psychology. *Training and Education in Professional Psychology*, 8, 1–12.
- Rozensky, R. H. (2015). The biopsychosocial model in health service psychology: Has there really ever been another approach. *Psycritiques*, 60, article 3.

- Rozensky, R. H., Grus, C. L., Belar, C. D., Nelson, P. D., & Kohout, J. L. (2007). Using workforce analysis to answer questions related to the internship imbalance and career pipeline in professional psychology. *Training and Education in Professional Psychology, 1*, 238–248.
- Rozensky, R. H., Grus, C. L., Nutt, R. L., Carlson, C. I., Eisman, E. J., & Nelson, P. D. (2015). A taxonomy for education and training in professional psychology health service specialties. *American Psychologist, 70*, 21–32.
- Rozensky, R. H., Johnson, N., Goodheart, C., & Hammond, R. (Eds.) (2004). *Psychology builds a healthy world*. Washington, DC: American Psychological Association.
- Rozensky, R. H., & Kaslow, N. J. (2012). Specialization and lifelong learning. In G. J. Neimeyer & J. M. Taylor (Eds.), *Continuing education: Types, roles, and societal impacts* (pp. 345–358). Hauppauge, NY: Nova Publishers.
- Schofield, W. (1969). The role of psychology in the delivery of health services. *American Psychologist, 24*(6), 565.
- Schwartz, G. E., & Weiss, S. M. (1978). Yale conference on behavioral medicine: A proposed definition and statement of goals. *Journal of Behavioral Medicine, 1*(1), 3–12.
- Selye, H. (1956). *The stress of life*. New York, NY: McGraw-Hill.
- Solomon, G. F., & Moos, R. H. (1964). Emotions, immunity, and disease: A speculative theoretical integration. *Archives of General Psychiatry, 11*(6), 657–674.
- Stone, G. C. (1983). National working conference on education and training in health psychology. *Health Psychology, 2*(5), 1–153.
- Stone, G. C., Cohen, F., & Adler, N. E. (Eds.) (1979). *Health psychology*. San Francisco, CA: Jossey-Bass.
- Sweet, J. J., Rozensky, R. H., & Tovian, S. M. (Eds.) (1991). *Handbook of clinical psychology in medical settings*. New York, NY: Plenum.
- Wallston, K. A. (1997). A history of Division 38 (Health Psychology): Healthy, wealthy, and Weiss. In D. A. Dewsbury (Ed.), *Unification through division: Histories of the divisions of the American Psychological Association* (Vol. 2, pp. 239–267). Washington, DC: American Psychological Association.
- Williams, S., & Wedding, D. (1999). Employment characteristics and salaries of psychologists in United States medical schools: Past and current trends. *Journal of Clinical Psychology in Medical Settings, 6*, 221–228.
- Wise, E. H., Sturm, C. A., Nutt, R. L., Rodolfä, E., Schaffer, J. B., & Webb, C. (2010). Life-long learning for psychologists: Current status and a vision for the future. *Professional Psychology: Research and Practice, 41*(4), 288.
- World Health Organization. (1948). Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19–22 June 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948.

Suggested Reading

- Belar, C. D. (2008). Clinical health psychology: A health care specialty in professional psychology. *Professional Psychology: Research and Practice, 39*, 229–233.
- Belar, C. D., Brown, R. A., Hersch, L. E., Hornyak, L. M., Rozensky, R. H., Sheridan, E. P., ... Reed, G. W. (2001). Self-assessment in clinical health psychology: A model for ethical expansion of practice. *Professional Psychology: Research and Practice, 32*, 135–141.
- Matarazzo, J. D. (1980). Behavioral health and behavioral medicine. *American Psychologist, 35*, 807–817.
- Rozensky, R. H. (2014). Implications of the Affordable Care Act for education and training in professional psychology. *Training and Education in Professional Psychology, 8*, 1–12.
- Stone, G. C. (1983). National working conference on education and training in health psychology. *Health Psychology, 2*(5), 1–153.

Older Adults and Perspectives for Researchers and Clinicians Working in Health Psychology and Behavioral Medicine

Louise Sharpe

School of Psychology, Faculty of Science, The University of Sydney, Sydney, NSW, Australia

Background

It is well established that there is an aging trend in the population among most Western nations and that this trend is also being seen increasingly in developing nations (e.g., Areean, Hegel, Vannoy, Fan & Unutzer, 2008; Cuijpers, van Straten, & Smit, 2006). As the population ages, people continue to develop chronic illnesses, such as cardiovascular conditions, respiratory problems, diabetes, pain, and cancer. However, the way in which these conditions are medically managed has improved, such that the life expectancy of patients diagnosed with these illnesses is increasing. The consequence of improved medical care for chronic illness is that older adults are still living for longer with the burden of chronic physical illnesses than they previously have. Chronic physical illness impacts on a person's ability to engage in important activities, and as a result most chronic physical illnesses have been shown to be associated with poorer quality of life. Indeed, the majority of the health psychology or behavioral medicine research has focused on individual illnesses (such as pain or diabetes or cancer) in trying to understand the impact of experience chronic illness on people's life. However, the major risk factors for disease are common across conditions. For example, smoking, physical inactivity, and obesity confer risk of cardiovascular disease, stroke, respiratory disease, cancer, and diabetes. All of these illnesses are also more common in older adults. Hence, there is an increasing problem that has been labeled "multi-morbidity" where people are living not with a single medical condition, but with a combination of multiple physical health problems. For example, in a recent population-based study in the United Kingdom, one-quarter of people

attending primary care settings had multi-morbidity, and the risk for multi-morbidity is strongly associated with age. That is, only 7% of those under 45 years of age experienced multi-morbidity, whereas over half of all elderly participants (51% of those over 65) had multi-morbidity (Mujica-Mota et al., 2015). The increase in prevalence of multi-morbidity notes an important change in the management of older adults in health psychology or behavioral medicine. As previously mentioned, health psychology has followed the lead of medical education and research, whereby studies are funded to examine the relationships between different diseases and psychological constructs. However, if the majority of older adults experience multi-morbidity rather than having a single illness, this poses a significant challenge to the way in which we study and treat older adults with multi-morbidity.

Consequences of Multi-morbidity

Multi-morbidity in older adults is associated with a number of consequences. Firstly, people who have more than one physical condition are often prescribed medications separately for each of their conditions. Therefore, older adults often have a complex medical regimen to follow, involving a range of medications that frequently have contraindications and produce side effects. Secondly, many chronic physical illnesses are accompanied by symptoms that can be difficult to manage, such as pain or fatigue. These symptoms are often secondary to the underlying pathologies (e.g., neuropathies in diabetes or angina in cardiovascular disease), but sometimes are seen as more constraining to the individual. As a result of the illness and/or the symptoms or treatments of that illness, individuals typically will have an increase in disability associated with multi-morbidity, which can lead to increased social isolation and a general reduction in physical activity. In some cases, disability can become sufficiently incapacitating to require long-term formal or informal care. These consequences compound to increase the risk of the development of psychological disorders. While there is no good evidence to suggest that older adults have higher rates of depression and anxiety overall (indeed anxiety disorders appear to be lower among older people), multi-morbidity does increase the risk of depressive disorders (Read, Sharpe, Modini, & Dear, 2017). Further, as patients become more disabled and frail, it is increasingly common for them to develop a fear of falling.

Adherence to Medication

Adherence to medication is a major problem across all age ranges and conditions, with a large proportion of people not taking their medications as prescribed. Nonadherence to medication most commonly refers to patients either not taking medications that are prescribed or not taking them as prescribed, including taking larger doses of medication than recommended. There are a number of reasons why older adults might be particularly at risk for nonadherence, including more complex medication regimens to follow, mild cognitive impairment that is common among older people, and the fact that prescribed polypharmacy can give rise to more unwanted side effects and interactions than taking a single medication. Indeed, a qualitative study of nonadherence among the elderly determined that the barriers toward adherence included concern about taking too much medication, a lack of information about the risks and benefits of medicines, a desire to share decision making in the medical setting that is not fulfilled, and the difficulties of trying to coordinate different treatments prescribed by different providers (Haverhals et al., 2011). There have been attempts to use both simple and complex

interventions to improve adherence specifically in older adults (Higgins & Regan, 2004) and among older adults requiring polypharmacy in particular (Cooper et al., 2015). While both identified small, statistically significant effects on improving adherence, *per se*, there was little evidence in either review that improving adherence influenced clinically significant outcomes (e.g., hospital admissions), and both noted that the quality of individual studies was poor. Few interventions were noted to be theoretically derived, and as such, this would be an important area of future research.

Management of Pain and Fatigue Among Older People

Pain and fatigue are the two of the most common symptoms of illness and both associated with poorer quality of life and increased depression. While the relationship between poorer psychological outcomes (such as depression) and pain and fatigue is likely bidirectional, there are very good chronic disease self-management approaches that appear to result in improvements in reported symptoms of fatigue and pain experienced in the context of illness. Importantly, however, these self-management approaches help the patients learn to pace their activities and develop coping skills to better manage their symptoms. There is a long history across individual illnesses of using chronic self-management approaches to improve health outcomes for patients; however, there is now strong evidence that such approaches can be used rolled out in primary settings with successful outcomes in terms of reducing pain, fatigue, and depression among those with chronic illness (Ory et al., 2013). While this study was not specifically with older adults, *per se*, the average age of participants was 65. In those studies that have attempted to replicate chronic disease self-management approaches with older adults, they have generally been effective. Since both pain and fatigue are among the strongest predictors of depression among older adults, offering opportunities for chronic disease self-management approaches in primary care settings is an evidence-based way in which clinicians can help patients learn to reduce these symptoms, or at least the impact of these symptoms on their daily lives.

Increasing Disability and Frailty

The consequence of the aging process itself is that people's ability for physical activity declines over time; however, this natural part of the aging process is compounded by the presence of one or more physical health conditions. This can lead to older people giving up their valued activities, which not only further increases their disability but also contributes to secondary effects such as social isolation and depression. As older people become more disabled and more unwell, they can become at risk of developing frailty. Frailty is a condition that is seen predominantly in older adults, which is often associated with a sudden decline in functioning associated with a particular stressor (e.g., an illness, a fall), which sparks a sudden decline in functioning. Typically, the functional decline is seen in a range of indicators, such as increased fatigue, weight loss, weakness, an inability to walk at a usual rate, and a reduction in energy expenditure (see Clegg, Young, Iliffe, Rikkert, & Rockwood, 2013). Frailty typically occurs when there is dysfunction in a range of major organ systems (e.g., inflammatory, hematological, neuromuscular, endocrine, hormonal, adiposity), but it is when abnormalities are detected across multiple systems that patients are at a higher risk of developing frailty (Clegg et al., 2013). Frailty has important implications for older patients because it is strongly associated with increased dependence and the need for care, delirium, falls, and a higher rate of mortality.

The Need for Formal and Informal Caring

When elderly people become frail, the level of care that they need increases, and, as such, the development of frailty can often mark the transition from the bulk of care being informal care to requiring the majority of care provision to be formalized. Informal care refers to care that is provided to a person by someone in their family or community when the recipient of care is no longer able to provide that care for themselves. In most societies, the majority of care is provided informally; some studies estimate that up to 90% of care is informally provided (Adelman, Tmanova, Delgado, Dion, & Lachs, 2014). Although formal and informal care are often described separately, in reality, much informal caring is supplement by organized and formal caring, and indeed, the availability of some formal care can reduce the need for the recipient of care requiring a more permanent formalized care plan, such as transition to a nursing home. The majority of care recipients, where possible, prefer to be cared for in the community by informal carers. From a health economics position, informal caring is extremely important as the costs of formal care provision are substantially higher. However, health psychology has become increasingly interested in the consequences of informal caregiving in recent years. There is strong evidence from a range of sources that providing care is associated with increased distress, depression, and caregiver burden. Most of the research into caregiver burden has been in the dementia literature, but increasingly it is recognized that caregiver burden is an important problem for those who provide care. It is recommended to reduce the burden that doctors are aware of who is the primary caregiver and allow them to have some role in decision making and care planning, where possible. It is also recommended that caregiver burden be assessed and that caregivers be offered both practical support (e.g., respite) and help with learning skills for dealing with the caring role, where necessary (Adelman et al., 2014). Where caregiver burden is a problem, there are a number of meta-analyses that confirm the efficacy of psychoeducational programs for caregivers, although the majority are with caregivers of those with dementia. However, effects sizes are modest. Hence, for clinicians, these offer viable alternatives to helping patients struggling with caregiver burden. However, there is a clear gap in the literature to research the efficacy of caregiver interventions for a broader set of caregivers, most notably interventions adapted to the needs of the caregivers of older people with multi-morbidity are absent from the literature.

Fear of Falling

In addition to the physical concerns that accompany illness in later life, and the psychological consequences for those who provide care to older adults, there are a number of psychological issues that also emerge for the elderly themselves. Data strongly support the fact that anxiety disorders are actually less common among older adults. A number of potential explanations for this drop in prevalence have been discussed in the literature. It has been suggested that this could be a cohort effect, such that those who are currently in the elderly age group were encouraged to be more stoic, and hence anxiety has always been lower in this cohort. It has also been suggested that it may be a physiological phenomenon, such that people's level of arousal generally reduces with aging and so the physical manifestations of anxiety reduce. Another suggestion has been that people learn better methods of coping as they age, through experience, and hence they are less likely to find anxiety overwhelming in later life. However, an alternative suggestion is that it could be that current diagnostic systems are based upon the concerns of younger adults and that the concerns of older adults are different. One unique fear among older people is the fear of falling.

Fear of falling is very common among older people and for good reason. Particularly in the frail elderly, falls are a leading cause of serious injury, increase the risk that an older person will be discharged from an acute hospital setting to a formal care setting, and have a risk of subsequent mortality. Hence, it is unsurprising that as people age, fears of falling develop. Anxiety is typically manifested when an individual appraises a risk as likely and the consequences as important. Given that estimates suggest that as many as one in three older adults fall each year, then older adults are arguably sensible to be mindful of the risk of falling. Moreover, particularly those with medical conditions that include symptoms of balance disturbance or dizziness, the risk is greater. Similarly, the consequences of falling among the elderly, particularly the frail elderly, are serious, and more so for those with conditions like osteoporosis where the risk of serious injury is heightened. While understandable that older people may be fearful of falling, one of the strongest predictors of actually falling is the fear of falling itself. One likely mechanism for this relationship is the fact that those who are more fearful of falling avoid physical activity and the reduction in physical activity actually leads to greater disability that in turn is thought to increase the risk of falling. As such, the most strongly evidence-based intervention for fear of falling in the elderly is exercise. A recent meta-analytic review found that there was an estimated reduction across available studies of 37% for all falls, but importantly even larger reductions of 43% of severe injuries following falls and a 61% reduction in falls resulting in a fracture (El-Khoury, Cassou, Charles, & Dargent-Molina, 2013). There is less evidence to support psychological approaches to managing fear of falling, and this is a likely area for future research. There is evidence that more complex, multifaceted programs (e.g., including balance training, home modifications) produce larger results than exercise alone. There are currently relatively few randomized controlled trials of psychosocial interventions as an adjunct to exercise reported in the literature. Those that are available have adopted a cognitive behavioral orientation and shown good results on measures of fear of falling, and some have shown improvements in mobility. However, researchers need to determine whether adding a psychosocial component to a predominantly exercise-based intervention will enhance the efficacy of these programs on the primary outcome of falls prevention, as well as subjective self-reports.

Management of Depression in Older Adults with Chronic Physical Health Problems

As with anxiety, there is little evidence to suggest that older people are more likely to become depressed than young adults. However, having one or more chronic physical illnesses is a risk factor for the development of late-life depression. As with younger adults with depression, there is evidence that late life can be treated with the same types of interventions that are efficacious in younger adults, that is, antidepressant medication and psychotherapy. Although there are considerably fewer studies of interventions in the treatment of late-life depression compared with the number with younger people, there is nonetheless strong meta-analytic evidence to show that both pharmacotherapy and psychotherapy are efficacious in the treatment of depression among the elderly (Pinquart, Duberstein, & Lyness, 2006). Importantly, cognitive behavioral therapy, problem-solving therapy, and reminiscence therapy all have sufficient trials demonstrating efficacy that we can consider all three to be strong evidence-based alternative therapies for late-life depression (Pinquart, Duberstein, & Lyness, 2007). However, there are some limitations to the evidence base for those patients with chronic physical health problems. Although many of the trials of late-life depression include patients with physical health problems, some trials specifically exclude patients with complex health needs. Indeed,

Pinquart et al. (2007) found evidence that the inclusion of participants with physical health problems was associated with smaller effect sizes across studies. Few studies have compared the efficacy of different interventions (e.g., cognitive behavior therapy and problem-solving therapy), and few studies have specifically targeted patients with chronic ill health problems or multi-morbidity and clinical depression (see Sharpe et al., 2012). As such, there is a pressing need for researchers to investigate the most efficacious ways in which to treat late-life depression in patients with complex medical needs, who are by virtue of their physical health problems more likely to be depressed. There have been some excellent attempts to incorporate collaborative care management (Unützer et al., 2002) or prevention (van't Veer-Tazelaar et al., 2009) of late-life depression. The success of problem-solving therapy in these trials indicates that problem-solving therapy is an effective treatment for patients with late-life depression and complex needs, although we need more comparative research to determine the relative efficacy of problem-solving therapy and other forms of psychotherapy.

Author Biography

Louise Sharpe, PhD, is professor in the School of Psychology at the University of London and associate head for research education. Professor Sharpe completed her undergraduate degree and postgraduate training as a clinical psychologist at the University of Sydney and received her PhD from the University of London. As a researcher, Professor Sharpe has published more than 170 peer-reviewed papers and received more than \$5 million from funding agencies, including the National Health and Medical Research Council and the Australian Research Council. Her research program examines adjustment to illness and involves the development and evaluation of psychosocial interventions for people with chronic physical illness. She has been awarded the Ian M. Campbell award from the Australian Psychological Society for her contributions to clinical psychology and the Distinguished Career Award from the Australian Association of Cognitive Behavioural Therapy for her research and clinical practice of cognitive behavior therapy.

References

- Adelman, R. D., Tmanova, L. L., Delgado, D., Dion, S., & Lachs, M. S. (2014). Caregiver burden: A clinical review. *JAMA*, *311*(10), 1052–1060.
- Arean, P., Hegel, M., Vannoy, S., Fan, M. Y., & Unutzer, J. (2008). Effectiveness of problem-solving therapy for older, primary care patients with depression: Results from the IMPACT project. *The Gerontologist*, *48*(3), 311–323.
- Clegg, A., Young, J., Iliffe, S., Rikkert, M. O., & Rockwood, K. (2013). Frailty in elderly people. *The Lancet*, *381*(9868), 752–762.
- Cooper, J. A., Cadogan, C. A., Patterson, S. M., Kerse, N., Bradley, M. C., Ryan, C., & Hughes, C. M. (2015). Interventions to improve the appropriate use of polypharmacy in older people: A Cochrane systematic review. *BMJ Open*, *5*(12), e009235.
- Cuijpers, P., van Straten, A., & Smit, F. (2006). Psychological treatment of late-life depression: A meta-analysis of randomized controlled trials. *International Journal of Geriatric Psychiatry*, *21*(12), 1139–1149.
- El-Khoury, F., Cassou, B., Charles, M. A., & Dargent-Molina, P. (2013). The effect of fall prevention exercise programmes on fall induced injuries in community dwelling older adults: Systematic review and meta-analysis of randomised controlled trials. *BMJ*, *347*, f6234.

- Haverhals, L. M., Lee, C. A., Siek, K. A., Darr, C. A., Linnebur, S. A., Ruscin, J. M., & Ross, S. E. (2011). Older adults with multi-morbidity: Medication management processes and design implications for personal health applications. *Journal of Medical Internet Research*, 13(2), e44.
- Higgins, N., & Regan, C. (2004). A systematic review of the effectiveness of interventions to help older people adhere to medication regimes. *Age and Ageing*, 33(3), 224–229.
- Mujica-Mota, R. E., Roberts, M., Abel, G., Elliott, M., Lyratzopoulos, G., Roland, M., & Campbell, J. (2015). Common patterns of morbidity and multi-morbidity and their impact on health-related quality of life: Evidence from a national survey. *Quality of Life Research*, 24(4), 909–918.
- Ory, M. G., Ahn, S., Jiang, L., Smith, M. L., Ritter, P. L., Whitelaw, N., & Lorig, K. (2013). Successes of a national study of the chronic disease self-management program: Meeting the triple aim of health care reform. *Medical Care*, 51(11), 992–998.
- Pinquart, M., Duberstein, P. R., & Lyness, J. M. (2006). Treatments for later-life depressive conditions: A meta-analytic comparison of pharmacotherapy and psychotherapy. *American Journal of Psychiatry*, 163(9), 1493–1501.
- Pinquart, M., Duberstein, P. R., & Lyness, J. M. (2007). Effects of psychotherapy and other behavioral interventions on clinically depressed older adults: A meta-analysis. *Aging & Mental Health*, 11(6), 645–657.
- Read, J., Sharpe, L., Modini, M., & Dear, B. F. (2017). The relationship between multi-morbidity and depression: A meta-analysis. *Journal of Affective Disorders*, 221, 36–46.
- Sharpe, L., Gittins, C. B., Correia, H. M., Meade, T., Nicholas, M. K., Raue, P. J., ... Areán, P. A. (2012). Problem-solving versus cognitive restructuring of medically ill seniors with depression (PROMISE-D trial): Study protocol and design. *BMC Psychiatry*, 12(1), 1.
- Unützer, J., Katon, W., Callahan, C. M., Williams, J. W., Jr., Hunkeler, E., Harpole, L., ... Areán, P. A. (2002). Collaborative care management of late-life depression in the primary care setting: A randomized controlled trial. *JAMA*, 288(22), 2836–2845.
- van't Veer-Tazelaar, P. J., van Marwijk, H. W., van Oppen, P., van Hout, H. P., van der Horst, H. E., Cuijpers, P., ... Beekman, A. T. (2009). Stepped-care prevention of anxiety and depression in late life: A randomized controlled trial. *Archives of General Psychiatry*, 66(3), 297–304.

Suggested Reading

- Alexopoulos, G. S., Borson, S., Cuthbert, B. N., Devanand, D. P., Mulsant, B. H., Olin, J. T., & Oslin, D. W. (2002). Assessment of late life depression. *Biological Psychiatry*, 52(3), 164–174.
- Bryant, C., Jackson, H., & Ames, D. (2008). The prevalence of anxiety in older adults: Methodological issues and a review of the literature. *Journal of Affective Disorders*, 109(3), 233–250.
- Scheffer, A. C., Schuurmans, M. J., Van Dijk, N., Van Der Hooft, T., & De Rooij, S. E. (2008). Fear of falling: Measurement strategy, prevalence, risk factors and consequences among older persons. *Age and Ageing*, 37(1), 19–24.
- Sharpe, L., & Curran, L. (2006). Understanding the process of adjustment to illness. *Social Science & Medicine*, 62(5), 1153–1166.

Pediatric Psychology

Dianna Boone, Thomas J. Parkman, and Jason Van Allen

*Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University,
Lubbock, TX, USA*

Introduction

Pediatric psychology is defined by the Society of Pediatric Psychology as “an integrated field of science and practice in which the principles of psychology are applied within the context of pediatric health” (American Psychological Association, 2009). Pediatric psychologists can work in a variety of settings with children with a wide range of chronic illnesses including asthma, cancer, diabetes, obesity, pediatric sleep problems, sickle cell disease, and others (Aylward, Bender, Graces, & Roberts, 2009; Roberts, Biggs, Jackson, & Steele, 2011). The scope of practice for a pediatric psychologist can include screening for psychopathology in healthcare settings, developing interventions for children with chronic illnesses, and promoting the health and development of children and adolescents from all disease groups (Spirito et al., 2003). Pediatric psychologists are in great demand due to the prevalence of pediatric chronic conditions in the United States. For example, in the United States, 13,000 children are diagnosed with cancer each year, 13,000 children are diagnosed with type 1 diabetes, and 9 million children suffer from asthma (Compas, Jaser, Dunn, & Rodriguez, 2012). Obesity during childhood and adolescence is also a serious public health problem in the United States (Ogden, Carroll, Kit, & Flegal, 2014), and medical trauma results in many pediatric hospital admissions each year (Compas et al., 2012).

This chapter provides an overview of the clinical applications of pediatric health psychology including the importance of adjustment to new diagnoses and medication adherence. This chapter also discusses the various forms of treatment delivery and how the settings and modalities of treatments are changing. Finally, this chapter will discuss cultural and diversity issues in pediatric health psychology.

Adjustment to Diagnosis

Due to advances in the treatment of chronic illnesses, pediatric illnesses that were once fatal can now be treated much more effectively than in previous decades (Mokkink, Van der Lee, Grootenhuys, Offringa, & Heymans, 2008). These improved outcomes are a result of successful early detection and diagnosis, as well as effective treatment of many previously life-threatening illnesses (Compas et al., 2012). As a result, children and adolescents now have to adjust to living with chronic illnesses and medical conditions. These chronic illnesses present children, adolescents, and their families with significant stress that can contribute to emotional and behavioral problems as well as interfere with adherence to treatment regimens (Compas et al., 2012). Further, many pediatric illnesses are worsened by stress encountered in other aspects of children's lives. It is therefore essential to understand the ways that children and adolescents adjust to living with a chronic condition in order to develop effective interventions to enhance coping and adjustment.

Adjustment to Chronic Versus Acute Illnesses

Adjustment to a pediatric illness can be different for each child, depending on if he or she receives a diagnosis of a chronic illness versus a diagnosis of an acute illness. An acute illness is differentiated from a chronic illness by the speed of symptom onset and the duration of symptoms. An acute illness is a condition of short duration that starts quickly and has severe symptoms. However, a chronic illness is a health problem that lasts 3 months or more, affects a child's normal activities, and requires frequent hospitalizations, home healthcare, and/or extensive medical care (Mokkink et al., 2008). Research has revealed that chronic conditions may result in greater psychological and physical stress than acute illnesses that resolve quickly (Marin, Chen, Munch, & Miller, 2009). Individuals with a chronic illness may experience more psychological stress, especially during childhood or adolescence, because the illness can threaten one's identity, affect one's body image, and disrupt one's lifestyle (Gignac, Cott, & Badley, 2000). Chronic conditions are also typically characterized by different phases over the course of an individual's lifetime (e.g., acute periods, stable periods, remissions), in which each phase presents its own physical and psychological problems. In addition, one's family life could be dramatically altered due to a pediatric chronic illness, resulting in unfilled roles, loss of income, and costs of treatment. As a result, individual and familial adjustment to chronic illness is an important predictor of long-term outcomes.

Generally speaking, researchers have examined a variety of stress responses in children and adolescents, emphasizing both controlled responses and automatic responses (Compas et al., 2012). Automatic stress responses include "temperamentally based and conditioned ways of reacting to stress such as emotional and physiological arousal, automatic thoughts, and conditioned behaviors" (Compas et al., 2012). However, in order to effectively cope with stress, controlled responses are typically preferred, which involve things children and adolescents do to manage and adapt to stress (Compas et al., 2012). Overall, the strategies a child or adolescent uses to manage stress can provide an idea as to how they will adjust or cope with a chronic illness. It is important to note, however, that when coping with health- and illness-related stressors, the controllability or perceived controllability of the stressor may be a key dimension in determining the efficacy of particular coping strategies (Osowiecki & Compas, 1998).

Interventions That Improve Adjustment

In recent years, researchers have developed interventions that aim to reduce stress and improve coping strategies in children after being diagnosed with a pediatric illness. For example,

Kassam-Adams et al. (2015) assessed the effectiveness and feasibility of a web-based intervention aimed to reduce the stress of children who recently suffered an injury or were diagnosed with a chronic illness. Children ages 8–12 years were randomized to either a coping intervention (e.g., an interactive web-based game promoting coping strategies) or a 12-week wait list (Kassam-Adams et al., 2015). Outcome measures included child PTSD symptoms and pediatric quality of life. This study demonstrated that a novel, interactive, game-like web-based intervention for children exposed to medical trauma is feasible to deliver and has the potential to prevent posttraumatic stress after being diagnosed with a medical illness (Kassam-Adams et al., 2015).

Interventions targeting caregivers have also been developed, in light of research demonstrating an association between parental functioning and children's adjustment to a chronic illness (Palermo, Law, Essner, Jessen-Fiddick, & Eccleston, 2014). For instance, Palermo et al. (2014) examined the effectiveness and feasibility of a problem-solving skills training (PSST) intervention to reduce distress in caregivers of children with chronic pain. This study revealed that the parent-focused PSST intervention was effective in reducing distress among caregivers of children and adults with a variety of medical problems (Palermo et al., 2014). Results suggest that a PSST intervention is a feasible and acceptable intervention for parents of children with chronic pain. The intervention used in this study has the potential to help children with other chronic illnesses develop coping strategies and enable them to function effectively in their daily lives (Palermo et al., 2014).

In sum, chronic illnesses present children and their families with significant stress that can contribute to psychosocial problems, such as depression and anxiety, nonadherence to medication regimens, and disruptions to family life (Compas et al., 2012). These interventions have shown that they can help improve coping strategies and reduce stress in children adjusting to life with a chronic illness. Researchers need to continue examining how children and families adjust to pediatric illnesses to further inform and develop interventions.

Medication Adherence

The term “adherence” refers to the extent to which a person's behavior (e.g., taking medication, following a diet, and/or implementing lifestyle changes) corresponds with recommendations from a healthcare provider (World Health Organization [WHO], 2006). The assessment and treatment of medication adherence has become essential to improving health outcomes as the prevalence of pediatric chronic illnesses has increased (Quittner, Modi, Lemanek, Levers-Landis, & Rapoff, 2008). Research has illustrated that the overall treatment adherence rate is approximately 50% for pediatric populations (Rapoff, 1999). Nonadherence to medical regimes can potentially interfere with the efficacy of medications, resulting in failure to reach treatment goals, increased visits to the emergency room, and increased hospitalizations (Lee et al., 2014). In addition, other negative consequences of nonadherence include inappropriate changes in treatment regimens, decreased quality of life (Fredericks et al., 2008), incorrect medication dosage adjustments, and increased costs, among others (Lee et al., 2014).

Barriers That Affect Medication Adherence

Medication adherence in pediatric populations presents unique challenges. Types of barriers that are associated with poor medication adherence in pediatric patients include cognitive factors (e.g., forgetting, poor planning), aversive medication properties or difficulties ingesting medication (e.g., hard to swallow, bad taste, vomiting, spitting), high cost of medication, or

voluntary resistance toward taking the medication (Hommel & Baldassano, 2010; Lee et al., 2014; Simons, McCormick, Mee, & Blount, 2009). Medication adherence can be especially difficult for young pediatric patients who may not understand the purpose of their medication and who depend on their caregivers to administer their prescribed medication (Lee et al., 2014). Thus, it is extremely important that both children and their caregivers participate in the medication adherence process.

In addition, it is also common for adolescents to not regularly follow their medication regimens (Staples & Bravender, 2002). Factors that could negatively impact adolescents' medication adherence include embarrassment, stigmatization, and inability to self-regulate (e.g., ability to control one's behaviors, emotions, and cognitions; Berg et al., 2014). Berg et al. (2014) found that self-regulation in a number of domains (i.e., general executive functioning, attention, self-control, and emotion regulation) was associated with adolescents' adherence behaviors in a sample of youths with type 1 diabetes, such that youths who reported lower self-regulation abilities demonstrated worse adherence behaviors than those who reported higher self-regulation abilities.

In addition, barriers to adherence have been associated with negative psychosocial outcomes such as less family cohesion, less emotional expression, and greater conflict among family members (Simons & Blount, 2007), which highlights the importance of efforts aimed to reduce barriers and improve pediatric adherence.

Treatment Delivery

Recently, Western medicine is recognizing the importance of viewing patient care from the biopsychosocial model, which emphasizes the interplay of biological, psychological, and social factors as they either influence the maintenance of health or acquire the capacity to cause illness (Smith & Nicassio, 1995). The importance of approaching treatment from this holistic standpoint is so salient and necessary that it has been endorsed and adopted by the WHO (2002). Consequently, pediatric psychologists are now delivering treatment for acute and chronic pediatric physical and behavioral health conditions in a number of diverse settings. Such settings include inpatient and outpatient medical or psychiatric facilities and pediatric primary care clinics (Borschuk, Jones, Parker, & Crewe, 2015).

In these medical environments, pediatric psychologists are typically an integral component of integrated teams composed of psychologists, doctors, nurses, caseworkers, and even administrators who work collaboratively to determine the best course of treatment for the patient (Butler et al., 2008). One of the charges of the pediatric psychologist in this setting is to provide secondary care via psychodiagnostic assessment. Psychometric tests are often used in this environment in order to identify treatment needs, assist with differential diagnoses, monitor treatment progress, and facilitate risk management (Wahass, 2005). Assessments of intelligence, personality, mental status, motivation, health behavior, and presence of psychopathology are typically conducted by the pediatric psychologist and are useful aids in ensuring children receive the highest standard of care.

Another charge of pediatric psychologists in medical settings is to provide primary care via behavioral interventions (e.g., cognitive behavioral therapy) targeted at treating or even preventing behavioral disturbances or psychopathology (e.g., anxiety) that may be exacerbating the child's presenting concerns or impeding their recovery (Borschuk et al., 2015). Although limited, research does suggest that behavioral health interventions are effective at mitigating

anxiety (Kolko et al., 2014), behavioral problems (Berkovits, O'Brien, Carter, & Eyberg, 2010), and depression (Richardson, McCauley, & Katon, 2009).

Other crucial roles of pediatric psychologists include facilitating communication among medical professionals, managing difficult interpersonal situations between patients and their families, and providing grief counseling as necessary. Finally, the pediatric health psychologist also facilitates the post-discharge outpatient follow-ups in order to ensure the child is adhering to their treatment regimen, there are no residual complications, and, if necessary, the psychologist is prepared to provide referrals to community-based care providers (Kolko & Perrin, 2014).

With increasing frequency children are receiving care via community-based programs and facilities, such as schools (Borschuk et al., 2015). In fact, research suggests that more children are receiving services in community-based outpatient clinics (satellite healthcare facilities that provide health and wellness services outside of the primary care setting) and schools than they are in primary or specialty care clinics (National Survey on Drug Use & Health, 2009). The utility of the pediatric psychologist in the community can be found in their ability to facilitate support groups for children seeking emotional support due to terminal illnesses or chronic pain, assist in medication/treatment adherence, and administer psychometric tests of functioning (Straub, 2014). Furthermore, pediatric psychologists in the community setting also aim to disseminate public health information and provide psychoeducation to children regarding illnesses and disease prevention (Straub, 2014).

The Use of Telemedicine to Treat Pediatric Illnesses

Telemedicine can be broadly defined as the use of telecommunications technologies to provide medical information and services (Perednia & Allen, 1995). The use of telemedicine can help overcome barriers such as geographic challenges, a lack of healthcare specialists, and social and economic barriers (Marcin, Shaikh, & Steinhorn, 2016; Van Allen, Davis, & Lassen, 2011). In recent years, telemedicine has been increasingly used for pediatric health services, and research supports the utility of interventions incorporating telemedicine for children in rural settings (Van Allen et al., 2011). For example, Davis et al. (2013) demonstrated that a telemedicine intervention was equally as effective as an intervention involving visits from a physician in improving children's BMI, behavior problems, and dietary and physical activity behaviors. This study also demonstrated that telemedicine or similar methods of telehealth can be a feasible method of treatment delivery of empirically supported treatment interventions (Davis, Sampilo, Gallagher, Landrum, & Malone, 2013). Similarly, a study conducted by Chorianopoulou, Lialiou, Mechili, Mantas, and Diomidous (2015) also revealed that the utilization of telemedicine resulted in improved access to specialized healthcare, reduced hospitalizations, and emergency room visits in a sample of children from a rural population (Chorianopoulou et al., 2015). Izquierdo et al. (2009) found that children with diabetes who received telemedicine had lower hemoglobin A1c levels and reported higher levels of pediatric quality of life, compared with children who received usual care. Children in the telemedicine group also reported a reduction in urgent diabetes-related calls initiated by the school nurse and fewer hospitalizations and emergency department visits (Izquierdo et al., 2009). In addition, Romano, Hernandez, Gaylor, Howard, and Knox (2001) investigated whether specialist care delivered by telemedicine would result in comparable improvements in quality of life and asthma symptoms, compared with face-to-face encounters with

specialists. The results indicated that patients reported an increase in mean number of symptom-free days, a reduction in mean symptom scores, and improvements in quality of life (Romano et al., 2001).

It is likely that more interventions will be delivered through telemedicine technologies in the near future. The use of telemedicine and digital technologies can help children and families overcome numerous geographic and economic barriers as well as increase access to interventions and other forms of treatment.

Cultural/Ethical Disparities, Culturally Competent Care, and Future Directions

Disparities within the realm of pediatric psychology are predominantly driven by variables such as race, ethnicity, and socioeconomic status. Unfortunately, children of color, especially those born into low-income families, lag behind their more affluent, majority peers in health status (Cheng, Dreyer, & Jenkins, 2009). This is of particular concern given the fact that such disparities that arise during childhood positively correlate with chronic illness as an adult (Braveman & Barclay, 2009). Also, the dearth of available pediatric psychologists in underprivileged or rural areas is also a force driving these disparities in health status.

These disparities point to the need for pediatric psychology to embrace and practice culturally competent care, care that is sensitive to the needs, health literacy, and health beliefs of pediatric patients and their families (Cheng, Emmanuel, Levy, & Jenkins, 2015). Current research demonstrates that health promotion and prevention interventions may be more effective in improving quality of care and patient safety if they are tailored to the cultural identity of the target population. However, there is a paucity of research as to what constitutes a “culturally sensitive” intervention, and whether such an initiative could be capable of facilitating greater changes in behavior than non-culturally specific interventions is unclear (Resnicow et al., 2002).

With that said, there are a number of measures the field of pediatric psychology can enact in order to ameliorate disparities in health status among children of racial and ethnic minorities and socioeconomic status. Research suggests that almost all children in the United States have annual well-child visits (Bloom, Cohen, & Freeman, 2011); thus, increasing the involvement of pediatric psychologists in the primary care setting—in the role of gatekeeper to behavioral health services—will have the effect of fostering normative development and decreasing health disparities. Furthermore, given the powerful deleterious effect of socioeconomic status on service availability, the field of pediatric health psychology must seek to make access to its services more universally accessible and cost-effective. This can be done through the proliferation of pediatric psychologists throughout community-based care providers, perhaps with the future aid of government or community cost subsidization.

Conclusion

Pediatric psychology is a rapidly expanding field of psychology. Pediatric psychologists work in a multitude of professional settings in the evaluation and treatment of children who present with a wide range of chronic illnesses. It is critical to understand how children and families adjust to pediatric illness to further inform and develop more effective interventions for these individuals. Pediatric psychologists work with children and families, in conjunction with

physicians, to reinforce the importance of medication adherence to maximize positive health outcomes. Pediatric psychologists are working with children in schools and community settings, not just the primary care setting, to promote medical and mental health. The use of telemedicine and digital technologies has become important delivery systems for providing education and interventions for children and families in the rural setting. Identifying pertinent cultural variables and considerations are critical for effective health promotion and prevention interventions. Pediatric psychologists and researchers must continue to develop evidence-based treatments and examine resilience factors (e.g., self-esteem, internal locus of control, motivation, health behaviors, coping strategies, emotional regulation) that prevent children from developing chronic illnesses and a lower quality of life. Through these measures, the field of pediatric psychology can ensure that children grow into healthy and well-adjusted adults.

Author Biographies

Dianna Boone, MA, is a second year student in the Clinical Psychology Program at Texas Tech University, under the mentorship of Dr. Jason Van Allen. She received her MA in Rehabilitation and Mental Health Counseling from the University of South Florida. Her research interests include pediatric psychology and childhood obesity.

Thomas J. Parkman, BS. T.J. is a third year clinical psychology doctoral student and research assistant in the ENERGY Lab at Texas Tech University, under the mentorship of Dr. Jason Van Allen, PhD. T.J.'s interests rest within the realms of pediatric health psychology and behavioral medicine.

Jason Van Allen, PhD. Jason is an assistant professor in the Clinical Psychology Program at Texas Tech University. He received his PhD in Clinical Child Psychology from the University of Kansas, and his research focuses on pediatric psychology and childhood obesity.

References

- American Psychological Association. (2009). *Society of pediatric psychology*. Retrieved from <http://www.apa.org/about/division/div54.aspx>.
- Aylward, B. S., Bender, J. A., Graves, M. M., & Roberts, M. C. (2009). Historical developments and trends in pediatric psychology. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (pp. 3–18). New York, NY: Guilford.
- Berg, C. A., Wiebe, D. J., Suchy, Y., Hughes, A. E., Anderson, J. H., Godbey, E. I., ... White, P. C. (2014). Individual differences and day-to-day fluctuations in perceived self-regulation associated with daily adherence in late adolescents with type 1 diabetes. *Journal of Pediatric Psychology, 39*(9), 1038–1048.
- Berkovits, M. D., O'Brien, K. A., Carter, C. G., & Eyberg, S. M. (2010). Early identification and intervention for behavior problems in primary care: A comparison of two abbreviated versions of parent-child interaction therapy. *Behavior Therapy, 41*(3), 375–387.
- Bloom, B., Cohen, R. A., & Freeman, G. (2011). Summary health statistics for U.S. children: National Health Interview Survey, 2010. *Vital And Health Statistics. Series 10, Data From The National Health Survey, 250*, 1–80.
- Borschuk, A. P., Jones, H. A., Parker, K. M., & Crewe, S. (2015). Delivery of behavioral health services in a pediatric primary care setting: A case illustration with adolescent depression. *Clinical Practice In Pediatric Psychology, 3*(2), 142–153.

- Braveman, P., & Barclay, C. (2009). Health disparities beginning in childhood: A life-course perspective. *Pediatrics*, 124(Supplement 3), S163–S175.
- Butler, M., Kane, R. L., McAlpine, D., Kathol, R. G., Fu, S. S., Hagedorn, H., & Wilt, T. J. (2008). Integration of mental health/substance abuse and primary care. *Agency for Healthcare Research and Quality (AHRQ) Evidence Report/Technology Assessment*, 173, 1–362.
- Cheng, T. L., Dreyer, B. P., & Jenkins, R. R. (2009). Introduction: Child health disparities and health literacy. *Pediatrics*, 124(Supplement 3), S161–S162.
- Cheng, T. L., Emmanuel, M. A., Levy, D. J., & Jenkins, R. R. (2015). Child health disparities: What can a clinician do? *Pediatrics*, 136(5), 961–968.
- Chorianopoulou, A., Lialiou, P., Mechili, E. A., Mantas, J., & Diomidous, M. (2015). Investigation of the quality and effectiveness of telemedicine in children with diabetes. *Studies in Health Technology and Informatics*, 210, 105–109.
- Compas, B. E., Jaser, S. S., Dunn, M. J., & Rodriguez, E. M. (2012). Coping with chronic illness in childhood and adolescence. *Annual Review of Clinical Psychology*, 8, 455–480.
- Davis, A. M., Sampilo, M., Gallagher, K. S., Landrum, Y., & Malone, B. (2013). Treating rural pediatric obesity through telemedicine: Outcomes from a small randomized controlled trial. *Journal of Pediatric Psychology*, 38(9), 932–943.
- Fredericks, E. M., Magee, J. C., Opiari-Arrigan, L., Shieck, V., Well, A., & Lopez, M. J. (2008). Adherence and health-related quality of life in adolescent liver transplant recipients. *Pediatric Transplantation*, 12(3), 289–299.
- Gignac, M. A., Cott, C., & Badley, E. M. (2000). Adaptation to chronic illness and disability and its relationship to perceptions of independence and dependence. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 55(6), P362–P372.
- Hommel, K. A., & Baldassano, R. N. (2010). Brief report: Barriers to treatment adherence in pediatric inflammatory bowel disease. *Journal of Pediatric Psychology*, 35(9), 1005–1010.
- Izquierdo, R., Morin, P. C., Bratt, K., Moreau, Z., Meyer, S., Ploutz-Snyder, R., ... Weinstock, R. S. (2009). School-centered telemedicine for children with type 1 diabetes mellitus. *The Journal of Pediatrics*, 155(3), 374–379.
- Kassam-Adams, N., Marsac, M. L., Kohser, K. L., Kenardy, J., March, S., & Winston, F. K. (2015). Pilot randomized controlled trial of a novel web-based intervention to prevent posttraumatic stress in children following medical events. *Journal of Pediatric Psychology*, 41(1), 138–148.
- Kolko, D. J., Campo, J., Kilbourne, A. M., Hart, J., Sakolsky, D., & Wisniewski, S. (2014). Collaborative care outcomes for pediatric behavioral health problems: A cluster randomized trial. *Pediatrics*, 133(4), e981–e992.
- Kolko, D. J., & Perrin, E. (2014). The integration of behavioral health interventions in children's health care: Services, science, and suggestions. *Journal of Clinical Child & Adolescent Psychology*, 43(2), 216–228.
- Lee, J. L., Eaton, C., Gutiérrez-Colina, A. M., Devine, K., Simons, L., Mee, L., & Blount, R. L. (2014). Longitudinal stability of specific barriers to medication adherence. *Journal of Pediatric Psychology*, 39(7), 667–676.
- Marcin, J. P., Shaikh, U., & Steinhorn, R. H. (2016). Addressing health disparities in rural communities using telehealth. *Pediatric Research*, 79(1-2), 169–176.
- Marin, T. J., Chen, E., Munch, J. A., & Miller, G. E. (2009). Double-exposure to acute stress and chronic family stress is associated with immune changes in children with asthma. *Psychosomatic Medicine*, 71(4), 378–384.
- Mokink, L. B., Van der Lee, J. H., Grootenhuys, M. A., Offringa, M., & Heymans, H. S. (2008). Defining chronic diseases and health conditions in childhood (0–18 years of age): National consensus in the Netherlands. *European Journal of Pediatrics*, 167(12), 1441–1447.
- National Survey on Drug Use and Health. (2009). *Substance abuse and mental health services administration*. Rockville, MD: Office of Applied Studies.

- Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011–2012. *Journal of the American Medical Association*, 311(8), 806–814.
- Osowiecki, D., & Compas, B. E. (1998). Psychological adjustment to cancer: Control beliefs and coping in adult cancer patients. *Cognitive Therapy and Research*, 22(5), 483–499.
- Palermo, T. M., Law, E. F., Essner, B., Jessen-Fiddick, T., & Eccleston, C. (2014). Adaptation of problem-solving skills training (PSST) for parent caregivers of youth with chronic pain. *Clinical Practice in Pediatric Psychology*, 2(3), 212–223.
- Perednia, D. A., & Allen, A. (1995). Telemedicine technology and clinical applications. *Journal of the American Medical Association*, 273(6), 483–488.
- Quittner, A. L., Modi, A. C., Lemanek, K. L., Levers-Landis, C. E., & Rapoff, M. A. (2008). Evidence-based assessment of adherence to medical treatments in pediatric psychology. *Journal of Pediatric Psychology*, 33(9), 916–936.
- Rapoff, M. A. (1999). *Adherence to pediatric medical regimens*. Dordrecht, Netherlands: Kluwer Academic Publishers.
- Resnicow, K., Jackson, A., Braithwaite, R., DiIorio, C., Blisset, D., Rahotep, S., & Periasamy, S. (2002). Healthy body/healthy spirit: A church-based nutrition and physical activity intervention. *Health Education Research*, 17(5), 562–573.
- Richardson, L., McCauley, E., & Katon, W. (2009). Collaborative care for adolescent depression: A pilot study. *General Hospital Psychiatry*, 31(1), 36–45.
- Roberts, M. C., Biggs, B. K., Jackson, Y., & Steele, R. G. (2011). Clinical child psychology: Research and practice applications. In P. Martin, F. Cheung, M. Kyrios, L. Littlefield, M. Knowles, B. Overmier, & J. M. Prieto (Eds.), *The IAAP handbook of applied psychology* (pp. 3–27). New York, NY: Wiley-Blackwell.
- Romano, M. J., Hernandez, J., Gaylor, A., Howard, S., & Knox, R. (2001). Improvement in asthma symptoms and quality of life in pediatric patients through specialty care delivered via telemedicine. *Telemedicine Journal and E-Health*, 7(4), 281–286.
- Simons, L. E., & Blount, R. L. (2007). Identifying barriers to medication adherence in adolescent transplant recipients. *Journal of Pediatric Psychology*, 32(7), 831–844.
- Simons, L. E., McCormick, M. L., Mee, L. L., & Blount, R. L. (2009). Parent and patient perspectives on barriers to medication adherence in adolescent transplant recipients. *Pediatric Transplantation*, 13(3), 338–347.
- Smith, T. W., & Nicassio, P. M. (1995). Psychological practice: Clinical application of the biopsychosocial model. In P. M. Nicassio, T. W. Smith, P. M. Nicassio, & T. W. Smith (Eds.), *Managing chronic illness: A biopsychosocial perspective* (pp. 1–31). Washington, DC: American Psychological Association.
- Spirito, A., Brown, R. T., D'Angelo, E., Delamater, A., Rodrigue, J., & Siegel, L. (2003). Society of Pediatric Psychology Task Force Report: Recommendations for the training of pediatric psychologists. *Journal of Pediatric Psychology*, 28(2), 85–98.
- Staples, B., & Bravender, T. (2002). Drug compliance in adolescents: Assessing and managing a modifiable risk factors. *Pediatric Drugs*, 4(8), 503–513.
- Straub, R. O. (2014). *Health psychology* (4th ed., pp. 488–499). New York, NY: Worth Publishers.
- Van Allen, J., Davis, A. M., & Lassen, S. (2011). The use of telemedicine in pediatric psychology: Research review and current applications. *Child and Adolescent Psychiatric Clinics of North America*, 20(1), 55–66.
- Wahass, S. H. (2005). The role of psychologists in health care delivery. *Journal of Family & Community Medicine*, 12(2), 63–70.
- World Health Organization. (2002). *The world health report 2002: Reducing risks, promoting healthy life*. Retrieved from <http://www.who.int/whr/2002/en/>.
- World Health Organization. (2006). *Adherence to long-term therapies: Evidence for action*. Retrieved from http://www.who.int/chp/knowledge/publications/adherence_introduction.pdf.

Suggested Reading

- Borschuk, A. P., Jones, H. A., Parker, K. M., & Crewe, S. (2015). Delivery of behavioral health services in a pediatric primary care setting: A case illustration with adolescent depression. *Clinical Practice in Pediatric Psychology*, 3(2), 142–153.
- Compas, B. E., Jaser, S. S., Dunn, M. J., & Rodriguez, E. M. (2012). Coping with chronic illness in childhood and adolescence. *Annual Review of Clinical Psychology*, 8, 455–480.
- Kolko, D. J., & Perrin, E. (2014). The integration of behavioral health interventions in children's health care: Services, science, and suggestions. *Journal of Clinical Child & Adolescent Psychology*, 43(2), 216–228.

Reproductive Health

Jennifer L. Brown¹ and Nicole K. Gause²

¹*Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati College of Medicine, Cincinnati, OH, USA*

²*Department of Psychology, University of Cincinnati, Cincinnati, OH, USA*

As defined by the World Health Organization (WHO), reproductive health refers to the well-being of reproductive processes throughout all stages of life; this includes the ability to have satisfying and safe sex, as well as the ability to reproduce (WHO, 2016a). Additionally, reproductive health involves the capability to decide when and how often to engage in sexual and reproductive behaviors (WHO, 2016a). According to the WHO, to maintain reproductive health, men and women should also have access to safe, effective, and affordable contraception, as well as access to healthcare services that facilitate healthy pregnancies and childbirths (WHO, 2016a).

This chapter provides an overview of reproductive health issues that may be of clinical relevance to a health psychologist. Effective strategies for conducting sexual health histories to assess patients' sexual behavior engagement and sexual orientation are provided. Sexual behaviors that are associated with increased risk for adverse sexual health consequences such as sexually transmitted infections (STIs) and unintended pregnancies are described. Epidemiological data regarding STI prevalence is provided, as well as an overview of efficacious human immunodeficiency virus (HIV) and STI prevention intervention approaches. Modern contraceptive options that provide safe, effective methods of pregnancy prevention are discussed. Disorders related to patients' sexual health including sexual dysfunction disorders, hypersexuality, and paraphilic disorders are discussed, as well as relevant diagnostic considerations and efficacious treatment options. A brief summary of sexual behavior engagement from a developmental perspective is provided at the end of the chapter.

Sexual Behaviors

There is a wide variety of activities that individuals engage in to express their sexuality (Crooks & Baur, 2008). Abstinence and celibacy refer to the act of refraining from engaging in certain sexual behaviors or any sexual behaviors. Self-stimulation of one's genitals for the purpose of sexual pleasure is a sexual behavior known as masturbation. Sexual behaviors such as kissing and touching stimulate the erogenous zones of oneself and one's partner. Individuals may also sexually stimulate a partner via oral stimulation of a partner's genitals. Terms such as oral sex (referring broadly to oral-genital stimulation), cunnilingus (oral stimulation of the vulva), and fellatio (oral stimulation of the penis) describe oral-genital sexual behaviors. Anal stimulation may involve touching around the anus or penile insertion in the anus (often referred to as anal sex). Penile insertion in a female's vagina is referred to as penile-vaginal intercourse, as well as other terms such as vaginal sex or coitus. There is great variability between individuals in how frequently these and other sexual behaviors occur; this variability may be due to a number of factors, including age and the perceived social acceptability of certain behaviors.

Furthermore, reasons for engaging in sexual behaviors may differ among individuals and even within the same individual depending on situational circumstances. People may engage in sexual behaviors in order to experience sexual pleasure, sexual arousal, or orgasm. Motivation for sexual behaviors may also involve a desire to procreate. Some individuals may engage in sexual behaviors to earn money or acquire other goods or services; prostitution is the exchange of a sexual behavior for monetary or other compensation. Unfortunately, there are circumstances under which engagement in sexual behaviors is coerced or nonconsensual (e.g., rape), a form of abuse (e.g., child sexual abuse), or a means of sexual exploitation (e.g., pedophilia). Even when sexual behavior is intended and consensual, it may have unintended consequences (e.g., unplanned pregnancy) or put one at risk for HIV and other STI.

A comprehensive general health history should include a sexual history to assess for engagement in risky sexual behaviors, as well as for potential sexual dysfunction (Peck, 2001). Structured patient questionnaires may also be incorporated into sexual histories. The core domains that should be assessed when gathering sexual history information are sexual practices (i.e., which specific behaviors the individual engages in), any concerns related to sexual functioning, information regarding sexual partners (e.g., number of partners, partners' sex(es), relationship status), use of pregnancy prevention method(s), use of STI/HIV prevention method(s), and STI and/or pregnancy history. Gathering this information requires providers discuss sensitive topics; this may make some providers uneasy or uncomfortable and result in avoidance of performing a comprehensive sexual history assessment (Peck, 2001). Provider discomfort in discussing sexual health may cause patient embarrassment or discomfort, which may in turn have a negative impact on provider-patient rapport and the accuracy of a patient's self-reported sexual information. Furthermore, failure to perform a comprehensive sexual history assessment could interfere with patient care and may result in incorrect diagnoses or not providing appropriate referrals and treatments. In order to increase patient comfort, build rapport, and obtain the most accurate information, providers should promote open communication, emphasize confidentiality surrounding patient disclosure, employ open-ended questions, and utilize neutral language when discussing sexual behaviors and sexual health topics (Peck, 2001).

Sexual Orientation

Sexual orientation refers to the sex or sexes to which one is attracted. Although an individual may be sexually attracted to a particular sex or sexes, he or she may not necessarily identify with

the corresponding sexual orientation. Further, an individual may engage in sexual behaviors that are associated with particular sexual orientations (e.g., a man who reports sex with other men), but not identify with that particular sexual orientation; in this example, this man would endorse being heterosexual as opposed to homosexual or bisexual. Some argue that sexual orientation is an aspect of an individual's broader sense of identity, which is not only based on their sexual attractions but also on group membership with others who share a similar attraction. Sexual orientation is often classified by society according to distinct categories; individuals are either heterosexual (sexually attracted to the opposite sex), homosexual/lesbian/gay (sexually attracted to the same sex), or bisexual (sexually attracted to both sexes). However, some have suggested that sexual orientation may perhaps be better understood as existing along a continuum (Crooks & Baur, 2008). For example, Kinsey, Pomeroy, and Martin (1948) proposed in their early work that sexual orientation be viewed as a range from "exclusively heterosexual with no homosexual" to "exclusively homosexual with no heterosexual" attractions and/or sexual contact.

Individuals who identify as lesbian, gay, bisexual, or transgender (LGBT) experience a variety of health disparities, including increased STI prevalence, decreased screening for cervical cancer, and experiences of abuse or violence. Knowing a patient's sexual orientation can help to identify potential areas of concern to be addressed via further assessment or treatment. Using information obtained via a sexual history assessment not only improves patient care at the individual level but also has the potential to reduce health disparities among LGBT individuals. Sexual orientation can be assessed by asking a patient whether he or she (a) identifies with a particular sexual orientation (e.g., "Do you consider yourself (i) lesbian, gay, or homosexual; (ii) straight or heterosexual; (iii) bisexual; (iv) something else; (v) don't know?") and whether he or she (b) has any potential concerns related to his/her sexual orientation (e.g., "Do you have concerns related to your sexual orientation?"). In addition, *gender identity* should be assessed by asking patients about their (a) self-identified gender identity (e.g., "Do you identify as (i) male, (ii) female, (iii) female to male/transgender male, (iv) male to female/transgender female, (v) gender queer/neither exclusively male nor female, (vi) additional/other category?") and asking patients about their (b) sex assigned at birth (e.g., male, female). Given that the LGBT community encounters heightened stigma and discrimination, it is especially important that providers ensure patients' confidentiality and that assessment of sexual orientation and gender identity are conducted in a nonthreatening, nonjudgmental and appropriate manner.

Sexual Risk Behaviors

Estimates of the number of newly acquired HIV infections per year in the United States remain relatively stable, with approximately 50,000 incident cases diagnosed annually (Centers for Disease Control and Prevention [CDC], 2015c). Globally, there were approximately 2.1 million incident HIV cases in 2015 (WHO, 2016b). Furthermore, HIV prevalence continues to rise globally, with an estimated 36.7 million people now living with HIV worldwide (WHO, 2016b). In the United States, Black/African Americans and Hispanic/Latinos have higher HIV prevalence rates as compared with other racial or ethnic groups (CDC, 2015b, 2016a). In addition, men who have sex with men (MSM) are disproportionately affected by HIV in the United States (CDC, 2015a). Unlike trends that emerged early in the HIV epidemic, US women (particularly racial/ethnic minority women) who have sex with men have also experienced increased incident HIV infections in recent years (CDC, 2016b).

In addition to HIV, there are a number of other STIs including bacterial vaginosis (BV), chlamydia, gonorrhea, viral hepatitis, genital herpes, human papillomavirus, pelvic

inflammatory disease (PID), syphilis, and trichomoniasis. Negative health consequences may result from STI, and untreated STI may result in increased biological susceptibility to HIV. MSM, and racial/ethnic minority MSM in particular, experience elevated rates of STIs including chlamydia, gonorrhea, syphilis, and, as noted in a previous section, HIV (CDC, 2015a). Furthermore, individuals between the ages of 15 and 24 years of age (i.e., emerging adults), and racial/ethnic minority adolescents in particular, experience elevated rates of chlamydia and gonorrhea (CDC, 2014a).

Certain sexual behaviors, as well as substance use, may increase STI/HIV risk. For example, engaging in sex without a condom, having multiple sexual partners, or injecting drugs (and needle sharing) may increase likelihood of STI/HIV exposure. Moreover, there are structural factors that affect STI/HIV exposure risk; for example, community STI/HIV prevalence and access to adequate STI/HIV testing or treatment services as well as access to needle exchange programs affect STI and HIV prevalence. A comprehensive assessment of engagement in behaviors that increase STI/HIV risk facilitates and informs appropriate STI/HIV testing and treatment recommendations. Practitioners should assess for engagement in key behaviors that may increase an individual's STI/HIV risk; these include (a) condom use for vaginal and anal sex, (b) frequency of particular sexual behaviors (e.g., frequency of anal sex), (c) number of sexual partners, and (d) use of injection drugs and related behaviors (e.g., needle sharing). Of course, given the personal and sensitive nature of these behaviors, supplementing a face-to-face patient sexual history assessment interview with paper-and-pencil or computerized measures may enhance the accuracy of patients' self-reported behaviors. Furthermore, focusing on shorter, more discrete time periods (e.g., "How often in the *past month* have you had vaginal sex without a condom?") rather than extended time periods (e.g., "How often in your *lifetime* have you had vaginal sex without a condom?") may improve the accuracy with which patients report their behaviors. Providers should deliver appropriate risk reduction counseling (e.g., encouraging consistent condom use) to patients who report engaging in sexual risk behaviors and may also want to consider further STI/HIV testing in accordance with national guidelines for high-risk patients. For example, the US CDC recommends that all sexually active women under the age of 25 be annually screened for chlamydia (CDC, 2015d).

STI/HIV Prevention Intervention Approaches

Behavioral intervention strategies focus on decreasing high-risk practices (e.g., non-condom-protected sexual encounters and sharing of contaminated needles) to reduce and prevent the spread of STI/HIV. Indeed, there are a number of behavioral STI/HIV prevention approaches that have been reviewed, evaluated, and identified as efficacious behavioral HIV prevention interventions by the CDC (2014b). These behavioral intervention approaches utilize a variety of intervention modalities (e.g., one on one, small group) and generally target at-risk populations (e.g., MSM, African American adolescents; CDC, 2014b). For example, HORIZONS (DiClemente et al., 2009) is an STI/HIV prevention intervention that is gender and culturally tailored for African American young women. This particular intervention includes two group-based sessions designed to reduce sexual risk behavior engagement and incident STI among African American adolescents; results from the original randomized controlled trial indicated that HORIZONS increased condom use practices and reduced incident chlamydial infections (DiClemente et al., 2009). When used alone, behavioral interventions may reduce STI/HIV incidence, as well as engagement in risky sexual behaviors, use of contaminated needles, and other behaviors that increase STI/HIV risk.

The HIV prevention “toolkit” has expanded to include biomedical approaches as adjuncts to efficacious behavioral strategies. Biomedical HIV prevention approaches include a diverse set of strategies at different stages of product development, of varying efficacy, and at various stages of approval, including (a) microbicides that are applied to the vagina or the rectum; (b) pre-exposure prophylaxis (PrEP) antiretroviral (ARV) medications (ARV medications for high-risk seronegative persons); (c) post-exposure prophylaxis (PEP) drugs (use of ARV medications following HIV exposure); (d) HIV vaccination; (e) medical male circumcision; (f) HIV testing, linkage, and retention in HIV care (“test and treat”); and (g) enhanced ARV adherence among HIV-seropositive individuals (treatment as prevention). While the use of microbicides and an HIV vaccination have not been established as efficacious or approved for widespread use, they present possible avenues to pursue in future research; should efficacious treatments be established, they may offer additional means to curtail the HIV epidemic. Medical male circumcision, test-and-treat interventions, and ARV medication adherence strategies have demonstrated promise in reducing HIV transmission risk. In 2012, the US Food and Drug Administration approved Truvada (emtricitabine/tenofovir disoproxil fumarate) as a PrEP medication to be used in conjunction with safer sexual practices to prevent the spread of HIV. Combination HIV prevention approaches that integrate both efficacious behavioral and biomedical strategies may enhance the potential to reduce HIV transmission among certain high-risk populations (Brown, Sales, & DiClemente, 2014).

Contraceptive Methods

There are a variety of contraceptive methods available, which may be more or less desirable depending on an individual’s reproductive healthcare needs and preferences. Contraceptive methods vary in the degree of pregnancy prevention efficacy, STI prevention efficacy, and availability, which may also impact an individual’s contraceptive method choices. Male condoms are effective at preventing pregnancy and protect against STI; they are widely available and do not require a prescription or doctor visit. Hormonal contraceptives include oral contraceptive pills, contraceptive patch, vaginal ring, implants, intrauterine devices (IUD), and Depo-Provera, which consists of monthly injections. According to the American Sexual Health Association (ASHA), oral contraceptives are 92–97% effective in preventing pregnancy (ASHA, 2013); however they do not protect against STI and they require a prescription. The contraceptive patch delivers hormones transdermally; it is 92% effective in preventing pregnancy, does not protect against STI, and requires a prescription. The vaginal ring (e.g., NuvaRing) is 92% effective in preventing pregnancy, does not protect against STI, requires a prescription, and must be inserted and removed by a medical professional. Hormonal implants (e.g., Implanon) are inserted beneath the dermis by a medical professional; they are 99.99% effective in preventing pregnancy, but do not protect against STI (ASHA, 2013). Hormonal IUD (e.g., Mirena) are 99.99% effective in preventing pregnancy, but do not protect against STI; they require a prescription and must be inserted and removed by a medical professional. Depo-Provera is a monthly injection that is 99.7% effective in preventing pregnancy, but does not protect against STI; it requires a prescription and must be administered by a medical professional (ASHA, 2013). Overall, hormonal contraceptives are highly effective in preventing pregnancy, when used correctly and consistently; however male condoms are the only form of contraception that is also effective in preventing STI/HIV (ASHA, 2013).

For over a decade, promoting the use of multiple prevention strategies (i.e., dual protection) to reduce the risk of both unintended pregnancy and STI/HIV has been recognized as “best

practice” by medical practitioners and researchers (Workowski & Berman, 2006). Various protection strategies exist and can be combined to reduce both unintended pregnancy and STI/HIV, including hormonal contraception used in conjunction with correct and consistent condom use, STI/HIV screening of sex partner(s), decreasing number of sex partners, monogamy, and abstinence. These strategies, when used conjointly, enhance both pregnancy and STI/HIV prevention. Moreover, it is important to consider the unique social milieu within which these strategies occur, particularly regarding negotiation between sex partners, cost of the protection methods, potential side effects of the methods, acceptability, and other barriers.

Sexual Dysfunction

The Diagnostic and Statistical Manual of Mental Disorders (5th ed.; *DSM-5*; American Psychiatric Association [APA], 2013) outlines a group of disorders that are specifically related to sexual dysfunctions. These disorders are characterized by clinically significant disturbances in an individual’s ability to respond to sexual stimulation or to experience sexual pleasure. The onset and frequency of a particular disturbance are important considerations in classifying a sexual dysfunction, as this information may be helpful in determining the etiology of the sexual dysfunction and appropriate treatment interventions.

Some sexual dysfunctions are gender specific, whereas others may apply to both males and females (e.g., substance/medication-induced sexual dysfunction). Male hypoactive sexual desire disorder is a gender-specific condition marked by a lack (either persistent absence or recurrent absence) of sexual thoughts/fantasies or a deficit in desire for sexual activity. Similarly, female sexual interest/arousal disorder includes a lack of or significantly reduced sexual interest or arousal/pleasure. Male erectile disorder is indicated by the presence of at least one of the following symptoms: difficulty obtaining an erection during sexual activity, difficulty maintaining an erection during sexual activity, or a decrease in erectile rigidity. Premature ejaculation is a persistent or recurrent pattern of early (within 1 min following penetration) ejaculation during partnered sexual activity, whereas delayed ejaculation is either a significant delay in ejaculation or infrequency or absence of ejaculation. Correspondingly, female orgasmic disorder is a delay in infrequency of or absence of orgasm or reduced orgasm intensity. Genito-pelvic pain/penetration disorder (a female-specific disorder) includes one or more of the following symptoms, which generally tend to be co-occurring: (a) difficulty having intercourse, (b) genito-pelvic pain during intercourse, (c) fear of pain or vaginal penetration, and (d) tension or tightening of the pelvic floor muscles during intercourse (APA, 2013).

According to the APA (2013), healthcare and mental healthcare providers should consider several important factors when assessing a potential sexual dysfunction: (a) partner factors (e.g., partner’s sexual or health-related problems), (b) relationship factors (e.g., discord in desires, difficulties with communication), (c) individual vulnerability (e.g., body image issues, history of sexual/emotional abuse), (d) psychiatric comorbidity (e.g., depression, anxiety, PTSD), (e) other stressors (e.g., job loss, bereavement), (f) cultural or religious factors (e.g., inhibitions or prohibitions regarding sexual behaviors, attitudes toward sexuality), (g) aging (normative age-related reductions in sexual responses and physical activity), and (h) medical factors impacting prognosis, course, or treatment of a sexual dysfunction. Before diagnosing a sexual dysfunction, the possibility that symptoms result from inadequate sexual stimulation must be ruled out. Additionally, symptoms must be present in all or most sexual encounters

(75–100% of sexual encounters), for at least 6 months, and not otherwise attributable to a nonsexual mental disorder, relationship discord, other stressors, the effects of a substance/medication, or a medical condition (APA, 2013).

Restoration of sexual functioning and the experience of sexual pleasure are the focus of treatment approaches for sexual dysfunctions. Efficacious interventions for sexual dysfunctions include cognitive behavioral therapy (CBT) (e.g., stimulus control, cognitive restructuring), behavioral therapy (e.g., systematic desensitization, relaxation), psychoeducation/sexual education, and couples therapy. A lack of sexual desire may be addressed by using cognitive restructuring techniques to explore negative attitudes about sex and replace those views with new ways of thinking about sex. Arousal disorders may also be treated with cognitive restructuring (in this case targeting beliefs about sex and sexual abilities), as well as with behavioral interventions such as relaxation, systematic desensitization, and sensate focusing. Sensate focusing involves encouraging a couple to experience pleasure from both sexual and nonsexual forms of touch. Treatments for erectile dysfunction typically target sexual performance anxiety and sexual self-esteem. Behavioral treatments for premature ejaculation involve having a man learn to withstand stimulation and postpone ejaculation for increasingly longer periods of time; this approach has been shown to be particularly effective. Treatments for female orgasm disorders encourage realistic expectations about sexual pleasure and comfort with one's body and sexual desires, as well as gradual self-stimulation and relaxation exercises. Relaxation training is also used to treat painful or unpleasurable intercourse that cannot otherwise be attributed to a medical condition. Pharmacological and medical interventions are also available for sexual dysfunctions; these approaches may be used in combination with psychotherapy.

Hypersexuality

In the United States, hypersexual disorder has an estimated prevalence rate of 3–6% and is thought to occur mostly in men (Kuzma & Black, 2009). The specific behaviors that are considered hypersexual and the etiology of hypersexuality are debated upon in the literature (Brown, Gause, & Garos, 2020; Moser, 2011; Winters, 2010).

Hypersexual disorder was not included in the DSM-5 (APA, 2013); as such there are no formal clinical guidelines or diagnostic criteria for hypersexuality. However, the proposed theories of hypersexuality often include the following symptoms: a subjective loss of control; disinhibited sexual activity, which can involve culturally normative or nonnormative sexual behaviors; the presence of subjective distress; continuation of the behavior(s) in spite of escalating negative consequences; and significant impairment in areas of social, educational, occupational, or relationship functioning (Brown et al., 2020). An assessment of a potential hypersexual disorder should include a thorough sexual history, psychosocial history, psychiatric history, substance use history, relationship history, and medical history, including assessment for sexually transmitted diseases, unplanned pregnancies, abortions, and other medical conditions. In addition, it is important that the clinician assess for disorders that often co-occur with hypersexual behaviors, such as bipolar disorder, anxiety, mood disorders (Kafka & Hennen, 2002; Krueger & Kaplan, 2001; Reid & Carpenter, 2009), substance abuse, and ADHD (Krueger & Kaplan, 2001), as well as other characteristics such as impulsive behavior and emotional dysregulation (Reid, Carpenter, Spackman, & Willes, 2008; Reid, Stein, & Carpenter, 2011).

The treatment of hypersexuality is often multi-method and typically involves some form of psychotherapy. CBT is often used to identify triggers for hypersexual impulses or behaviors, manage sexual urges, and change negative, self-defeating internalized beliefs. In addition, mindfulness interventions that teach patients to focus on the present and steer patients away from worrisome or distressing thoughts (Kabat, 1990; Kingston, Dooley, Bates, Lawlor, & Malone, 2007) are efficacious (Reid, Garos, Hook, & Hook, 2014). Treatment approaches should also include a relapse prevention component and determine if the patient's behavior puts him or her at risk for occupational, legal, health, or other severe consequences. Family and couples therapy may also be part of treatment for hypersexuality, as this disorder can have devastating effects on families and intimate partnerships or marriages. Group therapy and self-help groups (e.g., Sex Addicts Anonymous) often augment treatment. Pharmacological medications have been shown to be efficacious in reducing sexual urges and obsessive sex-related thoughts. Selective serotonin reuptake inhibitors (Bradford, 2001; Coleman, Gratzner, Nesvacil, & Raymond, 2000) and antiandrogens have demonstrated promising effects in reducing sexual drive and desire (Guay, 2009). These drugs may be combined with psychotherapeutic treatments that target hypersexuality.

Paraphilic Disorders

As described in the DSM-5, a paraphilia is "...any intense and persistent sexual interest other than sexual interest in genital stimulation or preparatory fondling with phenotypically normal, physically mature, consenting human partners" (APA, 2013, p. 685). In order for a paraphilia to be considered a paraphilic disorder, it must cause an individual either distress or impairment, or be a paraphilia that entails personal harm, or risk of harm to others when or if it is satisfied (APA, 2013). Paraphilic disorders include voyeuristic disorder (sexual pleasure or excitement from spying on others in private activities), exhibitionistic disorder (exposing one's genitals), frotteuristic disorder (touching or rubbing against a non-consenting individual), sexual masochism disorder (undergoing personal humiliation, bondage, or suffering), sexual sadism disorder (inflicting humiliation, bondage, or suffering on another), pedophilic disorder (sexual attraction toward children), fetishistic disorder (sexual attraction to nonliving objects or a highly specific sexual focus on nongenital body parts), and transvestic disorder (sexual arousal from cross-dressing; APA, 2013). The sexual behaviors individuals with a particular paraphilia may engage in to fulfill their sexual urges or desires may pose potential harm or negative consequences to others, and they may even be classified as criminal offenses. Furthermore, it is not uncommon for multiple paraphilic disorders to co-occur or be comorbid with other psychological diagnoses (e.g., depressive disorders; APA, 2013).

When assessing for possible paraphilic disorders, a clinician should investigate of the specific nature of the paraphilia and potential negative consequences of the paraphilia (APA, 2013). It is important to note that individuals who report a paraphilia without negative consequences, or with the potential to harm others if/when the sexual desire is fulfilled, do not meet DSM-5 criteria for a paraphilic disorder (APA, 2013). Both clinical interviews and self-report measures may be used by clinicians to assess for possible paraphilic disorders. CBT, intensive community supervision, and adjunctive pharmaceutical treatments (Briken & Kafka, 2007) are among the available efficacious treatments for paraphilic disorders. Pharmaceutical treatments may either lower testosterone levels or treat potential comorbid psychological conditions (e.g., depressive disorders).

Sexual Behavior Engagement Across the Lifespan

Sexual activity changes across the lifespan, and sexual development varies between individuals (Crooks & Baur, 2008). Sexual behaviors that occur during childhood may include self-stimulation of the genitals or play that may be viewed as sexual in nature (e.g., “playing doctor” with other children). During adolescence and puberty secondary sex characteristics (e.g., breasts, pubic hair) develop and result in dramatic physical changes. Sexual activity typically increases during adolescence, including both self-stimulation behavior and sexual behaviors with partners. Engagement in sexual behavior as people age throughout adulthood varies. During older adulthood, engagement in sexual activities often correlates with sexual activity levels in earlier adulthood. Sexual activity during older adulthood may be adversely affected by co-occurring health conditions and physical limitations.

Acknowledgements

Support provided by R03DA0377860 from the National Institute on Drug Abuse to the first author.

Author Biographies

Jennifer L. Brown is an associate professor in the Department of Psychiatry and Behavioral Neuroscience at the University of Cincinnati. She is a licensed clinical psychologist who specializes in the development and evaluation of HIV prevention interventions. Her research aims to reduce health disparities and improve individuals’ reproductive health.

Nicole K. Gause is a clinical psychology doctoral student at the University of Cincinnati. Her research and clinical interests include sexual risk behaviors among populations that are disproportionately affected by HIV/STI, substance use among HIV-infected populations, and the etiology and treatment of substance use disorders.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th. ed.). Arlington, VA: Author.
- American Sexual Health Association. (2013). *Birth control method comparison chart*. Retrieved from <http://www.ashasexualhealth.org/pdfs/ContraceptiveOptions.pdf>
- Bradford, J. M. (2001). The neurobiology, neuropharmacology, and pharmacological treatment of the paraphilias and compulsive sexual behaviour. *Canadian Journal of Psychiatry*, 46(1), 26–34. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=mnh&AN=11221487&site=ehost-live>
- Briken, P., & Kafka, M. P. (2007). Pharmacological treatments for paraphilic patients and sexual offenders. *Current Opinion in Psychiatry*, 20(6), 609–613. doi:10.1097/YCO.0b013e3282f0eb0b
- Brown, J. L., Gause, N. K., & Garos, S. (2020). Sexual behavior. In S. R. Waldstein (Ed.), *Behavioral and social sciences in medicine*. New York, NY: Springer Publishing Company.
- Brown, J. L., Sales, J. M., & DiClemente, R. J. (2014). Combination HIV prevention interventions: The potential of integrated behavioral and biomedical approaches. *Current HIV/AIDS Reports*, 11(4), 363–375. doi:10.1007/s11904-014-0228-6

- Centers for Disease Control and Prevention. (2014a). *2013 sexually transmitted diseases surveillance: STDs in adolescents and young adults*. Atlanta, GA: Centers for Disease Control and Prevention.
- Centers for Disease Control and Prevention. (2014b). *Effective interventions: HIV prevention that works*. Retrieved from <https://effectiveinterventions.cdc.gov/>
- Centers for Disease Control and Prevention. (2015a). *HIV among gay and bisexual men*. Retrieved from <http://www.cdc.gov/hiv/group/msm/index.html>
- Centers for Disease Control and Prevention. (2015b). *HIV among hispanics/latinos*. Retrieved from <http://www.cdc.gov/hiv/group/raciaethnic/hispaniclatinos/index.html>
- Centers for Disease Control and Prevention. (2015c). *HIV in the United States: At a glance*. Retrieved from <http://www.cdc.gov/hiv/statistics/overview/ata glance.html>
- Centers for Disease Control and Prevention. (2015d). *Screening recommendations referenced in the 2015 STD treatment guidelines and original recommendation sources*. Retrieved from <https://www.cdc.gov/std/tg2015/screening-recs-2015-std-tx-guidelines.pdf>
- Centers for Disease Control and Prevention. (2016a). *HIV among African Americans*. Retrieved from <http://www.cdc.gov/hiv/group/raciaethnic/africanamericans/index.html>
- Centers for Disease Control and Prevention. (2016b). *HIV among women*. Retrieved from <http://www.cdc.gov/hiv/group/gender/women/index.html>
- Coleman, E., Gratzner, T., Nesvacil, L., & Raymond, N. C. (2000). Nefazodone and the treatment of nonparaphilic compulsive sexual behavior: A retrospective study. *The Journal of Clinical Psychiatry*, 61(4), 282–284. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=mnh&AN=10830149&site=ehost-live>
- Crooks, R., & Baur, K. (2008). *Our sexuality* (10th ed.). Pacific Grove, CA: Thomson.
- DiClemente, R. J., Wingood, G. M., Rose, E. S., Sales, J. M., Lang, D. L., Caliendo, A. M., ... Crosby, R. A. (2009). Efficacy of sexually transmitted disease/human immunodeficiency virus sexual risk-reduction intervention for african american adolescent females seeking sexual health services: A randomized controlled trial. *Archives of Pediatrics & Adolescent Medicine*, 163(12), 1112–1121. Retrieved from <https://login.proxy.library.emory.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cmedm&AN=19996048&site=ehost-live>
- Guay, D. R. P. (2009). Drug treatment of paraphilic and nonparaphilic sexual disorders. *Clinical Therapeutics: The International Peer-Reviewed Journal of Drug Therapy*, 31(1), 1–31. doi:10.1016/j.clinthera.2009.01.009
- Kabat, Z. J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain and illness*. New York, NY: Bantam Books.
- Kafka, M. P., & Hennen, J. (2002). A DSM-IV axis I comorbidity study of males ($n = 120$) with paraphilias and paraphilia-related disorders. *Sexual Abuse: Journal of Research and Treatment*, 14(4), 349–366. doi:10.1023/A:1020007004436
- Kingston, T., Dooley, B., Bates, A., Lawlor, E., & Malone, K. (2007). Mindfulness-based cognitive therapy for residual depressive symptoms. *Psychology and Psychotherapy: Theory, Research and Practice*, 80(2), 193–203. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=rzh&AN=105992288&site=ehost-live>
- Kinsey, A., Pomeroy, W., & Martin, C. (1948). *Sexual behavior in the human male*. Philadelphia, PA: Saunders.
- Krueger, R. B., & Kaplan, M. S. (2001). The paraphilic and hypersexual disorders: An overview. *Journal of Psychiatric Practice*, 7(6), 391–403. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=cmedm&AN=15990552&site=ehost-live>
- Kuzma, J. M., & Black, D. (2009). Epidemiology, prevalence, and natural history of compulsive sexual behavior. *The Psychiatric Clinics of North America*, 31(4), 603–611.G.
- Moser, C. (2011). Hypersexual disorder: Just more muddled thinking. *Archives of Sexual Behavior*, 40(2), 227–229. doi:10.1007/s10508-010-9690-4
- Peck, S. A. (2001). The importance of the sexual health history in the primary care setting. *Journal of Obstetric, Gynecologic & Neonatal Nursing*, 30(3), 269–274 266 p. doi:10.1111/j.1552-6909.2001.tb01544.x

- Reid, R. C., & Carpenter, B. N. (2009). Exploring relationships of psychopathology in hypersexual patients using the MMPI-2 (English). *Journal of Sex and Marital Therapy*, 35(4), 294–310. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=fcs&AN=21713899&site=ehost-live>
- Reid, R. C., Carpenter, B. N., Spackman, M., & Willes, D. L. (2008). Alexithymia, emotional instability, and vulnerability to stress proneness in patients seeking help for hypersexual behavior. *Journal of Sex and Marital Therapy*, 34(2), 133–149. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=fcs&AN=20137283&site=ehost-live>
- Reid, R. C., Garos, S., Hook, J. N., & Hook, J. (2014). Hypersexual disorder. In S. W. L. Grossman (Ed.), *Translating psychological research into practice* (pp. 413–418). New York, NY: Springer.
- Reid, R. C., Stein, J. A., & Carpenter, B. N. (2011). Understanding the roles of shame and neuroticism in a patient sample of hypersexual men. *The Journal of Nervous and Mental Disease*, 199(4), 263–267. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=rzh&AN=104859672&site=ehost-live>
- Winters, J. (2010). Hypersexual disorder: A more cautious approach. *Archives of Sexual Behavior*, 39(3), 594–596. doi:10.1007/s10508-010-9607-2
- Workowski, K. A., & Berman, S. M. (2006). Clinical prevention guidance. Sexually transmitted diseases treatment guidelines 2006. *Morbidity and Mortality Weekly Report*, 55(RR-11), 2–6.
- World Health Organization. (2016a). *Health topics: Reproductive health*. Retrieved from http://www.who.int/topics/reproductive_health/en/
- World Health Organization. (2016b). *Global summary of the AIDS epidemic 2015*. Retrieved from http://www.who.int/hiv/data/epi_core_2016.png?ua=1

Suggested Reading

Comprehensive Textbooks

- Lehmiller, J. J. (2013). *The psychology of human sexuality*. Oxford, UK: Wiley.
- Van den Akker, O. B. (2012). *Reproductive health psychology*. Oxford, UK: Wiley.

STI & HIV Screening Recommendations

- CDC. (2014). *STD & HIV screening recommendations*. Atlanta, GA: CDC. Retrieved from <http://www.cdc.gov/std/prevention/screeningReccs.htm>

Effective HIV Behavioral Interventions

- CDC. (2014). *Effective interventions: HIV prevention that works*. Atlanta, GA: CDC. Retrieved from <http://www.effectiveinterventions.org/en/Home.aspx>

Diagnosis of Sexual Dysfunction and Paraphilic Disorders

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Schools, Eating, and Health Psychology

Catherine Cook-Cottone and Rebecca K. Vujnovic

*Department of Psychology, University at Buffalo, The State University of New York,
Buffalo, NY, USA*

Introduction

This chapter focuses on the development of the healthy and successful student with specific attention to the development of healthy eating and physical self-care behaviors. Since the beginning of public education, school personnel have understood that in order to teach students, they must first be physically and emotionally well. It is not possible to fully achieve academic potential without a solid physical and emotional foundation to support learning. Therefore, in addition to the substantial charge of providing a strong academic education, schools must also address the physical and mental health of their students. Further, some suggest that by providing a healthy structure for physical and mental health, schools help students thrive, reaching their fullest potential in both academic and personal life. It is now well accepted that physical and mental health are deeply intertwined within a reciprocal process that either increases risk and pathology or decreases risk and promotes healing and positive development. Hence, consideration and promotion of physical health is now considered a necessary ingredient in psychological and educational interventions. With this understanding the field of health psychology has grown and along with it the notion of the *healthy school* (Cook-Cottone, Tribole, & Tylka, 2013). Research showing the negative effects of stress and the role of nutrition and physical self-care on both physical and mental health continues to grow. Accordingly, school personnel have become increasingly important in the delivery of health and health psychology interventions in schools (e.g., Cook-Cottone et al., 2013).

The school setting is the ideal place that impacts all children, delivers effective services, and promotes change from the individual to the systemic levels in a manner that can have a positive

impact on academics. Specifically in terms of health promotion, schools can help to address issues critical for enhancing resilience and health, as well as to reduce social class disparities in both physical and mental health, to enhance subjective well-being (Cook-Cottone et al., 2013; Shoshani & Steinmetz, 2013; Stephens, Markus, & Fryberg, 2012). A review of the literature suggests that subjective well-being (i.e., life satisfaction, presence of positive affect, absence of negative emotional experiences, and self-efficacy) be associated with several positive constructs, including academic achievement (Shoshani & Steinmetz, 2013). Within the school setting, models of change to promote well-being must address both the individual student characteristics and skills, as well as the larger structural level influences, such as material resources (e.g., access to healthy food, safe places to exercise, and access to quality healthcare; Stephens et al., 2012).

Consistent with the focus on system, community, and school-level factors in the promotion of health, the World Health Organization (WHO, 2015) defines a health-promoting school as a school that is constantly strengthening its capacity as a health setting, within which students can live, learn, and work. This is done by fostering health and striving to provide a healthy school environment through the provision of school services, curriculum, collaborative projects within the community, and programs for mental health promotion (WHO, 2015). It is critical to engage key stakeholders (e.g., health and education officials, teachers, parents, health providers, and community leaders) in making the school a healthy place to learn (Cook-Cottone et al., 2013; WHO, 2015). As such, schools and communities must implement policies and practices that respect individual dignity and well-being to allow many chances for success. The school, and not simply the students or the family, is striving to improve the health of all students, families, school personnel, and community members (WHO, 2015).

Specific to eating behaviors, there are several ways that students can move toward risk and disorder. The three primary clinical disorders are anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED) (American Psychiatric Association [APA], 2013). Specifically, AN is marked by a strong drive for and pursuit of thinness, body dissatisfaction, disordered body image, and behavior organized around weight loss (APA, 2013; Cook-Cottone et al., 2013). Although BN also involves dissatisfaction with the body, BN is characterized by difficulty with self-regulation that manifests in a cyclic behavioral pattern of bingeing on food and compensatory behaviors intended to either counter the caloric intake through exercise or purging (e.g., vomiting, use of laxative; APA, 2013; Cook-Cottone, 2015a). BED involves repeated binge episode in which the individual eats much more than typical in a discrete amount of time (APA, 2013). BED is also believed to involve difficulties with emotion and self-regulation (Cook-Cottone, 2015; Cook-Cottone et al., 2013). Although not considered to be a clinical eating disorder, but directly associated with weight, obesity results from a chronic disruption of the energy balance (i.e., energy intake and energy expenditure; Cook-Cottone et al., 2013; Loos & Bouchard, 2003). It is believed that current societal conditions (e.g., easy access to energy-dense foods and increased sedentary activities) among Westernized cultures have led to rapid increases in obesity rates (Cook-Cottone et al., 2013).

Although AN and BN are somewhat rare, affecting less than approximately 1–3% and 5–11% of the population, respectively (APA, 2013), rates suggest that over 20% of students may struggle at some point with clinical-level disorders associated with eating (Cook-Cottone et al., 2013). Further, if children and adolescents in the overweight category are included, *well over half* of the student population could feasibly struggle at some point with food, exercise, and self-care (Cook-Cottone, 2013). Thus, collectively, AN, BN, BED, and obesity affect a substantial number of children in schools.

The Healthy Student

The healthy student approach views each student within the context of his or her individual strengths and challenges and takes into account the influences of the family, school, community, and cultural supports and risk factors. The individual student behaviors are not seen in isolation; thus, the student's struggle for nutritional and physical health and stress management is not viewed as a failure of individual willpower or personal effort. Rather, the student, the school, and the community take ownership of their own responsibility to provide a healthy context from which the individual can make choices that serve their physical and mental health. Accordingly, the self is most effectively conceptualized as a self in which sociocultural context and individual physical and mental health characteristics both play a role in individual positive development, health, and self-regulation.

More recent models of the self-integrate physical well-being and balance the role of cognitions and emotions and the role of sociocultural context (Cook-Cottone, 2006, 2015a; Cook-Cottone et al., 2013; Stephens, et al., 2012). The attuned representational model of self (Cook-Cottone, 2006) demonstrates the role of the internal aspects of self such as cognitive, emotional, and the physical self. The physical self is viewed as a base for emotional and cognitive well-being. For example, daily nutrition, hydration, and exercise provide a stable physiological framework for emotional stability and positive cognitive functioning. See Cook-Cottone et al. (2013) and Cook-Cottone (2015b) for a detailed description of the role of self-care practices in well-being.

When self-care is intact, the aspects of the internal system work in an attuned manner to serve the functioning of the self within the sociocultural context (Cook-Cottone, 2006, 2015a). Basic structural conditions present within the student's sociocultural contexts (e.g., access to healthy food, school educational practices, school resources and funding, community and larger cultural values and priorities) as well as family influences also play a role in student well-being and performance (Cook-Cottone, 2006; Stephens et al., 2012). These processes are viewed as reciprocal, as the student's ability to engage in active self-care of his or her physical, emotional, and cognitive needs supports the student's ability to manage the challenges presented by sociocultural context. Conversely, the practices, supports, and tools provided within and by those in the sociocultural context (e.g., school program and practices) can enhance the students development of self-care and self-regulation tools (Cook-Cottone, 2006; Cook-Cottone et al., 2013). The self functions to witness and manage the internal aspects of self within the context of the external aspects of self. The self that presents to the world, school friend, teachers, and family members is an aggregation of the ongoing attunement or aggregated risk manifest by the ongoing dynamics within the self-system (Cook-Cottone, 2006; Cook-Cottone et al., 2013). See Cook-Cottone (2006) or Cook-Cottone (2015) for a full description of the model.

In order to increase understanding regarding school health and well-being promotion within the school setting, the history and foundations of the three-tier approach to school-based interventions are described. Next, health promotion approaches, including the promotion of healthy eating and positive mind and body relationships, will be described within the context of the three-tier approach in schools. To illustrate the approach, mind-body and eating-related interventions will be presented as examples of service provision within the three-tier model.

Health Psychology and the History of the Three-Tier Model

The three-tier model of prevention became a classification system pertinent to the field of prevention and public health, highlighting the importance of promoting and maintaining

public health, rather than solely focusing on the prevention of disease (Leavell & Clark, 1953, 1958, 1965). The first tier became known as the primary prevention level, intended for the promotion of health and well-being and to provide specific protection against onset of disease. The primary prevention level included the implementation of procedures (i.e., immunizations, genetic screening) to identify potential risk and to intercept such risk prior to infection of the disease. Subsequently, secondary prevention was intended for early detection and timely treatment to prevent infection from spreading to others or to limit prolonged disability in the individual. Finally, the tertiary level of prevention was considered for the treatment of symptoms, rehabilitation, and limitations of the consequence of the disease to prevent progression or premature death (Leavell & Clark, 1953, 1958, 1965). Since its establishment, the fields of preventative health, medicine, and the social sciences have adopted this framework, although definitions of these three categories have varied across disciplines (Machlem, 2013). By the 1990s, the field of prevention adopted the terminologies endorsed by the Institute of Medicine (IOM): universal, selective, and indicated (Strein, Kuhn-McKearin, & Finney, 2014).

At the same time, the field of education increasingly embraced the prevention model framework for several reasons, including the increased development and research documenting the efficacy of evidence-based interventions, the promotion of well-being, and the increasing levels of need among students along with the recognition of the limitation of resources and cost-effectiveness. Further, such a multitier model accommodated recommendations outlined by the National Association of School Psychologists (NASP, 2010) that recommended a ratio of one school psychologist to every 500–700 students, depending on the need within the student population (NASP, 2010). This tremendously high ratio of students to school psychologists highlights the necessity of using a prevention model for the provision of services, as direct service to treat all individuals in need of services would be impossible.

Applied within the school framework, the three-tier model is incredibly effective for providing services to all students, especially as school personnel are frequently the first line of defense against emerging eating disorders and health problems related to obesity (Cook-Cottone et al., 2013). The first of the three tiers is Tier 1, the universal level, and is conceptualized as the core instructional or social-behavioral program. Tier 1 programs are research supported and aim to have an impact on the majority of students by meeting the needs of 80–90% of students. Accordingly, the majority of students, including those who may be at risk for concerns, respond sufficiently to this level of programming (Stoiber, 2014). Tier 2 is the targeted level of supports that are intended to serve a smaller group of students who require increased support to enhance the core programs at the Tier 1 level (Stoiber, 2014). Tier 3 is the intensive level and is the highest level of intensity. Tier 3 is designed to be individualized and strategically aligned with the needs of the individual student being serviced. This level is intended to be implemented for the smallest percentage of students, 1–5% of students who continue to require supports beyond those in the first two tiers (Stoiber, 2014).

Tier 1: Approaches for the Promotion of Health and Well-Being

Tier 1 approaches for health and well-being promotion are interventions at the universal level. In general, they are core instructional or social-behavioral programs that are implemented for all students within the school and likely to be sufficient in meeting the needs of the majority of students (Stoiber, 2014). There is a long-standing history in the provision of universal programs in education that illustrate the link between physical health and learning. For example, school lunch programs were among the first interventions provided within the school

setting to address a foundational health need that was notably interfering with student learning (Cook-Cottone et al., 2013). Since first initiated, school-based programs to promote nutrition and physical health have grown substantially. These programs have developed to include large-scale federally funded school lunch programs, integrate physical and health education, and target all levels of well-being support from counseling services to family support. Current implementation issues hinge on funding, challenges related to scheduling and time constraints, and finding evidence-based programs that meet school needs.

Beyond the provision of school lunches, there is emerging evidence that universal practices designed to promote well-being and health are effective in schools (Cook-Cottone et al., 2013; Shoshani & Steinmetz, 2013). However, research is in the early stages; much of the evidence is based on pilot studies or short-term controlled trials as opposed to more rigorous randomized trials. At the universal level, there is a significant need for higher-quality research with randomized controlled trials and assessment of long-term outcomes (Serwacki & Cook-Cottone, 2012; Shoshani & Steinmetz, 2013). A review of available research related to universal programs has identified several key factors across programs that were believed to play a role in student well-being at the universal level, including the cultivation of positive emotions, gratitude, and hope, goal setting, and development of character strengths (Shoshani & Steinmetz, 2013). These foci can easily be woven into daily curriculum, although some schools integrate such themes more formally through structured social emotional learning (SEL) programs. Overall research on SEL programs indicates that they result in an increase in student well-being (Ashdown & Bernard, 2012; Durlack, Weissberg, Dynamicki, Taylor, & Schellinger, 2011). Benefits include increased emotional regulation, self-awareness, stress management, relationship skills, and social awareness and have demonstrated an impact on learning (Ashdown & Bernard, 2012; Durlack et al., 2011). These larger-scale, often school- and district-wide approaches provide a foundational context for more specific programs that focus on healthy eating and physical health promotion.

Effective implementation of school and district-wide interventions requires an organization around a set of principles, which facilitates communication and aligns efforts. Cook-Cottone et al. (2013) present an approach to the *healthy student*, which includes three essential components: (a) intuitive eating and nutrition, (b) healthy physical activity, and (c) mindfulness, self-care, and emotion regulation. It is important that the overall focus is on promoting health, rather than avoiding obesity (Cook-Cottone et al., 2013). To do this schools must adopt a *transcontextual approach* in which adults within schools, families, and the community work together to provide a healthy environment (Cook-Cottone et al., 2013). This approach helps the entire community “walk the walk” of healthy eating, physical exercise, and mindful self-care. The entire school community is encouraged to embrace a focus on overall health over weight and weight loss. This community must also create a zero-tolerance policy for teasing and bullying, especially related to weight. Additionally, students, teachers, and families should be provided with opportunities to learn about nutrition. Nutrition education should also be included in health class and other appropriate sections of the curriculum. Farm-to-school programs and school farm program can enhance student knowledge of food sourcing, agriculture, botany, and nutrition. Further, through collaboration between school lunch providers and food vendors, the school should provide an environment full of healthy food choices with minimal exposure to unhealthy, competing foods (i.e., foods that have little nutritional value and are offered as a purchasing option during meal times, outside of, or as part of the school meal program; Cook-Cottone et al., 2013). Finally, opportunities for physical activity and access to water throughout the school day are also critical for supporting and promoting overall health (Cook-Cottone et al., 2013).

One approach that can be applied at the universal level and is aligned with the positive psychology paradigm is the Health at Every Size (HAES) program. The HAES program is based on supporting a fundamental understanding of the body, suggesting that an individual is more likely to treat his or her body with respect and care, as well as provide his or her body with good nutrition and exercise, if an individual likes their body (Mukai, Kambara, & Sasaki, 2013). Research suggests that when the body is experienced with a sense of shame, an individual may be more likely to engage in self-neglect and self-destructive eating behaviors and is less likely to engage in physical exercise, nutrition, and self-care (e.g., Duarte, Pinto-Gouveia, & Ferreira, 2014). The HAES principles include (a) a respect and acceptance of the body (all body types, shapes, and sizes), (b) a focus on making physical activity enjoyable, (c) a decreased focus on food (i.e., not using food as an external reward for achievement or behavior or as a way of celebrating achievement), and (d) a decreased focus on weight and body shaming. These HAES general principles can be readily applied to the school setting (Cook-Cottone et al., 2013). For example, there is no need to weigh students in school when they are routinely monitored by their primary healthcare provider or during their individual school physical (Cook-Cottone et al., 2013). For some students, the school weighing day is the beginning of their eating disorder story. This policy has been criticized not only for the risk it induces but also for the frequent lack of connection with any intervention to address variations in body mass index. Finally, schools should help students focus on being present, active, and living fully in their present body no matter its shape or size (Cook-Cottone et al., 2013). There are several programs that have been shown effective in promoting healthy eating behavior and a positive relationship with the body: Planet Health (Austin et al., 2012; Gortmaker et al., 1999), New Moves (Neumark-Sztainer et al., 2010), Healthy Buddies (Stock et al., 2007), and Learning Gardens.

It is important to note that mindfulness and mindful self-care strategies are also central to the healthy student approach and schools are increasingly integrating them into their daily classroom practices and social and emotional learning programs. Mindful practice and self-care are ways of being present in your mind and body that allows for a shift from externally oriented stimuli and standards to an ongoing presence with your own experience. Specifically, mindfulness includes formal mindful practices such as meditation, yoga, and systematic relaxation, as well as an increase in mindful awareness throughout the day. For example, a student could eat lunch, practice his or her instrument, do math, and read mindfully. Although more rigorous research is needed, mindful practices, yoga, and mindful awareness are believed to reduce risk for eating disorder behavior and increase student well-being (Cook-Cottone et al., 2013; Greenberg & Harris, 2012; Scime & Cook-Cottone, 2008; Serwacki & Cook-Cottone, 2012). For an expanded review of school-wide interventions, see Cook-Cottone et al. (2013). Overall, within the context of honoring and respecting bodies of all shapes and sizes, the district-wide focus is on respect for the body and the body's wisdom to know when and what to eat, healthy physical exercise, and mindful practice and self-care.

Tier 2: Approaches to Facilitate Healthy Eating Behaviors and Self-Care

Tier 2 approaches are targeted level supports that are intended to serve a smaller group of students who require increased support to enhance the core programs at the Tier 1 level programs (Stoiber, 2014). Tier 2 programs often target a specific risk group (e.g., elite athletes) or students already showing risk factors (e.g., dieting, body dissatisfaction) without manifesting full diagnostic criteria. There are many different types of programs that range from those that

focus on physical exercise, or are solely focused on nutrition and eating or mood regulation and distress tolerance, to integrated programs that include both yoga and didactic skills training (e.g., media literacy, assertiveness, emotion regulation; see Cook-Cottone et al. (2013) for a review of school-based programs).

Here, two examples are provided as illustrations of a Tier 2 approach. First, Martinsen et al. (2014) designed and studied an eating disorder prevention intervention for elite high school athletes that addressed self-confidence, motivation, growth and development, and sports nutrition as related to health, eating disorders, and performance. Results indicated that those in the intervention group showed no onset of clinical eating disorder, whereas control group members showed significant increases in eating disordered behavior. Other programs have focused on students who are referred to the intervention due to suspect increased risk. Programs such as *Girls Growing in Wellness and Balance: Yoga and Life Skills to Empower* use yoga, and a risk-based curriculum can be used as a Tier 2 intervention for girls at risk for eating disorders. This program has been found, in controlled trials, to reduce body dissatisfaction, increase self-care, and decreased drive for thinness (Cook-Cottone, Kane, Keddie, & Haugli, 2013; Scime & Cook-Cottone, 2008; Serwacki & Cook-Cottone, 2012). However, there is still much work needed before there is a strong evidence base supporting a variety of Tier 2 interventions. Specifically relevant for school settings are issues such as dosage of the intervention may matter. For example, how much yoga, mindfulness, or prevention curriculum is needed to create change (Cook-Cottone, 2013)? Notably, dissonance-based programs in which students are led through a process of media literacy, psychoeducation on the thin ideal, activities designed to help students internalize a healthy body weight perspective and counter the thin ideal have also found to be effective (Rohde et al., 2014).

Tier 3: Assessing and Supporting Students With Eating Problems

Once a student is showing symptoms of a disorder, there should be considered for a Tier 3 level of intervention. Tier 3 is the highest level of intensity and is designed to be intensive, individualized, and strategically aligned with the student's individual needs (Stoiber, 2014). Screening and assessment can help identify those students who are in need of more intensive interventions. The SCOFF is a brief five-item screening instrument that can be very helpful. The questions are the following: (S) "Do you make yourself sick (vomit) because you feel comfortably full?" (C) "Do you worry that you have lost control over how much you eat?" (O) "Have you recently lost more than one stone (i.e., 15 pounds) in a 3-month period?" (F) "Do you believe yourself to be fat when others say you are thin?" (F) "Would you say the food dominates your life?" (Solmi, Hatch, Hotopf, Treasure, & Micali, 2015) Other behaviors of concern include eating in the absence of hunger, emotional eating, body dissatisfaction, and distortions in or over concern with body image (Cook-Cottone et al., 2013). For a more detailed review of screening and assessment tools for disordered eating, refer to Cook-Cottone et al. (2013), Berg and Peterson (2013), and Palmer (2014).

Following screening and assessment, if it is determined that the student is at risk for disordered eating, a referral should be made to an eating disorder specialist. Treatment may be done either on an inpatient or outpatient basis depending on the level of symptomatology and the patient's health status (Cook-Cottone et al., 2013). Those with clinical-level eating disorders require comprehensive and multifaceted care (American Academy of Pediatrics [AAP], 2003). The treatment of disordered eating utilizes a multidisciplinary team that specializes in the treatment of eating issues that includes a medical doctor specializing in eating disorders, a

mental health specialist, and a nutritionist (Cook-Cottone et al., 2013). See Cook-Cottone and Vujanovic (2015) for a review of evidence-based interventions for children and adolescents with eating disorders.

Although treatment is likely to occur outside of the school setting, adequate support in the school setting can be a critical part of the treatment and recovery process. Support of the treatment of disordered eating can be most effectively done within the context of a prevention-oriented school atmosphere already promoting zero tolerance of in-school advertising, body teasing, and encouraging healthy nutritional behaviors and opportunities for positive physical activity (e.g., soccer, yoga classes, track, swimming; Cook-Cottone et al., 2013). If a student requires hospitalization or day treatment, providing support for the student's transition back to school is vital to recovery maintenance (Manley, Rickson, & Standeven, 2000). Student needs may include supportive counseling, medical monitoring, release from physical education classes, meal monitoring, and communication with the treatment team and family (Cook-Cottone et al., 2013). Special academic accommodations may be necessary, which include adjusted workload, alternative assignments for some physical education requirements, extended time on assignments and tests, tutoring for missed coursework, copies of class notes, and access to quiet study locations (Cook-Cottone et al., 2013; Manley et al., 2000). Given the medical complications sometimes involved in more chronic or severe cases, the school nurse may play an important role (Cook-Cottone et al., 2013). In-school counseling can nicely augment outpatient treatment team efforts via relaxation work, supportive and reflective listening, and short-term, solution-focused or problem-solving approaches for in-school issues. In addition, the treatment team therapist may have specific objectives with which he or she would like supported within the school setting (Cook-Cottone et al., 2013). Other areas of school support for those with disordered eating include supporting student during meals, alternative assignments in physical education and health classes, and flexibility and support with academic assignments and attendance (Cook-Cottone et al., 2013).

Conclusion

Physical and mental health provides the foundation for student learning. Today, schools have increasing responsibility for the provision of education beyond academics and are now charged with supporting overall well-being, not only for their students but also for families, school staff, and members of the larger community. One important deterrent to health is the increasing prevalence of eating disorders and obesity, with over half of the student population potentially struggling with food, exercise, and self-care. Addressing well-being and health in the school setting is best conceptualized in a three-tier approach, focusing not on the avoidance of the disorder itself, but on providing a level of programming to all students to support health and wellness within the school community. At the Tier 1 universal level, programs target all students and are usually implemented at the larger school or district-wide levels, focusing on healthy eating and physical health promotion. At the Tier 2 selective level, programs are intended to be implemented only to a smaller group of students and often target a specific risk group or students already showing risk factors. At this level, programming is varied and focuses on the students' needs. Thus, programs for use at Tier 2 might include those that focus on physical exercise, nutrition and eating, or mood regulation and distress tolerance. Tier 2 programs with a more broad focus integrate both yoga and didactic skills training to support well-being. Finally, at the Tier 3 indicated level, treatment is likely to occur outside of the school setting, making coordination and support for treatment in the school setting essential.

Students may require accommodations to support treatment progress and maintenance, and communication with treatment providers is important. Overall, schools provide an important setting for supporting health and well-being in the school and community.

Author Biographies

Catherine Cook-Cottone, PhD, is a licensed psychologist, registered yoga teacher, professor at SUNY at Buffalo, and co-editor in chief of *Eating Disorders: The Journal of treatment and Prevention*. She is founder and president of Yogis in Service, Inc., a not-for-profit organization. Her research specializes in embodied self-regulation (i.e., yoga, mindfulness, and self-care) and psychosocial disorders (e.g., eating disorders, trauma). She has written 6 books and over 60 peer-reviewed articles and book chapters.

Rebecca K. Vujnovic, PhD, is a licensed psychologist and nationally certified school psychologist. She is director of the University at Buffalo, State University of New York School Psychology Program. She maintains a private practice specializing in youth with attention and anxiety disorders. She has authored a workbook to support the management of anxiety through mindfulness strategies anxiety and has published 4 book chapters and 12 research articles.

References

- American Academy of Pediatrics. (2003). Identifying and treating eating disorders: Policy statement by the Committee on Adolescence. *Pediatrics*, 111, 204–211.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Ashdown, D. M., & Bernard, M. (2012). Can explicit instruction in social emotional learning skills benefit the social emotional learning, well being, and academic achievement of young children? *Early Childhood Education*, 39, 397–405.
- Austin, S. B., Spadano-Gasbarro, J. L., Greaney, M. L., Blood, E. A., Hunt, A. T., Richmond, T. K., ... Peterson, K. E. (2012). Peer reviewed: Effect of the planet health intervention on eating disorder symptoms in Massachusetts Middle Schools, 2005–2008. *Preventing Chronic Disease*, 9, E171.
- Commission on Chronic Illness: Chronic Illness in the United States, vol. 1. Published for the Commonwealth Fund by Harvard University Press, Cambridge, MA, 1957
- Cook-Cottone, C., & Vujnovic, R. K. (2015). Evidence-based interventions for eating disorders in children and adolescents. In L. Theodore (Ed.), *Handbook of applied interventions for children and adolescents*. New York, NY: Springer.
- Cook-Cottone, C. P. (2006). The attuned representation model for the primary prevention of eating disorders: An overview for school psychologists. *Psychology in the Schools*, 43, 223–230.
- Cook-Cottone, C. P. (2013). Dosage as a critical variable in yoga therapy research. *International Journal of Yoga Therapy*, 23, 11–12.
- Cook-Cottone, C. P. (2015a). Incorporating positive body image into the treatment of eating disorders: A model for attunement and mindful self-care. *Body Image*, 14, 158–167.
- Cook-Cottone, C. P. (2015b). *Mindfulness and yoga for self-regulation: A primer for mental health professionals*. New York, NY: Springer.
- Cook-Cottone, C. P., Kane, L., Keddle, E., & Haugli, S. (2013). *Girls growing in wellness and balance: Yoga and life skills to empower*. Stoddard, WI: Schoolhouse Educational Services, LLC.
- Cook-Cottone, C. P., Tribble, E., & Tylka, T. (2013). *Healthy eating in schools: Evidence-based interventions to help kids thrive*. Washington, DC: American Psychological Association.
- Duarte, C., Pinto-Gouveia, J., & Ferreira, C. (2014). Escaping from body image shame and harsh self-criticism: Exploration of underlying mechanisms of binge eating. *Eating Behaviors*, 15, 638–643.

- Durlack, J. A., Weissberg, R. P., Dynamicki, A. B., Taylor, R. D., & Schellinger, K. B. (2011). The impact of enhancing students' social and emotional learning: A meta-analysis of school-based universal interventions. *Child Development*, 82, 405–532.
- Gortmaker, S. L., Peterson, K., Wiecha, J., Sobol, A. M., Dixit, S., Fox, M. K., & Laird, N. (1999). Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Archives of Pediatrics & Adolescent Medicine*, 153, 409–418.
- Greenberg, M. T., & Harris, A. R. (2012). Nurturing mindfulness in children and youth: Current state of research. *Child Development Perspectives*, 6, 161–166.
- Leavell, H. R., & Clark, E. G. (1953). *Textbook of preventive medicine*. New York, NY: McGraw-Hill Book Company.
- Leavell, H. R., & Clark, E. G. (1958). *Preventive medicine for the doctor in his community* (2nd ed.). New York, NY: McGraw-Hill Book Company.
- Leavell, H. R., & Clark, E. G. (1965). *Preventive medicine for the doctor in his community* (3rd ed.). New York, NY: McGraw-Hill Book Company.
- Loos, R., & Bouchard, C. (2003). Obesity—Is it a genetic disorder? *Journal of Internal Medicine*, 254, 401–425.
- Machlem, G. L. (2013). *Preventative mental health at school: Evidence-based services for students*. New York, NY: Springer.
- Manley, R. S., Rickson, H., & Standeven, B. (2000). Children and adolescents with eating disorders: Strategies for teachers and school counselors. *Intervention in School and Clinic*, 35, 228–231.
- Martinsen, M., Bahr, R., Børresen, R. U. N. I., Holme, I., Pensgaard, A. M., & Sundgot-Borgen, J. (2014). Preventing eating disorders among young elite athletes: A randomized controlled trial. *Medicine & Science in Sports & Exercise*, 46, 435–447.
- Mukai, T., Kambara, A., & Sasaki, Y. (2013). Health at every size approach. *Eating Disorders: An Encyclopedia of Causes, Treatment, and Prevention*, 231.
- NASP. (2010). *Integrated model for comprehensive and integrated school psychological services*. Washington, DC: NASP.
- Neumark-Sztainer, D. R., Friend, S. E., Flattum, C. F., Hannan, P. J., Story, M. T., Bauer, K. W., ... Petrich, C. A. (2010). New moves—Preventing weight-related problems in adolescent girls: A group-randomized study. *American Journal of Preventive Medicine*, 39, 421–432.
- Palmer, B. (2014). *Helping people with eating disorders: A clinical guide to assessment and treatment*. New York, NY: Wiley.
- Rohde, P., Auslander, B. A., Shaw, H., Raineri, K. M., Gau, J. M., & Stice, E. (2014). Dissonance-based prevention of eating disorder risk factors in middle school girls: Results from two pilot trials. *International Journal of Eating Disorders*, 47, 483–494.
- Scime, M., & Cook-Cottone, C. (2008). Primary prevention of eating disorders: A constructivist integration of mind and body strategies. *International Journal of Eating Disorders*, 41, 134–142.
- Serwacki, M., & Cook-Cottone, C. (2012). Yoga in the schools: A systematic review of the literature. *International Journal of Yoga Therapy*, 22, 101–110.
- Shoshani, A., & Steinmetz, S. (2013). Positive psychology at school: A school-based intervention to promote adolescents' mental health and well being. *Journal of Happiness Studies*, 15, 1289–1311.
- Solmi, F., Hatch, S. L., Hotopf, M., Treasure, J., & Micali, N. (2015). Validation of the SCOFF questionnaire for eating disorders in a multiethnic general population sample. *International Journal of Eating Disorders*, 48, 312–316.
- Stephens, N. M., Markus, H. R., & Fryberg, S. A. (2012). Social class disparities in health and education: Reducing inequality by applying a sociocultural self model of behavior. *Psychological Review*, 119, 723–744.
- Stock, S., Miranda, C., Evans, S., Plessis, S., Ridley, J., Yeh, S., & Chanoine, J. P. (2007). Healthy Buddies: A novel, peer-led health promotion program for the prevention of obesity and eating disorders in children in elementary school. *Pediatrics*, 120, e1059–e1068.
- Stoiber, K. C. (2014). A comprehensive framework for multitiered system of support in school psychology. In A. Thomas & J. Grimes (Eds.), *Best practices in school psychology* (5th ed.). Bethesda, MD: NASP.

- Strein, W., Kuhn-McKearin, M., & Finney, M. (2014). *Best practices in developing prevention strategies for school psychology practice*.
- World Health Organization (WHO). (2015). *What is a health promoting school?*. Retrieved from http://www.who.int/school_youth_health/gshi/hps/en/.

Suggested Reading

- Cook-Cottone, C. P. (2015). *Mindfulness and yoga for self-regulation: A primer for mental health professionals*. New York, NY: Springer.
- Cook-Cottone, C. P. (2017). *Mindfulness and yoga in schools: A guide for teachers and practitioners*. New York, NY: Springer.
- Cook-Cottone, C. P., Tribble, E., & Tylka, T. (2013). *Healthy eating in schools: Evidence-based interventions to help kids thrive*. Washington, DC: American Psychological Association.

Sport and Exercise in Health Psychology

Erika D. Van Dyke^{1,2} and Judy L. Van Raalte²

¹*Department of Sport Sciences, College of Physical Activity and Sport Sciences,
West Virginia University, Morgantown, WV, USA*

²*Department of Psychology, Springfield College, Springfield, MA, USA*

Sport and exercise psychology is a field devoted to understanding biological, psychological, and social influences on performance and well-being, as well as the ways in which these forces interact to influence participation in sport and exercise (e.g., Biddle, 2000). Scientific findings and practical applications derived from sport and exercise psychology are of direct relevance to health psychology. That is, sport, exercise, and physical activity contribute to health outcomes. Understanding the factors that influence participation in sport, exercise, and physical activity can facilitate the development of effective interventions. Important health-related factors may also be considered concurrently with sport, exercise, and physical activity.

Physical Activity, Sport, and Exercise as a Cause

Much of the health psychology literature pertaining to sport and exercise is focused on the ways in which participation in physical activity influences aspects of health and well-being. Particular attention has been paid to the effects of sport and exercise on cognition, mood, personal factors, and psychobiological processes. A body of literature indicates that fit older adults display superior cognitive functioning relative to their less fit counterparts. These results are dependent on the task, with attention-demanding and rapid tasks showing the most pronounced effects (Boutcher, 2000). Meta-analyses further show that physical activity enhances cognition among both healthy elderly people and those with cognitive impairment and dementia (Colcombe & Kramer, 2003). Such cognitive benefits may accrue to exercisers because engaging in regular aerobic exercise minimizes age-related declines in brain volume and increases brain volume (Colcombe et al., 2006). Among the elderly, physical activity is associated with larger gray

matter volumes and reduced brain volume losses due to neurodegeneration (Raji et al., 2016). Because the specific brain regions positively affected by physical activity are also related to Alzheimer's disease and mild cognitive impairment, physical activity is believed to reduce the risk of developing such cognitive deficits (Raji et al., 2016). Further research is needed to clarify the dose-response relationship between physical activity and cognition, as well as to discover the underlying mechanisms by which physical activity exerts its effects on cognitive functioning (Boutcher, 2000; Carron, Hausenblas, & Estabrooks, 2003).

People who engage in regular physical activity often report that exercise makes them feel good (Leith, 2010). Indeed, research indicates that exercise reduces anxiety, depression, and confusion as measured by the Profile of Mood States (POMS) (McNair, Lorr, & Droppleman, 1971). Given these findings, it is not surprising that there has been a proliferation of research on the influence of physical activity behaviors on mood and affect particularly in the areas of anxiety and depression.

The state anxiety-reducing effects of physical activity are clear (the relationship between physical activity and trait anxiety is addressed below with other personality factors). Review papers report low to moderate reductions in anxiety following bouts of physical activity (Leith, 2010). Exercising for at least 15 min can reduce state anxiety for several hours post-exercise (Leith, 2010). The anxiety-reducing effects of physical activity have most commonly been found for aerobic, rhythmic activities including running, walking, cycling, and swimming (Leith, 2010). Both aerobic and non-aerobic forms of exercise reduce anxiety symptoms (Jayakody, Gunadasa, & Hosker, 2014). Although much research indicates that reductions in anxiety are found following physical activity regardless of the intensity of the exercise (Leith, 2010), some research with competitive athletes and exercisers has shown that high-intensity physical training is associated with increases in anxiety and negative affective states (Szabo, 2000). Exercise prescription recommendations may therefore be best focused on low- to moderate-intensity physical activity for anxiety reduction.

Physical activity is as effective as other known treatments (e.g., hypnosis, meditation, and relaxation) in reducing anxiety (Carron et al., 2003). Among individuals with clinically defined anxiety disorders including social phobia and panic disorder, exercise can reduce symptoms of anxiety and offers additional benefits compared with medication or psychological treatment alone (Jayakody et al., 2014). Similar positive effects of physical activity have been found with youth sport athletes who experience lower levels of anxiety in social situations (Schumacher Dimech & Seiler, 2011). Taken together, various sport and exercise behaviors have been found effective in reducing state anxiety.

Mild forms of nonclinical depression often abate without intervention in a relatively short amount of time. When treatment is sought, medication and psychotherapy are traditional methods that can alleviate symptoms of depression (Leith, 2010). Several meta-analyses dating back to the 1990s show that exercise reduces depression in individuals relative to non-exercising controls (North, McCullagh, & Tran, 1990), that these benefits accrue from both aerobic and anaerobic exercise, and that exercise is as effective a treatment for depression as other established methods including forms of psychotherapy and relaxation (North et al., 1990). Antidepressant effects have been most widely found in exercise programs of at least 10 weeks in length, with bouts of moderate-intensity activity performed for 15–30 min three times per week (Leith, 2010). Although high-intensity exercise has also been shown to effectively reduce symptoms of depression (Leith, 2010), the potential for individuals to experience negative affective responses at high intensities may make moderate-intensity activity a preferred approach for participants (Ekkekakis, Parfitt, & Petruzzello, 2011). Given the relatively few adverse side effects of exercise relative to the many health benefits,

these findings have encouraging implications for including exercise in treatment prescriptions for depression.

Personality is a complex phenomenon—one that is relatively stable and enduring but also dynamic and modifiable (Carron et al., 2003). That is, people's personalities include characteristics displayed across a broad range of situations, as well as some characteristics that are malleable and subject to change. Physical activity has been found to be related to aspects of personality including trait anxiety, body image, and self-esteem.

Regular participation in sport and aerobic exercises (e.g., jogging, walking, cycling, swimming, and aerobic dance) is related to significant reductions in trait anxiety. People who engage in exercise programs of at least 9 weeks typically experience reductions in trait anxiety regardless of the exercise intensity level tested (Leith, 2010). A minimum of 20 min of exercise performed three times per week is necessary to promote a positive influence on trait anxiety (Leith, 2010). Thus, physical activity is associated with favorable effects on both state and trait forms of anxiety.

Body image has also been found to be positively related to involvement in sport and exercise (Hausenblas & Fallon, 2006; Hausenblas & Symons Downs, 2001). For instance, exercisers tend to have more favorable body image than non-exercisers, and body image is thought to improve throughout the course of exercise programs (Hausenblas & Fallon, 2006). When considering the body image of athletes in particular, athletes report more positive body image than non-athletes, and college athletes' body image is more positive than club/recreational athletes (Hausenblas & Symons Downs, 2001). However, involvement in sports that emphasize highly trained and lean bodies may contribute to body image disturbances (Carter, 2009). Additional research exploring the relationship between physical activity and body image may provide a strong foundation for the development of evidence-based interventions designed to reduce body image disturbances (Hausenblas & Symons Downs, 2001).

Physical activity has been shown to have a positive influence on self-esteem via such mechanisms as enhanced body image, body satisfaction, body acceptance, physical competence, and personal control over the body (Fox, 2000). Weight training and aerobic exercise programs of at least 12 weeks have the strongest self-esteem enhancing benefits (Fox, 2000; Leith, 2010). Physical self-esteem (Bowker, 2006) and perceived sport competence (Wagnsson, Lindwall, & Gustafsson, 2014) mediate the relationship between adolescent sport participation and self-esteem. Adult involvement in competitive physical activity can have adverse effects on self-esteem and self-concept (Leith, 2010), but such effects may be contingent upon the population in question. Competitive sport athletes report significantly higher self-esteem and self-concept than non-athletes. Among adolescent competitive sport athletes, competitive orientation is a positive predictor of self-esteem and self-concept (Findlay & Bowker, 2009). In sum, sport and exercise have been shown to positively influence self-esteem and self-concept, both directly and via the mediating roles of physical competence and body image-related constructs.

Psychobiological factors such as sleep, reactivity to stress, pain, and injury have all been explored in terms of their relationships to physical activity. Acute exercise has small to moderate effects on slow-wave sleep, rapid eye movement (REM) sleep, REM latency, and total sleep time (Youngstedt, O'Connor, & Dishman, 1997). With regard to the timing of the exercise bout relative to sleep, the majority of studies are conducted with good sleepers and include exercise bouts that are completed at least four hours before the onset of sleep, thus making it difficult to explore hypotheses related to the disruptive effects of exercise on sleep. It should be noted, however, that some individuals involved in physical activity just 30 min prior to bedtime do not experience sleep disruption (Youngstedt et al., 1997). These results suggest

that evenings may serve as an ideal time for individuals with later preferences to exercise (Youngstedt, 2000).

Exercise and physical activity reduce reactivity to psychosocial stressors (Crews & Landers, 1987). Aerobically fit individuals often have reduced reactivity to psychosocial stress relative to their unfit counterparts (Crews & Landers, 1987). Exercise can be an effective way to cope with stress or can serve as an “inoculator” whereby repeated exposure to exercise enhances the ability to handle psychosocial stressors. Students who regularly use sport and exercise as coping strategies tend to have high stress tolerance (Bland, Melton, Bigham, & Welle, 2014). These results highlight the value of physical activity, exercise, and sport as effective coping mechanisms for reducing stress reactivity.

Pain is a common, and for some activities an integral, part of engaging in sport and exercise (Carron et al., 2003). Interestingly, exercise also has pain-reducing effects, although the specific mechanisms by which exercise achieves these analgesic effects are not fully understood (Koltyn, 2000). Aerobic and strengthening exercises reduce musculoskeletal pain tied to labor, menstruation, fibromyalgia, low back issues, and forms of arthritis (Carron et al., 2003). Pain tolerance and pain threshold increase following acute bouts of exercise, and ratings of the intensity of a pain stimulus decrease following exercise (Koltyn, 2000). Elite athletes have higher pain thresholds than non-athletes and control participants (Koltyn, 2000). Anecdotal evidence suggests that athletes and dancers may continue to perform vigorous exercise while injured and report afterward that they did not experience pain (Koltyn, 2000). Such accounts highlight how pain perception can be altered by engaging in sport and exercise. Parallel processing models of pain suggest that sport and exercise activities can serve a distraction function, keeping attention directed toward task-relevant stimuli and away from pain (Brewer & Redmond, 2017).

A common cost associated with participation in sport and exercise is injury. Cognitive emotional and behavioral responses to sport injury have been documented in the literature (Brewer & Redmond, 2017). Cognitive appraisals range from relatively benign (e.g., “my body can use a rest anyway”) to very distressed (e.g., “this is the worst thing that ever happened to me”). Negative emotional responses typically occur immediately post injury, including experiences of anger, anxiety, bitterness, confusion, depression, disappointment, devastation, fear, frustration, helplessness, resentment, and shock. Effective coping behaviors and adherence to sport injury rehabilitation can speed the process of recovery from sport injury (Brewer & Redmond, 2017). Positive emotions as well as mixed feelings of apprehension, excitement, and fear of reinjury often occur as return to sport and physical activity near. Although sport, exercise, and physical activity can lead to injury, physical activity behaviors, particularly forms of strength training, have also been associated with lowered risk of injury in both youth and adults (Leppänen, Aaltonen Parkkari, Heinonen, & Kujala, 2014; Rössler et al., 2014). Thus, strength and exercise-based activities can be effective in preventing the occurrence of injury in sport and exercise settings.

Physical Activity, Sport, and Exercise as an Effect

Social factors such as group cohesion, leadership, group size, and social support have been studied with a particular focus on their effects on participation in sport, exercise, and physical activity. Group cohesion has been explored as a primary factor in individual adherence to physical activity. Perceptions of group cohesion are associated with individual adherence across several different physical activity behaviors including group fitness classes, recreational team

sports, and elite team sports (Carron, Widmeyer, & Brawley, 1988). Perceptions of cohesion are higher among regular exercise class attendees than among dropouts in both university fitness class and private club fitness class settings (Spink & Carron, 1994). Although the specific aspects of group cohesion and moderators (e.g., belonging, enjoyment) that are related to individual adherence vary across settings, the overall conclusions highlight the value of cohesion in sports teams and exercise groups for fostering adherence to physical activity (Carron et al., 1988).

In the sport domain, coaches and peers high in transformational leadership qualities have positive influences on competitive team sport athletes (Price & Weiss, 2013). Specifically, transformational leadership behaviors such as inspirational motivation, idealized influence-individualized consideration, and intellectual stimulation are linked to greater team cohesion, enjoyment, confidence, perceived competence, and intrinsic motivation, variables likely to have a positive impact on athletes' participation in sport (Price & Weiss, 2013). In exercise settings, however, individual intentions to participate in fitness classes are not primarily dependent upon leadership style. Rather, leadership style contributes to group environment, and this interaction favorably influences enjoyment of exercise (Fox, Rejeski, & Gauvin, 2000). Thus, leadership characteristics exert influence at both individual and group levels to impact the experiences people have in physical activity.

In the context of recreational sport, increasing team size has been found to decrease perceptions of both enjoyment and cohesion (Widmeyer, Brawley, & Carron, 1990). In exercise settings, there is a curvilinear relationship between group size and attendance/retention such that small and large classes are best attended (Carron, Brawley, & Widmeyer, 1990). Exercisers' satisfaction is related to class size in a linear fashion such that the larger the class, the less satisfaction, perhaps due to reductions in individualized attention, reinforcement, and instruction from group leaders in larger groups (Carron et al., 1990).

Exercisers' perceptions of social support from their families and important others are associated with exercise behavior, cognitions pertaining to exercise participation, and attitudes regarding exercise experiences (Carron, Hausenblas, & Mack, 1996). Structural equation modeling has shown that social support has an indirect influence on exercise behavior that is mediated by self-efficacy (Duncan & Stoolmiller, 1993). That is, social support enhances self-efficacy, which is in turn related to increases in exercise behavior. Social support is also a key influence on the sport-related behaviors of youth, adolescents, and adults (Beets, Cardinal, & Alderman, 2010; Johnson, Kubik, & McMorris, 2011; Santi, Bruton, Pietrantonio, & Mellalieu, 2014). Direct parental involvement in sport activities with children, parental presence or supervision of sport activities, and parental encouragement and praise of children's physical activity behaviors all foster youth physical activity (Beets et al., 2010). Social support from friends and school staff are associated with increased participation in sports teams among adolescents (Johnson et al., 2011). Supportive coaches and teammates increase participation in masters teams and nonobligatory training sessions (Santi et al., 2014). There is strong evidence that social support is positively related to physical activity, exercise, and sport involvement across the lifespan.

Physical Activity, Sport, and Exercise and Concurrent Factors

Several factors such as exercise dependence, eating disorders, and substance use issues, as well as individual demographic/biological and psychological characteristics, have been found to occur concurrently with sport and exercise behavior. Individuals experiencing exercise

dependence exercise to such an extent that they may sustain injury, feel controlled by their physical activity, and experience a range of problematic physiological, cognitive, and affective symptoms when deprived of physical activity (Szabo, 2000). Symptoms of exercise deprivation experienced by exercise-dependent people include confusion, impaired concentration, anxiety, depression, irritability, hostility, anger, tension, guilt, frustration, decreased self-esteem, muscle soreness, disturbed sleep, lethargy, fatigue, increased galvanic skin response, gastrointestinal problems, and decreased vigor (Carron et al., 2003). The specific amount of exercise that is “too much” and leads to exercise dependence has yet to be determined. Factors associated with the development of exercise dependence include high levels of strenuous exercise, perfectionism, and self-efficacy (Hausenblas & Symons Downs, 2002). Clarification of conceptual and definitional issues related to exercise dependence may allow researchers to gain a deeper understanding of this phenomenon.

High levels of athleticism and exercise have been found to co-occur with eating disorder symptoms (Szabo, 2000). Athletes, particularly male athletes, report more eating disorder symptoms than non-athletes (Hausenblas & Carron, 1999). Male and female athletes in aesthetic sports and in sports involving weight categories are at heightened risk of developing eating disorder symptoms (Szabo, 2000). The subjective evaluation inherent to aesthetic sport outcomes and the heightened attention toward maintaining a certain body appearance and/or weight are considered probable explanations for these relationships (Hausenblas & Carron, 1999). Among female collegiate athletes, symptoms of eating disorders are related to sociocultural pressure for thinness, negative appraisals of athletic achievement, and anxiety about athletic performance (Williamson et al., 1995). Physical activity in and of itself is not necessarily related to eating disorders (Szabo, 2000). Indeed, walking exercise regimens are effective in managing binge eating disorders among obese women (Levine, Marcus, & Moulton, 1996). Furthermore, anorexia often leads to fatigue that can necessitate reductions in physical activity (Carron et al., 2003). Only a small percentage of exercisers appear to experience eating disorder symptoms (Szabo, 2000).

Exercise is associated with a number of positive health outcomes. Physical activity is often incorporated into alcohol treatment programs, and exercise training is effective in helping to alleviate alcohol dependency (Gutgesell & Canterbury, 1999). Additionally, exercise has been found to significantly increase abstinence rates, ease withdrawal symptoms, and reduce symptoms of anxiety and depression among individuals with substance use disorders (Wang, Wang, Wang, Li, & Zhou, 2014). When exploring substance abuse among high school and college athletes, however, participation on sports teams is associated with higher use of alcohol but lower use of cigarettes and illegal drugs than non-athletes (Lisha & Sussman, 2010). Thus, sport participation may serve a protective function against the use of cigarettes and illegal drugs.

With regard to performance-enhancing substances, anabolic-androgenic steroid use is particularly salient in the context of sport and exercise. Athletes are more likely to use anabolic-androgenic steroids than non-athletes (Sagoe, Molde, Andreassen, Torsheim, & Pallesen, 2014). Both male gender and involvement in power sports are predictive of future anabolic-androgenic steroid use (Wichstrøm, 2006). Given the known adverse health effects of using anabolic-androgenic steroids (Momaya, Fawal, & Estes, 2015), this is an area within sport and exercise warranting continued attention. Although engaging in sport and exercise may provide a protective function relative to the use of some substances, participation in physical activity is also predictive of substance use, particularly the use of alcohol and anabolic-androgenic steroids.

Several noteworthy trends have been identified connecting demographic factors and participation in sport and exercise. Physical activity tends to decline with advancing age across the lifespan (Carron et al., 2003). From preschool-age children to older adults, males typically

engage in more moderate to vigorous physical activity than females. This trend may be partially due to sociocultural differences. Ethnic differences also exist relative to sedentary lifestyles, with 54% of Hispanic Americans, 52% of Black or African Americans, 46% of American Indians, 42% of Asians or Pacific Islanders, and 36% of White Americans being sedentary (USDHHS, 2000). Regarding occupation trends, blue-collar workers are less inclined to engage in leisure time physical activity than white-collar workers, potentially due to the impression that job-related tasks are sufficient for health benefits or to the physical fatigue associated with blue-collar jobs. Education is positively associated with leisure time physical activity, and levels of parental education are also positively related to the physical activity of their children. Healthy individuals tend to be more active than people with physical and mental health conditions (Carron et al., 2003).

Frequently reported barriers to participating in physical activity include lack of time, lack of access to facilities, and lack of safe environments (Carron et al., 2003). People who have more positive attitudes toward exercise and who have strong intentions to exercise are more likely to actually engage in physical activity behaviors. In addition, people tend to do the things that they enjoy. Thus, those who enjoy participating in sport and exercise are likely to be motivated to engage in these activities. Although the majority of people know that physical activity is good for health, many people are still not regularly active. This identified gap between knowledge and behavior indicates that interventions aiming to enhance physical activity levels should focus on more than the provision of knowledge regarding the health benefits of physical activity (Carron et al., 2003). Affective responses to exercise may be an area toward which focus might be appropriately placed given the notion that people are more likely to engage in physical activity if the experience is enjoyable (Ekkekakis et al., 2011).

Involvement in physical activity, sport, and exercise is related to biological, psychological, and social factors as well as overall health and well-being. Biological, psychological, and social factors also interact to influence participation in various physical activity behaviors. Engaging in physical activity, sport, and exercise may therefore be viewed as a cause of health outcomes, as an effect of the context and social environment, and as co-occurring with other personal health factors. Thus, a mounting body of evidence irrevocably ties sport and exercise to health psychology and highlights the importance of physical activity for maintaining physical and mental health across the lifespan.

Author Biographies

Erika D. Van Dyke received her master of science degree in athletic counseling from Springfield College and is currently a doctoral student of sport and exercise psychology at West Virginia University. She is a certified group exercise instructor through the Athletics and Fitness Association of America and is a member of both the Association for Applied Sport Psychology and the American Psychological Association (APA) Society for Sport, Exercise and Performance Psychology (Division 47). Erika has coauthored a book chapter on supervision for applied sport psychology consultants in training and has presented at both national and international conferences. In 2016, she received the APA Division 47 Master's Thesis Award for her research exploring self-talk and competitive performance among collegiate gymnasts. During her first year of doctoral study, she was granted a Provost Fellowship based on her scholarship and presently serves as an instructor and success coach for students at the university.

Judy L. Van Raalte, PhD, is professor of psychology at Springfield College, is certified consultant, Association for Applied Sport Psychology, and is listed in the US Olympic

Committee Sport Psychology Registry. Dr. Van Raalte has presented at conferences in 18 countries and published over 100 articles in peer-reviewed journals. Dr. Van Raalte served as president of the American Psychological Association's Division of Exercise and Sport Psychology (Division 47) and as the vice president of the International Society of Sport Psychology. She is a fellow of the American Psychological Association and the Association for Applied Sport Psychology.

References

- Beets, M. W., Cardinal, B. J., & Alderman, B. L. (2010). Parental social support and the physical activity-related behaviors of youth: A review. *Health Education & Behavior*, 37, 621–644.
- Biddle, S. J. (2000). Emotion, mood and physical activity. In S. J. Biddle, K. R. Fox, & S. H. Boutcher (Eds.), *Physical activity and psychological well-being* (pp. 63–87). London, UK: Routledge.
- Bland, H. W., Melton, B. F., Bigham, L. E., & Welle, P. D. (2014). Quantifying the impact of physical activity on stress tolerance in college students. *College Student Journal*, 48, 559–568.
- Boutcher, S. H. (2000). Cognitive performance, fitness, and ageing. In S. J. Biddle, K. R. Fox, & S. H. Boutcher (Eds.), *Physical activity and psychological well-being* (pp. 118–129). London, UK: Routledge.
- Bowker, A. (2006). The relationship between sports participation and self-esteem during early adolescence. *Canadian Journal of Behavioural Science*, 38, 214–229.
- Brewer, B. W., & Redmond, C. (2017). *Psychology of sport injury*. Champaign, IL: Human Kinetics.
- Carron, A. V., Brawley, L. R., & Widmeyer, W. N. (1990). The impact of group size in an exercise setting. *Journal of Sport & Exercise Psychology*, 12, 376–387.
- Carron, A. V., Hausenblas, H. A., & Estabrooks, P. A. (2003). *The psychology of physical activity*. New York, NY: McGraw-Hill.
- Carron, A. V., Hausenblas, H. A., & Mack, D. (1996). Social influence and exercise: A meta-analysis. *Journal of Sport & Exercise Psychology*, 18, 1–16.
- Carron, A. V., Widmeyer, W. N., & Brawley, L. R. (1988). Group cohesion and individual adherence to physical activity. *Journal of Sport & Exercise Psychology*, 10, 127–138.
- Carter, J. E. (2009). Disordered eating in college student-athletes. In E. F. Etzel (Ed.), *Counseling and psychological services for college student-athletes* (pp. 303–322). Morgantown, WV: Fitness Information Technology.
- Colcombe, S., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychological Science*, 14, 125–130.
- Colcombe, S. J., Erickson, K. I., Scalf, P. E., Kim, J. S., Prakash, R., McAuley, E., ... Kramer, A. F. (2006). Aerobic exercise training increases brain volume in aging humans. *Journal of Gerontology: Medical Sciences*, 61A, 1166–1170.
- Crews, D. J., & Landers, D. M. (1987). A meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. *Medicine & Science in Sports & Exercise*, 19, 114–120.
- Duncan, T. E., & Stoolmiller, M. (1993). Modeling social and psychological determinants of exercise behaviors via structural equation systems. *Research Quarterly for Exercise and Sport*, 64, 1–16.
- Ekkekakis, P., Parfitt, G., & Petruzzello, S. I. (2011). The pleasure and displeasure people feel when they exercise at different intensities: Decennial update and progress towards a Tripartite Rationale for Exercise Intensity Prescription. *Sports Medicine*, 41, 641–671.
- Findlay, L. C., & Bowker, A. (2009). The link between competitive sport participation and self-concept in early adolescence: A consideration of gender and sport orientation. *Journal of Youth and Adolescence*, 38, 29–40.
- Fox, K. R. (2000). The effects of exercise on self-perceptions and self-esteem. In S. J. H. Biddle, K. R. Fox, & S. H. Boutcher (Eds.), *Physical activity and psychological well-being* (pp. 88–117). London, UK: Routledge.

- Fox, L., Rejeski, W., & Gauvin, L. (2000). Effects of leadership style and group dynamics on enjoyment of physical activity. *American Journal of Health Promotion, 14*, 277–283.
- Gutgesell, M., & Canterbury, R. (1999). Alcohol usage in sport and exercise. *Addiction Biology, 4*, 373–383.
- Hausenblas, H. A., & Carron, A. V. (1999). Eating disorder indices and athletes: An integration. *Journal of Sport & Exercise Psychology, 21*, 230–258.
- Hausenblas, H. A., & Fallon, E. A. (2006). Exercise and body image: A meta-analysis. *Psychology & Health, 21*, 33–47.
- Hausenblas, H. A., & Symons Downs, D. (2001). Comparison of body image between athletes and non-athletes: A meta-analytic review. *Journal of Applied Sport Psychology, 13*, 323–339.
- Hausenblas, H. A., & Symons Downs, D. (2002). How much is too much? The development and validation of the Exercise Dependence Scale. *Psychology & Health, 17*, 387–404.
- Jayakody, K., Gunadasa, S., & Hosker, C. (2014). Exercise for anxiety disorders: Systematic review. *British Journal of Sports Medicine, 48*, 187–196.
- Johnson, K. E., Kubik, M. Y., & McMorris, B. J. (2011). Prevalence and social-environmental correlates of sports team participation among alternative high school students. *Journal of Physical Activity & Health, 8*, 606–612.
- Koltyn, K. (2000). Analgesia following exercise: A review. *Sports Medicine, 29*, 85–98.
- Leith, L. M. (2010). *Foundations of exercise and mental health* (2nd ed.). Morgantown, WV: West Virginia University.
- Leppänen, M., Aaltonen, S., Parkkari, J., Heinonen, A., & Kujala, U. (2014). Interventions to prevent sports related injuries: A systematic review and meta-analysis of randomised controlled trials. *Sports Medicine, 44*, 473–486.
- Levine, M. D., Marcus, M. D., & Moulton, P. (1996). Exercise in the treatment of binge eating disorder. *The International Journal of Eating Disorders, 19*, 171–177.
- Lisha, N. E., & Sussman, S. (2010). Relationship of high school and college sports participation with alcohol, tobacco, and illicit drug use: A review. *Addictive Behaviors, 35*, 399–407.
- McNair, D. M., Lorr, M., & Droppleman, L. F. (1971). *Manual for the profile of mood states*. San Diego, CA: Educational and Industrial Testing Service.
- Momaya, A., Fawal, M., & Estes, R. (2015). Performance-enhancing substances in sports: A review of the literature. *Sports Medicine, 45*, 517–531.
- North, T., McCullagh, P., & Tran, Z. (1990). Effect of exercise on depression. *Exercise & Sport Sciences Reviews, 18*, 379–415.
- Price, M. S., & Weiss, M. R. (2013). Relationships among coach Leadership, peer leadership, and adolescent athletes' psychosocial and team outcomes: A test of transformational leadership theory. *Journal of Applied Sport Psychology, 25*, 265–279.
- Raji, C. A., Merrill, D. A., Eyre, H., Mallam, S., Torosyan, N., Erickson, K. I., ... Kuller, L. H. (2016). Longitudinal relationships between caloric expenditure and gray matter in the cardiovascular health study. *Journal of Alzheimer's Disease, 52*(2), 719–729. doi:10.3233/JAD-160057
- Rössler, R., Donath, L., Verhagen, E., Junge, A., Schweizer, T., & Faude, O. (2014). Exercise-based injury prevention in child and adolescent sport: A systematic review and meta-analysis. *Sports Medicine, 44*, 1733–1748.
- Sagoe, D., Molde, H., Andreassen, C. S., Torsheim, T., & Pallesen, S. (2014). The global epidemiology of anabolic-androgenic steroid use: A meta-analysis and meta-regression analysis. *Annals of Epidemiology, 24*, 383–398. doi:10.1016/j.annepidem.2014.01.009
- Santi, G., Bruton, A., Pietrantonio, L., & Mellalieu, S. (2014). Sport commitment and participation in masters swimmers: The influence of coach and teammates. *European Journal of Sport Science, 14*, 852–860.
- Schumacher Dimech, A., & Seiler, R. (2011). Extra-curricular sport participation: A potential buffer against social anxiety symptoms in primary school children. *Psychology of Sport & Exercise, 12*, 347–354.
- Spink, K. S., & Carron, A. V. (1994). Group cohesion effects in exercise classes. *Small Group Research, 25*, 26–42. doi:10.1177/1046496494251003

- Szabo, A. (2000). Physical activity as a source of psychological dysfunction. In S. J. H. Biddle, K. R. Fox, & S. H. Boutcher (Eds.), *Physical activity and psychological well-being* (pp. 130–153). London, UK: Routledge.
- Wagnsson, S., Lindwall, M., & Gustafsson, H. (2014). Participation in organized sport and self-esteem across adolescence: The mediating role of perceived sport competence. *Journal of Sport & Exercise Psychology*, 36, 584–594.
- Wang, D., Wang, Y., Wang, Y., Li, R., & Zhou, C. (2014). Impact of physical exercise on substance use disorders: A meta-analysis. *PLoS One*, 9, 1–15. doi:10.1371/journal.pone.0110728
- Wichstrøm, L. (2006). Predictors of future anabolic androgenic steroid use. *Medicine & Science in Sports & Exercise*, 38, 1578–1583.
- Widmeyer, W. N., Brawley, L. R., & Carron, A. V. (1990). The effects of group size in sport. *Journal of Sport & Exercise Psychology*, 12, 177–190.
- Williamson, D. A., Netemeyer, R. G., Jackman, L. P., Anderson, D. A., Funsch, C. L., & Rabalais, J. Y. (1995). Structural equation modeling of risk factors for the development of eating disorder symptoms in female athletes. *The International Journal of Eating Disorders*, 17, 387–393.
- Youngstedt, S. D. (2000). The exercise-sleep mystery. *International Journal of Sport Psychology*, 31, 241–255.
- Youngstedt, S. D., O'Connor, P. J., & Dishman, R. K. (1997). The effects of acute exercise on sleep: A quantitative synthesis. *Sleep: Journal of Sleep Research & Sleep Medicine*, 20, 203–214.
- USDHHS. (2000). United States Department of Health and Human Services, Washington, DC, USA.

Sexual Minority Populations and Health

Mike C. Parent¹ and Teresa Gobble²

¹*Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA*

²*Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA*

Introduction

Sexual orientation and gender minority health has been a topic of interest for health researchers for decades. Sexual orientation minorities are persons whose sexual behaviors or attractions are not exclusively heterosexual. The term LGB (meaning lesbian, gay, and bisexual; the order of letters may vary) has often been used to describe such persons, but there is increasing recognition that this term does not capture a full range of potential manifestations of human sexual expression. For example, some individuals may engage in sexual behaviors with individuals of the same gender exclusively, or with both men and women, yet identify as heterosexual. *Sexual orientation minority* is used as a broad term, to include persons with non-heterosexual behaviors, attractions, and identities. However, it is important to note that differences exist within this group, including differences in health disparities. *Gender minorities* are persons for whom a gender assigned at birth does not align with a felt sense of gender identity. A commonly used term for individuals in this group is transgender (in contrast to cisgender, or persons for whom gender assigned at birth and felt sense of gender identity match), though the more broad term of gender minority may also include other gender identities that persons may identify as, including non-binary gender identities. Again, it is important to note that differences exist within the gender minorities group (e.g., between persons who are transitioning from female to male or from male to female and also among persons who desire different degrees of transition). Further, although sexual orientation minority persons and gender minority persons share many of the same concerns, they also have many differences between the groups that are relevant to health (Donatone & Rachlin, 2013; Moradi, Mohr, Worthington, & Fassinger, 2009).

The Context of Sexual Minority Health Disparities

Research on health disparities among sexual orientation and gender minority persons, until relatively recently, has tended to focus on non-heterosexual and non-cisgender identities as themselves reflective of pathology. That is, the locus of health disparities was often placed within sexual orientation and gender minority individuals themselves. In recent decades, awareness has shifted away from viewing sexual orientation and gender minority persons as intrinsically pathologized to understanding the social context of living as a sexual orientation or gender minority individual. In light of this, research has increasingly turned toward understanding person–environment interactions and the ways in which one’s circumstances may exacerbate typical stressors or present unique stressors for sexual orientation and gender minority persons. The leading theory of the influence of social structures on the well-being of sexual orientation and gender minority persons is the *minority stress model* (Meyer, 1995, 2003).

The minority stress model describes the ways in which psychological distress is perpetrated by unique, chronic, and socially based stressors for sexual orientation and gender minority persons. It was initially developed to describe how heterosexism, or behavior that grants preferential treatment to heterosexual people, reinforces the idea that heterosexuality is preferred or more highly valued over minority sexual orientations. Heterosexism is also associated with homophobia or biphobia, which includes a variety of negative attitudes (e.g., anger, fear, discomfort, resentment, or disgust) that an individual may have toward sexual minority persons. Heterosexism or homo/biphobia may include behaviors such as discrimination against sexual orientation minority persons, for example, in employment or housing. Indeed, in much of the world and including in many parts of the United States, sexual orientation and gender minority persons may face hiring or promotion discrimination, in some cases without legal recourse for such discrimination. Heterosexism may also include experiences of violence inflicted upon an individual due to actual or perceived status as a sexual orientation or gender minority. Such experiences of violence can begin early, such as in school bullying, and persist into adulthood, such as in experiences of workplace harassment. Heterosexism and homo/biphobia may also be experienced at a social level. For example, laws may be passed that uniquely impact the well-being of sexual and gender minority persons. Experiences of violence may also take on a range of manifestations but may include experiences such as being assaulted. Consistent exposure to homophobic or biphobic messages can lead to internalization of those messages, meaning that sexual orientation minority persons may feel similar feelings of fear, anger, discomfort, resentment, or disgust for themselves and other sexual orientation or gender minority persons. Although initially developed to understand the experiences of sexual orientation minorities, the model was quickly applied to gender minority populations and proved useful in research on the health of gender minorities (Bockting, Miner, Swinburne Romine, Hamilton, & Coleman, 2013).

The minority stress model conceptualizes these and related experiences of stress as aspects of minority stress. Stress in general is a chronic engagement of taxing coping mechanisms to the extent that the individual does not have the capacity to endure further, leading to mental health and medical concerns. Social stress in particular describes the effect that social environmental events and conditions, above and beyond personal events, can have a deleterious effect on individuals. The minority stress model further emphasizes that the specific stressors faced by sexual orientation and gender minority persons are unique, chronic, and socially based. The stresses are unique, because they are based upon actual or perceived sexual orientation identity. That is, they are not experienced by persons who are, or who are perceived to be, heterosexual or cisgender. The stressors are chronic because they are not reflective of

merely random and isolated events, but rather reflect a pervasive negative social attitude that promotes their reoccurrence across time, location, and context. Relatedly, the stressors are socially based because they are based in dominant social views within a society, which are socially constructed and maintained. It is important to note that the minority stress model emphasizes ways in which multiple minority identities can serve as both protective and risk factors for behaviors and experiences related to health disparities. For example, sources of stress and health concerns may differ significantly between gay men and lesbian women, or for sexual orientation and gender minority people of color compared with White sexual orientation and gender minority individuals. Indeed, the minority stress model has been applied several times to understand the experiences of ethnically and racially diverse populations since its conception in 1995 (Meyer, 1995).

The minority stress framework has been instrumental in helping to understand health disparities within their context. Numerous health disparities exist for sexual orientation and gender minority populations. Such disparities are known to begin early, being demonstrated even among adolescents who identify as sexual orientation and gender minorities. Sexual orientation and gender minority youth report higher levels of emotional distress and depressive symptoms than their heterosexual and cisgender counterparts. These symptoms are also associated with elevated levels of suicidal ideation and suicide attempts, as well as self-harm behaviors. Further, these disparities have been replicated across multiple countries, across racial/ethnic groups, and across genders. Transgender youth appear to be at specific and elevated risk for self-harm (McConnell, Birkett, & Mustanski, 2015; Peterson, Matthews, Copps-Smith, & Conard, 2017). Among adults, these health disparities persist and are linked to other negative outcomes for sexual orientation and gender minority individuals.

In minority stress theory, stresses are differentiated based on whether they arise from outside or within an individual. Outside stresses include circumstances in the environment. Such circumstances speak to the intersectional interactions of multiple minority statuses in considering minority stress. One clear example of circumstances in the environment is poverty. Poverty would impact factors such as access to basic needs such as healthcare, access to food (i.e., in a food desert), access to transportation, or safe housing. Unfortunately, limited empirical research has been conducted on the intersection of sexual orientation and gender minority identity and poverty, from within the minority stress framework. Nevertheless, sexual orientation and gender minority persons, especially adolescents, face levels of poverty and homelessness far higher than their heterosexual and cisgender peers (Keuroghlian, Shtasel, & Bassuk, 2014). Other circumstances in the environment may also depend on the specific circumstances of the individuals involved; for example, in considering a same-sex couple who wish to adopt, specific state laws regarding same-sex adoption may be an especially relevant circumstance in the environment. Such laws may ebb and flow in their relevance to individuals; for example, if a same-sex couple has adopted a child in a state in which adoption laws were being called into question, they may worry about the status of their legal rights as adoptive parents. Similarly, such stresses may arise of sexual orientation or gender minority persons when the legality of same-sex marriage is called into question. In prior research with sexual orientation minority persons, in states in which constitutional amendments to define marriage as between a man and a woman were on the ballot, sexual orientation minority persons reported higher exposure to negative media, higher negative affect, and higher levels of distress compared with persons in states where such laws were not on the ballot (Fingerhut, Riggle, & Rostosky, 2011). Myriad other circumstances in the environment may ultimately impact the well-being of sexual orientation and gender minority persons, including persons with multiple minority statuses or other relevant personal characteristics.

Circumstances in the environment are inextricably tied to minority statuses. There are innumerable personal characteristics that a minority of persons may possess, but do not represent socially stigmatized identities (e.g., having green eyes or completely attached earlobes). Minority stress theory focuses on stresses that arise due to social influence, and thus categories of stigmatized identities are socially defined and may intersect. For example, recent research has explored ways in which sexual orientation and gender or race may intersect. Such investigations have indicated that there may be some pervasive mental health issues that cut across different groups when faced with minority stress (e.g., psychological distress or depression, substance use) but also some specific concerns that may be especially relevant to specific groups. For example, obesity, obesogenic behaviors, and resultant health conditions (e.g., cardiovascular disease) appear to be a specific concern relevant to lesbian and bisexual women, use of club drugs or anabolic steroids may be relevant to gay and bisexual men, and risk behaviors for HIV or other sexually transmitted infections (STIs) may be relevant to men who have sex with men. Thus, specific minority identities, and the interactions among identities, may impact both which circumstances in the environment are relevant and potential outcomes of minority stress processes.

Minority Identity

Because minority stress theory focuses on socially defined identities, a concept similar to minority status is minority identity and its characteristics. Individuals may differ in the extent to which their identities align with the categories into which researchers or others may desire to put them. For example, the term “men who have sex with men” was coined, in part, to capture the experiences of men who have sex with men but who may not identify as gay or bisexual. Minority identity itself is closely tied to the characteristics of minority identity, such as prominence of that identity or integration of that identity into everyday life. Such processes have implications for the health of sexual orientation and gender minority populations. For example, among men who have sex with men, the desire to be perceived by others as heterosexual has been associated with decreased likelihood of asking for an HIV test when visiting a doctor (Parent, Torrey, & Michaels, 2012). Other investigations have linked integration of sexual minority identity with lower internalized heterosexism and lower psychological distress. At the same time, outness has also been associated with more experiences of heterosexism, which may exacerbate minority stress processes. In addition, outness has not always clearly been linked to health variables, with some studies finding no relationship between outness and instances of domestic violence among lesbian and bisexual women (Balsam & Szymanski, 2005; Velez, Moradi, & Brewster, 2013). Thus, the relationships among outness or other variables related to identity salience are not straightforward. Indeed, it is possible that in many circumstances, not being out may have some costs to personal well-being but may have substantial benefits as well, for example, if coming out meant a risk for being physically assaulted by family members or kicked out of one’s home.

Minority identity is also associated with stress processes. In minority stress theory, these processes are conceptualized as distal (arising directly from the behaviors of others, such as harassment or discrimination) or proximal (arising directly from within oneself, such as expectations of rejection or internalized homophobia). These proximal and distal stressors have been reliably associated with myriad health concerns for sexual orientation and gender minority persons (Meyer, 2003).

Finally, the minority stress model posits that coping and social support may moderate health outcomes for sexual orientation and gender minority persons. Notably, however, research has not always supported the posited roles of these variables. Some work has supported links between aspects of coping, such as spiritual coping, as a buffer between minority stressors and mental health outcome variables, such as substance use. Other work has demonstrated that emotional regulation mediates the relationship between minority status and depression and anxiety among sexual orientation minority adolescents (but also that sexual orientation minority adolescents demonstrate lower levels of emotional regulation than their heterosexual peers). However, other recent work has called into question the utility of concepts such as “coping” or “resilience” among minority persons who may face social or institutional discrimination, inasmuch as those terms may refer to returns to baselines of emotional or cognitive content following experiences of minority stress. By conceptualizing such returns to baseline following experiences of harassment, discrimination, or violence, the impetus for change is placed on an individual experiencing such harassment, discrimination, or violence to deal with the implications of those experiences, rather than emphasizing the need for change in the social or institutional power structures that allow or even encourage those experiences. Experiences of posttraumatic growth, transformative coping, or personal growth following minority stress may be fruitful areas to explore and may lead to a more clear understanding of the relationship between minority stresses and adaptive responses (Meyer, 2015). Regarding social support, a number of studies have supported social support as a buffer of the relationship between experiences of minority stress and negative outcomes. At the same time, involvement with the LGBT community is not always synonymous with social support and has indeed, in some cases, been linked with negative health outcomes for sexual orientation and gender minority persons. For example, more often attending clubs and parties has been linked to increased body image concerns and substance use among gay and bisexual men (Mattison, Ross, Wolfson, & Franklin, 2001).

Minority Stressors and Health Disparities

Experiences of bullying appear to be ubiquitous among sexual orientation and gender minority youth, with nearly all reporting some level of bullying or harassment. More severe forms of bullying including persistent bullying, threats of violence, or actual violence, are also common. Some of these instances of violence can result in physical harm, and sexual orientation and gender minority youth report that often these instances are either not reported to school officials or law enforcement, and if they are, that nothing is done about the behavior. Both harassment and violence are known to persist into adulthood, with sexual orientation and gender minority youth reporting workplace harassment and discrimination in housing or employment. Some disparities appear to be particularly relevant for specific groups within the sexual and gender minority umbrella. In particular, transgender persons may be especially vulnerable to experiences of violence (Berlan, Corliss, Field, Goodman, & Austin, 2010; Kim & Leventhal, 2011). As well, sexual orientation and gender minority persons who are sex workers are also at elevated risk for exposure to violence, again especially including transgender persons and especially male-to-female transgender persons (Nemoto, Bödeker, & Iwamoto, 2011).

Use of substances is also elevated among sexual orientation and gender minority persons, and this finding has been replicated in several countries. Rates of use of all substances are

elevated among gender and sexual orientation minority adolescents, though rates of hard drugs and club drugs are markedly elevated, and rates of smoking tobacco are also markedly higher among sexual orientation and gender minority adolescents compared with their cisgender counterparts. This elevation is occurring concurrently with marked drops in smoking behaviors in the general population. Regarding substance use in general, among adolescents, girls appear to have somewhat higher risk for substances than boys, and bisexual adolescents appear to be at somewhat greater risk than gay/lesbian adolescents. Gay and bisexual adolescent boys also appear to be at specific risk for use of anabolic-androgenic steroids. Among adults, rates of substance use remain elevated, including use of tobacco, alcohol, steroids, recreational drugs, and club drugs. Use of such substances may be related to attempts to cope with stresses such as discrimination and violence but also potentially linked to aspects of community engagement. For example, use of illicit substances is common in clubs and circuit parties (large dance parties). As well, use of steroids may facilitate development of a highly muscular body, which facilitates access to a larger number of potential sexual partners (and, potentially, increases risk for exposure to STIs). Among female-to-male transgender persons, use of steroids may also facilitate development of a highly masculinized physique, potentially increasing “passing” as male and decreasing risks associated with being perceived as transgender (Guss, Williams, Reisner, Austin, & Katz-Wise, 2017; Halkitis, Moeller, & DeRaleau, 2008; Lee, Matthews, McCullen, & Melvin, 2014; Marshal et al., 2008).

Sexual risk behaviors are also relevant to sexual orientation and gender minority individuals. Most research on sexual health behaviors has focused on boys and men and potential contraction or spread of STIs. Within this domain, health disparities have been found to emerge early. Adolescent gay and bisexual boys report greater risky sexual behaviors than their heterosexual counterparts, with some studies finding bisexual adolescent boys to be at particular risk. These risks persist into adulthood for gay and bisexual men and appear to be exacerbated by use of substances and engaging in sex while intoxicated. Most research in this domain focuses on risk for HIV infection, and HIV rates remain elevated among gay and bisexual men. Although use of pre-exposure prophylaxis (PrEP) (i.e., medication taken to drastically lower the risk of HIV infection should exposure occur) is increasingly common among men who have sex with men, use of PrEP does not protect individuals from other STIs, and rates of STIs among men who have sex with men on PrEP remain elevated (Rosario et al., 2013; Scott & Klausner, 2016).

Obesity is also a concern among sexual orientation and gender minority persons. This disparity appears to be most prevalent among lesbian and bisexual women and appears to arise relatively early in life as adolescent girls who identify as lesbian and bisexual report higher rates of obesity compared with heterosexual peers. Obesity, and engagement in obesogenic behaviors, may interact with other stressors such as unemployment or underemployment, further complicating health status. However, obesity is not markedly elevated among gay and bisexual men or among transgender persons. The precise cause of specific health disparities among sexual orientation minority women is not known. Indeed, predictors of overweight status and obesity among sexual orientation minority women and heterosexual women are similar (Bowen, Balsam, & Ender, 2008; Yancey, Cochran, Corliss, & Mays, 2003).

Gender minority persons may also face specific health concerns. Some, though not all, transgender persons may seek to undertake aspects of gender transition. Gender transition may involve numerous surgical and nonsurgical procedures. Gender transition processes may be social and legal changes, such as legal adoption of a new name; nonmedical alterations of physical appearance, such as changes in hair style, body hair management, or clothing choices; medical and not surgical, such as use of exogenous hormones; and medical and surgical.

Medical and surgical changes also take on a range of manifestations, including breast reductions or breast implants, alterations to facial bone structure, and genital reconfiguration. Importantly, not all transgender persons wish to undergo all aspects of gender transition. Research supports that completing desired aspects of gender transition is associated with improvements in mental health and well-being. However, numerous barriers exist to transition processes: primarily, the cost of the procedures and access to competent care providers. Due to these barriers, some gender minority individuals may seek to undergo transition processes outside of professional medical supervision. Such processes may include unsupervised use of illicit hormones or injection with silicone that may result in infection (Murad et al., 2010; Sanchez, Sanchez, & Danoff, 2009; Wilson, Rapues, Jin, & Raymond, 2014).

Discussion

Sexual orientation and gender minority persons face a range of health disparities. The study of health disparities within this population has moved beyond views of sexual orientation and gender minority status as intrinsically pathological toward a contextual understanding of social and cultural origins of health disparities within this population. To this end, the minority stress model has been instrumental in framing our understanding of unique, chronic, and socially based stresses that may contribute to health disparities among sexual orientation and gender minority populations. Still, many disparities exist across health behaviors including substance use, obesity, and sexual risk behaviors, and many other disparities affect specific groups within the umbrella of sexual orientation and gender minority, and many opportunities exist for system- and individual-level intervention and future research.

It is imperative to understand how public policy related to health impacts the well-being of sexual orientation and gender minority populations. Within minority stress theory, such a focus would aim to reduce the institutional or structural supports for distal causes of health disparities for sexual orientation and gender minority individuals. For example, enactment of anti-same-sex marriage laws in the United States (prior to the Supreme Court ruling in favor of marriage equality across the country) has been associated with negative impacts on the mental health and well-being of sexual minority persons (Riggle, Rostosky, & Horn, 2010). Similar research has explored how anti-bullying legislation impacts experiences of bullying among sexual minority youth (Hatzenbuehler, Schwab-Reese, Ranapurwala, Hertz, & Ramirez, 2015), though research indicates that specific implementations of such laws by teachers and in schools may affect their impact (Van Wormer & McKinney, 2003). It is important to explore ways in which public policy can be used to reduce health disparities, including ways in which individuals who work in the front lines of implementing such policy may be empowered to better advocate for sexual orientation and gender minority persons.

The minority stress model also emphasizes the importance of examining proximal, or within-person, stressors related to health disparities. Such stressors may include expectations of rejection, internalized stigma, and concealment of sexual orientation (Meyer, 1995). Despite the large body of research on minority stress, very limited empirical research on the treatment of stress among sexual orientation and gender minority populations has been undertaken. Although some treatment guidelines for sexual orientation and gender minority populations have been developed (Pachankis, Hatzenbuehler, Rendina, Safren, & Parsons, 2015), there have been very few trials of interventions that specifically address constellations of minority stress. Studies that have been conducted have tended to be relatively low quality

(e.g., including no-treatment wait list groups as controls, having small sample sizes, and having relatively homogeneous samples). It is important to extend extant work on minority stress beyond theoretical investigations of relations among constructs, which have generally been well established in the literature, and integrate these constructs with empirically supported interventions to provide quality, patient-centered care to sexual orientation and gender minority individuals.

Author Biographies

Dr. Mike C. Parent earned his PhD in Counseling Psychology from the University of Florida in 2013. He is now an assistant professor in counseling psychology and counselor education at the University of Texas at Austin. His program of research focuses on gender, sexuality, and behavioral health, as well as professional issues in psychology. He is the author of more than 40 peer-reviewed publications and has received numerous awards for his research and mentorship of students.

Teresa Gobble completed her BS in Psychology at the University of Central Florida. She is a doctoral student in the Counseling Psychology Program at the University of Texas–Austin. Her research focus includes examining risk factors for suicide in sexual and gender minority populations.

References

- Balsam, K. F., & Szymanski, D. M. (2005). Relationship quality and domestic violence in women's same-sex relationships: The role of minority stress. *Psychology of Women Quarterly*, 29, 258–269.
- Berlan, E. D., Corliss, H. L., Field, A. E., Goodman, E., & Austin, S. B. (2010). Sexual orientation and bullying among adolescents in the growing up today study. *The Journal of Adolescent Health*, 46, 366–371.
- Bockting, W. O., Miner, M. H., Swinburne Romine, R. E., Hamilton, A., & Coleman, E. (2013). Stigma, mental health, and resilience in an online sample of the US transgender population. *American Journal of Public Health*, 103, 943–951.
- Bowen, D. J., Balsam, K. F., & Ender, S. R. (2008). A review of obesity issues in sexual minority women. *Obesity*, 16, 221–228.
- Donatone, B., & Rachlin, K. (2013). An intake template for transgender, transsexual, genderqueer, gender nonconforming, and gender variant college students seeking mental health services. *Journal of College Student Psychotherapy*, 27, 200–211.
- Fingerhut, A. W., Riggle, E. D. B., & Rostosky, S. S. (2011). Same-sex marriage: The social and psychological implications of policy and debates. *Journal of Social Issues*, 67, 225–241.
- Guss, C. E., Williams, D. N., Reisner, S. L., Austin, S. B., & Katz-Wise, S. L. (2017). Disordered weight management behaviors, nonprescription steroid use, and weight perception in transgender youth. *Journal of Adolescent Health*, 60, 17–22.
- Halkitis, P. N., Moeller, R. W., & DeRaleau, L. B. (2008). Steroid use in gay, bisexual, and nonidentified men-who-have-sex-with-men: Relations to masculinity, physical, and mental health. *Psychology of Men & Masculinity*, 9, 106–115.
- Hatzenbuehler, M. L., Schwab-Reese, L., Ranapurwala, S. I., Hertz, M. F., & Ramirez, M. R. (2015). Associations between antibullying policies and bullying in 25 states. *JAMA Pediatrics*, 169, e152411–e152411.
- Keuroghlian, A. S., Shtasel, D., & Bassuk, E. L. (2014). Out on the street: A public health and policy agenda for lesbian, gay, bisexual, and transgender youth who are homeless. *American Journal of Orthopsychiatry*, 84, 66–72.

- Kim, Y. S., & Leventhal, B. (2011). Bullying and suicide. A review. *International Journal of Adolescent Medicine and Health*, 20, 133–154.
- Lee, J. G. L., Matthews, A. K., McCullen, C. A., & Melvin, C. L. (2014). Promotion of tobacco use cessation for lesbian, gay, bisexual, and transgender people. *American Journal of Preventive Medicine*, 47, 823–831.
- Marshall, M. P., Friedman, M. S., Stall, R., King, K. M., Miles, J., Gold, M. A., ... Morse, J. Q. (2008). Sexual orientation and adolescent substance use: A meta-analysis and methodological review. *Addiction*, 103, 546–556.
- Mattison, A. M., Ross, M. W., Wolfson, T., & Franklin, D. (2001). Circuit party attendance, club drug use, and unsafe sex in gay men. *Journal of Substance Abuse*, 13, 119–126.
- McConnell, E. A., Birkett, M. A., & Mustanski, B. (2015). Typologies of social support and associations with mental health outcomes among LGBT youth. *LGBT Health*, 2, 55–61.
- Meyer, I. H. (1995). Minority stress and mental health in gay men. *Journal of Health and Social Behavior*, 36, 38–56.
- Meyer, I. H. (2003). Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: Conceptual issues and research evidence. *Psychological Bulletin*, 129, 674–697.
- Meyer, I. H. (2015). Resilience in the study of minority stress and health of sexual and gender minorities. *Psychology of Sexual Orientation and Gender Diversity*, 2, 209–213.
- Moradi, B., Mohr, J. J., Worthington, R. L., & Fassinger, R. E. (2009). Counseling psychology research on sexual (orientation) minority issues: Conceptual and methodological challenges and opportunities. *Journal of Counseling Psychology*, 56, 5–22.
- Murad, M. H., Elamin, M. B., Garcia, M. Z., Mullan, R. J., Murad, A., Erwin, P. J., & Montori, V. M. (2010). Hormonal therapy and sex reassignment: A systematic review and meta-analysis of quality of life and psychosocial outcomes. *Clinical Endocrinology*, 72, 214–231.
- Nemoto, T., Bödeker, B., & Iwamoto, M. (2011). Social support, exposure to violence and transphobia, and correlates of depression among male-to-female transgender women with a history of sex work. *American Journal of Public Health*, 101, 1980–1988.
- Pachankis, J. E., Hatzenbuehler, M. L., Rendina, H. J., Safren, S. A., & Parsons, J. T. (2015). LGB-affirmative cognitive-behavioral therapy for young adult gay and bisexual men: A randomized controlled trial of a transdiagnostic minority stress approach. *Journal of Consulting and Clinical Psychology*, 83, 875–889.
- Parent, M. C., Torrey, C., & Michaels, M. S. (2012). “HIV testing is so gay”: The role of masculine gender role conformity in HIV testing among men who have sex with men. *Journal of Counseling Psychology*, 59, 465–470.
- Peterson, C. M., Matthews, A., Copps-Smith, E., & Conard, L. A. (2017). Suicidality, self-harm, and body dissatisfaction in transgender adolescents and emerging adults with gender dysphoria. *Suicide and Life-threatening Behavior*, 47, 475–482.
- Riggle, E. D., Rostosky, S. S., & Horne, S. G. (2010). Psychological distress, well-being, and legal recognition in same-sex couple relationships. *Journal of Family Psychology*, 24, 82–86.
- Rosario, M., Corliss, H. L., Everett, B. G., Reisner, S. L., Austin, S. B., Buchting, F. O., & Birkett, M. (2013). Sexual orientation disparities in cancer-related risk behaviors of tobacco, alcohol, sexual behaviors, and diet and physical activity: Pooled Youth Risk Behavior Surveys. *American Journal of Public Health*, 104, 245–254.
- Sanchez, N. F., Sanchez, J. P., & Danoff, A. (2009). Health care utilization, barriers to care, and hormone usage among male-to-female transgender persons in New York City. *American Journal of Public Health*, 99, 713–719.
- Scott, H. M., & Klausner, J. D. (2016). Sexually transmitted infections and pre-exposure prophylaxis: Challenges and opportunities among men who have sex with men in the US. *AIDS Research and Therapy*, 13, 1–5.
- Van Wormer, K., & McKinney, R. (2003). What schools can do to help gay/lesbian/bisexual youth: A harm reduction approach. *Adolescence*, 38, 409–420.
- Velez, B. L., Moradi, B., & Brewster, M. E. (2013). Testing the tenets of minority stress theory in workplace contexts. *Journal of Counseling Psychology*, 60, 532–542.

- Wilson, E., Rapues, J., Jin, H., & Raymond, H. F. (2014). The use and correlates of illicit silicone or “fillers” in a population-based sample of transwomen, San Francisco, 2013. *The Journal of Sexual Medicine*, 11, 1717–1724.
- Yancey, A. K., Cochran, S. D., Corliss, H. I., & Mays, V. M. (2003). Correlates of overweight and obesity among lesbian and bisexual women. *Preventive Medicine*, 36, 676–683.

Suggested Reading

- Frost, D. M., Lehavot, K., & Meyer, I. H. (2015). Minority stress and physical health among sexual minority individuals. *Journal of Behavioral Medicine*, 38, 1–8.
- Meyer, I. H., & Northridge, M. E. (2007). *The health of sexual minorities*. New York, NY: Springer.
- Saewyc, E. M. (2011). Research on adolescent sexual orientation: Development, health disparities, stigma, and resilience. *Journal of Research on Adolescence*, 21(1), 256–272.

Ethics Issues in the Integration of Complementary, Alternative, and Integrative Health Techniques into Psychological Treatment

Jeffrey E. Barnett

College of Arts and Sciences, Loyola University Maryland, Baltimore, MD, USA

Complementary, alternative, and integrative health techniques are widely used in America today, and their use has increased significantly in recent years. With the wide range of complementary and alternative medicine (CAM) practices currently being used in the United States, it is quite reasonable to assume that many psychologists will come in contact with patients who either are presently using one or more CAM techniques or who may benefit from the use of CAM interventions in addition to any mental health treatment to be provided. In fact, many individuals who value psychotherapy and seek it out are likely to also value and utilize CAM as well (Knaudt, Connor, Weisler, Churchill, & Davidson, 1999). Elkins, Marcus, Rajab, and Durgam (2005) found that 64% of psychotherapy clients they surveyed reported also using at least one CAM modality. Further, many psychotherapy patients may ask for or be open to the use of various CAM treatments.

These circumstances may create both opportunities and ethics challenges for psychologists. Relevant ethics issues to be addressed include clinical competence, informed consent, boundaries and multiple relationships, consultation and cooperation with other health professionals, advertising and public statements, integrating CAM techniques with psychological treatment, and making appropriate and effective referrals.

Clinical Competence

Psychologists are required to provide only those services for which they possess the necessary education, training, and supervised clinical experience to ensure that patients are not harmed and hopefully that patients are provided with the highest possible quality of care (American Psychological Association [APA], 2010). Clinical competence has been defined by Epstein and Hundert (2002) as “the habitual and judicious use of communication, knowledge, technical skills, clinical reasoning, emotions, values, and reflection in daily practice for the benefit of the individual and community served” (p. 226). In essence, the competent psychologist will possess the knowledge, skills, attitudes, and values necessary, and will implement them effectively, to provide care that maximally benefits each patient (Haas & Malouf, 2005).

Possessing the competence necessary to ensure that patients receive appropriate treatment is consistent with the aspirational general principles of the APA Ethics Code (APA, 2010) to include beneficence (doing good and providing benefit to patients), non-maleficence (avoiding harm to patients), and fidelity (fulfilling our obligations to our patients). It also is important to note that competence is not a static phenomenon; our knowledge and skills may degrade over time, and there will continually be new developments that may render our knowledge and skills obsolete. Thus, consistent with Standard 2.01, Boundaries of Competence (APA, 2010), psychologists “undertake relevant education, training, supervised experience, consultation or study” on an ongoing basis to ensure they maintain their competence and clinical effectiveness (p. 5).

With the great prevalence of CAM in our society today and with the significant percentage of the population that engages in the use of at least one CAM modality, an argument can be made that every practicing psychologist should possess at least general knowledge of CAM and its most widely used modalities to be considered competent and compliant with the APA Ethics Code (APA, 2010). This is true not just for those psychologists who may wish to integrate various CAM modalities into their clinical work with patients, but for all psychologists in clinical practice. Psychologists should be familiar with the current research literature on the clinical effectiveness, uses, contraindications, common side effects and interactions with other treatments, and mechanisms of action of the CAM modalities most likely to be used by our patients.

It is possible that many patients who seek out psychological treatment will already be utilizing one or more CAM modalities, often without medical supervision and of their own initiative. For many patients, what may seem like a harmless and “natural” practice may in fact bring with it great potential for harm. Many “natural” supplements may be quite dangerous and bring with them great risk if used inappropriately or in combination with prescription medications (Clauson, Santamarina, & Rutledge, 2008). Psychologists who are knowledgeable about CAM and who ask all their patients about the use of any CAM modalities can apply their knowledge to promote their patients’ best interests. This is especially important in light of the finding that upward of 35% of all psychotherapy patients use at least one CAM modality without informing their psychotherapist of this fact (Elkins et al., 2005).

Patients may “self-prescribe” various over-the-counter herbal substances that may interact with prescription medications and other over-the-counter substances they may be using with untoward effects. Knowledgeable psychologists can educate their patients about various CAM modalities and their appropriate uses as well as their relative risks and benefits. These psychologists may also be familiar with common side effects of CAM treatments that may easily be overlooked or misdiagnosed as mental health difficulties by those who are unfamiliar with

them. This knowledge can lead the psychologist to provide appropriate treatment to patients and, when indicated, refer these patients to their primary care physician to help ensure that medical conditions and treatments are managed appropriately.

Psychologists may also have patients they evaluate and treat who may benefit from the use of one or more CAM modalities. This form of integrative healthcare may be in many patients' best interest. Examples may include pain patients who may benefit from the use of hypnosis, biofeedback, acupuncture, or massage therapy in addition to ongoing psychotherapy and patients suffering from depression who may also benefit from yoga, meditation, dance movement therapy, aromatherapy, or the integration of spirituality and prayer with their ongoing psychotherapy. Accordingly, psychologists will need to possess two forms of clinical competence to effectively integrate CAM modalities with the psychological treatments they provide.

Psychologists will first need to possess knowledge about the various CAM modalities to know their uses, limitations, risks, and benefits. This will help psychologists know if a particular CAM modality may be potentially helpful for treating a patient's presenting problems. They will also need to complete the training necessary to be a competent practitioner of the CAM modality in question. Some CAM modalities require extensive education and supervised clinical experience as well as certification or licensure for a practitioner to be authorized to use that modality with patients. Examples of this include acupuncture, chiropractic, and massage therapy. Others may require extensive education and training to develop needed competence, but they do not require certification or licensure to practice them (e.g., hypnosis, biofeedback, yoga) but that are available as a means of demonstrating competence to the public. Finally, some forms of CAM, such as progressive muscle relaxation, have been well integrated into psychological practice with many psychologists developing competence in their use in the course of their training as psychologists.

Being a competent professional also means knowing the limits of one's competence. Being able to assess each patient's treatment needs and when not personally possessing needed clinical competence, making referrals to appropriately trained colleagues is in our patients' best interest. By being knowledgeable about the various CAM modalities and the training and expertise needed to practice them competently, we can then make referrals to appropriately trained and experienced professionals to best meet our patients' treatment needs.

Informed Consent

Informed consent involves providing patients with the information necessary so that they may make an informed decision about participating in the professional services being offered. This involves sharing information with patients at the outset of treatment about the treatment being proposed, reasonably available options and alternatives and their relative risks and benefits, and other relevant information that is likely to impact their decision to participate (Standard 10.01, Informed Consent to Therapy, APA, 2010). Thus, as has been highlighted, psychologists must be knowledgeable about widely available and empirically supported treatments for patients' presenting problems. This includes knowledge of CAM and the literature on the effectiveness and limitations of each modality so that this information can be shared with patients so they will have the information necessary to make informed decisions about the options available for their treatment.

Valid informed consent necessitates that patients have information provided to them verbally and in writing that it be presented in a manner that renders it "reasonably understandable" to

the patient, and we must actively ensure their understanding of the information being presented to them (APA, 2010, p. 6). Merely providing patients with a document to read and sign or asking them if they have any questions after all the information is shared with them is not sufficient. Further, consent must be given by the patient voluntarily and without any coercion or undue influence. Finally, the patient must be competent to give consent. This means that patients must have the cognitive ability to understand the information shared and they must have the legal right to give consent (e.g., an adult and not a minor).

It is also important to note that informed consent should not be considered a one-time event. Rather, it is an ongoing process that should be updated through open discussion with the patient anytime changes to the course of treatment are recommended or are under consideration. So, anytime a new treatment modality is suggested to the patient, a collaborative discussion should occur so that the patient may make the most informed decision possible about it. This very appropriately may occur after a patient's psychological treatment begins, and one or more CAM modalities are considered as means of assisting the patient to achieve agreed upon treatment goals.

Boundaries and Multiple Relationships

Boundaries provide ground rules and structure for the professional relationship to help ensure that patients are not exploited or harmed. Examples of boundaries that exist include touch, self-disclosure, time, space, location, and gifts (Pope & Keith-Spiegel, 2008). Boundaries may be avoided, crossed, or violated. To avoid a boundary is to never traverse it such as never engaging in touch with a patient, never engaging in any self-disclosure, and never accepting a gift from a patient under any circumstances. A boundary crossing is a clinically relevant and appropriate boundary incursion that is accepted by the patient and motivated by the patient's treatment needs and not the psychologist's personal needs. Examples include extending the time of a treatment session for a patient who is in crisis and holding a treatment session in a patient's home such as for a homebound patient who might otherwise not be able to receive needed treatment. In contrast, a boundary violation holds great potential for the exploitation of, and harm to, the patient, may not be welcomed by the patient, may not be motivated by and relevant to the patient's treatment needs, does not meet prevailing community standards, and violates the ethics code (Smith & Fitzpatrick, 1995).

A multiple relationship constitutes entering into a secondary relationship with a patient in addition to the treatment relationship. This may include social, personal, and business relationships, among others. Inappropriate multiple relationships typically include a conflict of interest and thus are likely to adversely impact the psychologist's objectivity and judgment in decision making. Multiple relationships that do not hold a significant potential for exploitation or harm are acceptable (APA, 2010); however when unsure, consultation with an experienced and trusted colleague is recommended.

When integrating various CAM modalities into ongoing psychological treatment, it is possible to violate boundaries and enter into an inappropriate multiple relationship inadvertently. This may occur when endeavoring to meet the patient's treatment needs while overlooking how the provision of certain psychological and CAM treatments should not be provided by the same individual. For example, a healthcare professional may be a licensed psychologist and a licensed massage therapist. A patient being treated in psychotherapy may also benefit from massage therapy. But for one individual to provide both services to a patient would likely

constitute an inappropriate multiple relationship with boundary violations occurring. While the boundary of touch may be appropriately crossed such as with a handshake or perhaps a hug to a grieving patient, to engage in the amount and type of physical contact involved in massage therapy holds great potential to violate the parameters of the psychotherapy relationship and process. While a licensed psychologist who is also a licensed massage therapist may appropriately provide psychotherapy and massage therapy to different patients, to provide both to a patient would be ill advised.

Psychologists should keep in mind that multiple relationships are not mandatory. Each situation is different, and we must consider each patient's treatment needs and the reasonably available options and alternatives for meeting them. While a patient may need a particular treatment service, this does not necessarily mean we are the ones who must provide it. Being knowledgeable of competent and credentialed CAM providers in the local area will be important so that appropriate referrals can be made for needed treatment. Thinking (erroneously) that we are the only ones who can meet a patient's needs can lead to impaired judgment and faulty decision making. Psychologists should also be mindful of conflict of interest situations such as financial motivations that may lead us to provide services to patients when a referral to another professional would be in their best interest. Again, psychologists should be aware of their motivations and keep their patients' best interests in mind.

Consultation and Cooperation With Other Professionals

As has been mentioned, psychologists should work collaboratively with professional colleagues in an effort to meet each patient's treatment needs. When we are not able to meet a patient's treatment needs personally, either due to limits to our clinical competence or the potential for an inappropriate multiple relationship, referrals to appropriately trained colleagues are essential.

The APA Ethics Code (APA, 2010) requires that psychologists avoid multiple relationships and conflicts of interests that may impair their objectivity and judgment. Yet, knowing when one's objectivity and judgment are impaired may be very challenging as individuals in general and health professionals in particular tend to be very ineffective at self-assessing our own abilities accurately (Dunning, Heath, & Suls, 2004). In these situations it is essential that we consult with colleagues who can provide an honest assessment of our judgment, functioning, and decision making. We must be open and honest with these colleagues, neither withholding nor distorting relevant information, to ensure that the consultation can be helpful so that patients receive the most appropriate treatment possible.

Consultation with colleagues is also relevant to finding the most appropriate treatment for patients as well as to ensure we personally are providing patients with optimal treatment. It is recommended that psychologists utilize the expertise of colleagues whenever questions arise about which treatment might be best for a patient or if the psychologist is the most appropriate individual to provide that treatment.

Advertising and Public Statements

While psychologists are entitled to advertise the clinical services they provide so that members of the public will know of and be able to access needed services, it is essential that all advertising

be done accurately and without any false or deceptive statements; only factual information may be shared (APA, 2010). When offering CAM services, as well as any psychological services, it is important to not overstate one's credentials or the likely benefits of treatment services offered. As it is stated in Standard 5.01, Avoidance of False or Deceptive Statements, psychologists should not make false or deceptive statements about "their training experience, or competence," "their credentials," "their services," or "the scientific or clinical basis for, or results or degree of success of, their services" (p. 8).

Thus, inappropriate and appropriate ways to advertise, respectively, include:

- Compy Tent, PhD, Licensed Psychologist
Internationally recognized expert in hypnosis, the number one treatment for pain control.
versus
- Hum Bul, PhD, ABPP, Licensed Psychologist
Diplomate in Health Psychology (American Board of Professional Psychology). Practice limited to the treatment of chronic pain.

To list one's relevant earned degrees and other relevant credentials that are based on demonstrating one's clinical competence and providing an objective description of the professional services offered meets the requirements of the APA Ethics Code's standard on advertising and public statements (APA, 2010). In contrast, making statements that reflect opinion instead of providing objective information is less useful to the public and may constitute a deceptive or misleading statement.

Standard 5, Advertising and Other Public Statements, of the APA Ethics Code (APA, 2010) requires that psychologists provide accurate and up-to-date information on all services provided, to include "the scientific or clinical basis for ... their services" (p. 8). This necessitates that psychologists maintain knowledge of the relevant literature on the various CAM modalities they utilize in their practices. Also, consistent with Standard 2.04, Bases for Scientific and Professional Judgments, psychologists should only offer services, and use techniques, with sufficient scientific evidence and support for their efficacy and use.

Additionally, the APA Ethics Code (APA, 2010) in Standard 5.05, Testimonials, forbids the use of testimonials from current patients or others who may be vulnerable to our undue influence. Testimonial endorsements involve utilizing statements from patients about the psychologist, the services received, and the outcomes achieved, which are used in the psychologist's advertising such as in an advertisement or on one's website. The use of testimonials should be considered a conflict of interest situation and an inappropriate multiple relationship. The patient who provides a testimonial becomes engaged in a new relationship with the psychologist in addition to the original treatment relationship.

With regard to ethical decision making, one can ask what the motivations are for utilizing a patient's testimonial in one's advertising (the motivation is the promotion of one's practice, not the patient's treatment needs), whose needs are being met by engaging in this action (clearly, the psychologist's needs), and how this action is relevant to the patient's treatment needs and treatment plan (there is no therapeutic benefit from taking this action). Even if the patient suggests providing a testimonial endorsement or if the patient is happy to provide it in response to the psychologist's request, this is a conflict of interest situation that is motivated by the psychologist's needs, which exploits the patient's trust and dependence. It will be very difficult for most patients to refuse such a request by their psychologist and in agreeing the nature of the treatment relationship is changed to the detriment of the patient.

Integrating CAM into Psychological Practice

As is hopefully evident, there are a number of issues and concerns relevant to integrating CAM into one's psychological practice. It is essential that first and foremost, the psychologist is competent to provide the CAM service in question. This will include possessing the necessary education and training but may also involve possessing any needed certification and/or licensure. Before integrating the use of any CAM modalities into one's psychology practice, credentialing processes should be researched. Barnett, Shale, Elkins, and Fisher (2014) provide a list of professional organizations, credentialing agencies and requirements, and their website addresses for each of the 14 most widely used forms of CAM. Education and training requirements may be found on many of these websites. Additionally, one may consult with experienced CAM practitioners about the knowledge, skills, and clinical experience needed to be a competent practitioner of that CAM modality. When licensure or certification is required, all relevant laws should be followed to ensure legal requirements are fulfilled.

Once clinical competence is established and any required certification or licensure is obtained, it is then important to ensure that CAM modalities are applied appropriately. As with any form of treatment, CAM modalities should only be integrated into a patient's treatment after a thorough assessment or evaluation is completed to ensure its relevance to the patient's treatment needs. No one CAM modality can be effective for the treatment of all presenting problems and conditions. Further, CAM modalities should only be applied in areas where credible research has demonstrated their effectiveness. If clear support for the use of a particular CAM modality is not established for a patient's treatment needs, the experimental nature of the treatment must be made clear to the patient as part of the informed consent process (APA, 2010).

Once a patient's treatment needs have been assessed and assuming that the psychologist possesses the needed competence to administer the treatment, one must consider the appropriateness of having both treatments (psychological and CAM) being offered by the same individual. As has been addressed, the administration of some CAM modalities is inconsistent with the psychotherapy relationship. One must consider boundary issues and the potential for adversely impacting the treatment relationship. Examples include integrating acupuncture, chiropractic, and massage therapy with psychotherapy. While one may be credentialed and competent in one or more of these in addition to psychotherapy, the type and amount of physical contact involved can be seen as inconsistent with prevailing professional standards for the psychotherapy relationship and process.

Psychologists integrating CAM modalities into psychological treatment should also be mindful of the risk of shifting professional roles. For example, a psychologist who integrates spirituality and prayer into psychotherapy may provide great benefit to a patient. But being supportive of a patient's personal use of prayer is very different from recommending specific Bible passages to the patient and praying together during psychotherapy sessions. One must be cautious about taking on the role of the clergy and leaving the psychologist role. Barnett and Johnson (2011) provide a decision-making model that can be applied to make such decisions.

When a patient's needs exceed the psychologist's clinical competence or when offering a treatment would risk boundary violations or entering into an inappropriate multiple relationship, referring the patient to a qualified colleague is recommended. Ideally, psychologists will maintain a list of competent CAM practitioners in the local area. Names can be added to or deleted from this list based on interactions with these CAM practitioners, reports from patients on their experiences with them, and from other trusted colleagues. It can be helpful to ask

colleagues who they refer patients to as well. Additionally, one can meet CAM practitioners through involvement in local professional organizations and by attending CAM conferences, workshops, and trainings.

Conclusion

Ultimately, it is each patient's welfare and best interests that should guide our decision making. Psychologists should respect patients' beliefs and preferences, be cautious about taking on multiple relationships and avoid conflict of interest situations, continually seek to maintain their clinical competence, and provide patients with needed information (truthfully and without deception or misrepresentation) to assist them to make informed decisions about participation in CAM treatments. When ethical dilemmas arise, the use of a decision-making process that includes self-reflection, consideration of the APA Ethics Code (both the aspirational general principles and the enforceable ethical standards), and consultation with colleagues is recommended to help ensure that patients receive the best care possible and to minimize the risk of exploitation of or harm to them.

Author Biography

Jeffrey E. Barnett, PsyD, ABPP, is a licensed psychologist as well as professor of psychology and associate dean at Loyola University Maryland. He is board certified by the American Board of Professional Psychology in clinical psychology and in clinical child and adolescent psychology, and he is a distinguished practitioner in psychology of the National Academies of Practice.

References

- American Psychological Association. (2010). *Ethical principles of psychologists and code of conduct*. Retrieved from <http://www.apa.org/ethics>.
- Barnett, J. E., & Johnson, W. B. (2011). Integrating spirituality and religion into psychotherapy: Persistent dilemmas, ethical issues, and a proposed decision-making process. *Ethics & Behavior, 21*(2), 147–164.
- Barnett, J. E., Shale, A. J., Elkins, G., & Fisher, W. (2014). *Complementary and alternative medicine for psychologists: An essential resource*. Washington, DC: American Psychological Association.
- Clauson, K. A., Santamarina, M. L., & Rutledge, J. C. (2008). Clinically relevant safety issues associated with St. John's wort product labels. *BMC Complementary and Alternative Medicine, 8*, 42.
- Dunning, D., Heath, C., & Suls, J. M. (2004). Flawed self-assessment: Implications for health, education, and the workplace. *Psychological Science in the Public Interest, 5*, 69–106.
- Elkins, G., Marcus, J., Rajab, M. H., & Durgam, S. (2005). Complementary and alternative therapy use by psychotherapy clients. *Psychotherapy: Theory, Research, Practice, Training, 42*, 232–235.
- Epstein, R. M., & Hundert, E. M. (2002). Defining and assessing professional competence. *Journal of the American Medical Association, 287*, 226–235.
- Haas, L. J., & Malouf, J. L. (2005). *Keeping up the good work: A practitioner's guide to mental health ethics* (4th ed.). Sarasota, FL: Professional Resource Press.
- Knaudt, P. R., Connor, K. M., Weisler, R. H., Churchill, L. E., & Davidson, J. R. (1999). Alternative therapy use by psychiatric outpatients. *Journal of Nervous and Mental Disease, 187*, 692–695.

- Pope, K. S., & Keith-Spiegel, P. (2008). A practical approach to boundaries in psychotherapy: Making decisions, bypassing blunders, and mending fences. *Journal of Clinical Psychology: In Session*, 64(5), 638–652.
- Smith, D., & Fitzpatrick, M. (1995). Patient-therapist boundary issues: An integrative review of theory and research. *Professional Psychology: Research and Practice*, 26(5), 499–506.

Suggested Reading

- Barnett, J. E., Shale, A. J., Elkins, G., & Fisher, W. (2014). *Complementary and alternative medicine for psychologists: An essential resource*. Washington, DC: American Psychological Association.
- Field, T. (2009). *Complementary and alternative therapies research*. Washington, DC: American Psychological Association.
- Lake, J. H., & Spiegel, D. (2007). *Complementary and alternative treatments in mental health care*. Washington, DC: American Psychiatric Publishing.
- National Center for Complementary and Integrative Health. (2015). Reports. Retrieved from <https://nccih.nih.gov/>.

Alcohol Use Disorder: Long-Term Consequences

Angela K. Stevens, Brittany E. Blanchard, Molin Shi,
and Andrew K. Littlefield

Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Long-Term Negative Health Outcomes from Alcohol Use

In addition to the host of short-term consequences associated with alcohol consumption, chronic use of alcohol, especially chronic misuse, is associated with several detrimental health outcomes. In fact, 25 chronic diseases in the *International Classification of Disease (ICD-10)* are completely attributable to long-term alcohol consumption, and chronic alcohol consumption is a component of more than 200 other diseases in the *ICD-10* (see Shield, Parry, & Rehm, 2014). Notably, alcohol shows a dose–response relation to many chronic diseases, in which the risk of disease onset and death from disease depends on the total volume of alcohol consumed (Shield et al., 2014). Further, between 1990 and 2010, alcohol was the third leading risk factor for global disease burden; however, for individuals between the ages of 15 and 49, it was the top risk factor worldwide (Lim et al., 2013).

Chronic, heavy drinking has been implicated as a risk factor for several deleterious health outcomes, including high blood pressure (Fuchs, Chambless, Whelton, Nieto, & Heiss, 2001), hemorrhagic and ischemic stroke (Rehm et al., 2009), Korsakoff's syndrome (i.e., a chronic memory disorder), and Wernicke's encephalopathy (i.e., alcohol-related dementia; see Pitel, Segobin, Ritz, Eustache, & Beaunieux, 2015). Indeed, alcohol-related Korsakoff's syndrome results from chronic, heavy alcohol consumption and thiamine (vitamin B1) deficiency, which is a consequence of a poor diet characteristic of excessive alcohol use (Pitel et al., 2015). Excessive alcohol consumption is also associated with epilepsy, or a predisposition for epileptic seizures (Shield et al., 2014). Research in this area has revealed a dose–response relation between alcohol consumption and the risk for epilepsy, such that repeated withdrawals from alcohol consumptions by heavy drinkers may increase risk for inducing an

epileptic episode (see Shield et al., 2014 for more details). Other neuropsychiatric conditions associated with chronic alcohol consumption include unipolar depression, Alzheimer's disease, and vascular dementia (Shield et al., 2014).

Because chronic alcohol misuse is also linked to several types of cancers, alcohol is considered a carcinogen. Some of the cancers associated with problematic use include those of the prostate, upper digestive track (e.g., oral cavity, esophagus, and larynx), lower digestive track (e.g., colon, rectum, and liver), and breast (Rehm et al., 2009). Further, chronic alcohol consumption is associated with liver diseases (e.g., liver cirrhosis, alcoholic hepatitis); in 2010, an estimated 493,300 deaths were attributed to alcohol-related cirrhosis of the liver (Rehm, Samokhvalov, & Shield, 2013). Further, a recent meta-analysis found that alcohol consumption increased the risk of developing pancreatitis (though the dose-response relation for acute pancreatitis differed across men and women; see Samokhvalov, Rehm, & Roerecke, 2015). Moreover, chronic alcohol use increases the likelihood of developing diabetes mellitus, a condition commonly caused by pancreatitis (Rehm et al., 2009). Alcohol consumption affects blood pressure, and chronic alcohol misuse is predictive of hypertensive heart disease and ischemic heart disease (Rehm et al., 2009). There is also evidence that chronic alcohol use is associated with infectious diseases such as tuberculosis (World Health Organization [WHO], 2014). Although the biological evidence to support the causal link is lacking, observational research has indicated that alcohol consumption may exacerbate the disease progression of psoriasis, which is a chronic inflammatory autoimmune skin disease (Shield et al., 2014). Thus, chronic, heavy alcohol consumption puts an individual at risk for a myriad of negative physical conditions and diseases.

Detrimental outcomes of alcohol consumption during pregnancy include low birth weight (Rehm et al., 2009) and the development of fetal alcohol spectrum disorders, such as fetal alcohol syndrome (FAS), alcohol-related birth defects, and alcohol-related neurodevelopmental disorders (see Riley, Infante, & Warren, 2011). Specifically, infants with FAS exhibit growth deficiencies, craniofacial abnormalities, and heart defects (Riley et al., 2011). Individuals with FAS experience significant problems in adaptive, academic, and behavioral functioning (Streissguth et al., 2004). More specifically, fetal alcohol effects are associated with lower levels of intelligence, disrupted school experiences, criminal behavior and/or incarceration, inappropriate sexual behavior, and alcohol/drug problems (Streissguth et al., 2004). FAS is the leading preventable (i.e., nongenetic) cause of intellectual development disorder (previously known as mental retardation) and birth defects (O'Leary et al., 2013). In sum, alcohol consumption during pregnancy can lead to debilitating long-term health-related consequences for the offspring.

Potential Health Benefits

Despite the many potential adverse consequences associated with alcohol consumption, empirical evidence suggests that light-to-moderate drinking may provide some health benefits. For example, some data indicated that light-to-moderate drinking yields a reduction in risk for coronary heart disease and stroke (Shield et al., 2014). Further, alcohol use may reduce the risk of type 2 diabetes (Shield et al., 2014) and dementia (Peters, Peters, Warner, Beckett, & Bulpitt, 2008). There is also some evidence that alcohol consumption might lower the risk of certain types of cancers, including renal cell carcinoma, Hodgkin's lymphoma, and non-Hodgkin's lymphoma (see Shield et al., 2014). Despite these findings, the health benefits of moderate alcohol use have recently been called into question (see Chikritzhs et al., 2015), and the benefits may not outweigh the consequences.

Treatment Approaches to Alcohol Use Disorders

Numerous treatment approaches to AUDs are available. Though non-exhaustive, provided below are brief summaries of several relevant, empirically supported treatments for AUDs and related problems.

Brief Interventions

Brief interventions are evidence-based practices designed to motivate individuals to understand, reduce, and/or eliminate alcohol use, usually delivered in a 5- to 20-min session (Center for Substance Abuse Treatment, Substance Abuse and Mental Health Services Administration, 1999). Findings from a meta-analytic review suggest that brief interventions are as efficacious as extended alcohol treatments (Moyer, Finney, Swearingen, & Vergun, 2002). Common brief interventions typically used on college campuses are variants of brief motivational interventions (Murphy et al., 2001), such as the *Brief Alcohol Screening and Intervention for College Students* (BASICS) (Dimeff, Baer, Kivlahan, & Marlatt, 1999). Brief interventions have also demonstrated effectiveness in community mental health and primary care settings (O'Donnell et al., 2014).

Motivational Interviewing (MI)

Motivational interviewing (MI) is a brief, client-centered method used in conjunction with individual therapy that has strong research support for its efficacy in treating individuals with alcohol-related problems (Rollnick, Miller, & Butler, 2008). MI seeks to evoke self-efficacy and motivation while encouraging individuals to use their own reasons for change to resolve ambivalence, reduce resistance, and progress in their readiness to change (Rollnick et al., 2008). Similarly, motivational enhancement therapy, which combines MI techniques and personal feedback, has also been used to promote motivation for positive change in individuals who engage in problematic drinking (Miller, Zweben, DiClemente, & Rychtarik, 1992). Research has demonstrated strong support for the integration and use of MI as part of treatment for substance use disorders, including AUDs (see Lundahl & Burke, 2009).

Cognitive Behavioral Approaches

Numerous cognitive behavioral (CB) interventions to treat alcohol dependence have been developed and empirically tested (see Witkiewitz, Marlatt, & Walker, 2005). The CB approach targets cognitive, affective, and behavioral factors related to problematic drinking. It is hypothesized that deficits in abilities to cope effectively with stress serve to maintain excessive and subsequent drinking (Morgenstern & Longabaugh, 2000). As such, CB interventions typically include standardized, performance-based techniques, such as behavioral rehearsal and modeling, which teach individuals to have a sense of mastery in stressful situations without resorting to alcohol consumption.

Behavioral Couples Therapy (BCT)

Behavioral couples therapy for AUD (ABCT) is the most extensively studied approach of treatment at the couple level and has strong research support. ABCT is an outpatient, conjoint treatment that may be used as an additional component of, or subsequent to, individual

therapy for those with AUDs and their partners (Epstein & McCrady, 1998). The underlying assumption posits that alcohol-related problems and intimate relationships are reciprocal and reinforcing of the alcohol use (Powers, Vedel, & Emmelkamp, 2008). A meta-analysis of 12 randomized controlled trials found that ABCT demonstrated a significantly greater effect compared with individual treatment alone for individuals with alcohol use problems in relationships (Powers et al., 2008).

Support Groups

Although support groups such as Alcoholics Anonymous (AA) are commonly utilized in the treatment of AUDs, findings are mixed with regard to effectiveness of AA (Tonigan, Toscova, & Miller, 1996). However, some components of AA are supported by research (e.g. recovering individuals as therapists, peer-led self-help groups; Kownacki & Shadish, 1999). Although AA and 12-step programs may not be effective for individuals with certain traits (e.g., severe mental illness), self-help groups such as these represent a viable option for many individuals who are motivated to maintain abstinence (Montalto, 2016).

Pharmacotherapy

The US Food and Drug Administration (FDA) has approved three pharmacological treatments for AUDs: disulfiram (e.g., Antabuse), oral and extended-release naltrexone (e.g., ReVia, Vivitrol), and acamprosate (e.g., Campral; Mark, Kassed, Vandivort-Warren, Levit, & Kranzler, 2009). Although disulfiram (an aldehyde dehydrogenase inhibitor) is meant to condition an individual to avoid alcohol intake by inducing nausea when individuals consume alcohol, it appears to reduce consumption but often fails to yield abstinence (Garbutt, West, Carey, Lohr, & Crews, 1999). Naltrexone (ReVia, Vivitrol) is an opioid antagonist, which lessens the positively reinforcing effects of alcohol (Petrakis et al., 2005). Studies have demonstrated its relative effectiveness in reducing overall alcohol consumption (Petrakis et al., 2005), as well as rate of relapse (Carmen, Angeles, Ana, & María, 2004). Naltrexone has also been shown to enhance treatment effectiveness when used in conjunction with other treatment modalities, such as psychosocial therapies or coping skills therapy (Jarosz, Miernik, Wachal, Walczak, & Krumpl, 2013). Acamprosate (Campral) is thought to enhance the inhibitory effects of the neurotransmitter gamma-aminobutyric acid (GABA) (Hunt, 1993) and acts primarily as a functional glutamate antagonist. Meta-analytic reviews suggest that acamprosate is also effective as a complementary treatment with different psychosocial interventions in treating alcohol dependence and appears to be as effective as naltrexone (Carmen et al., 2004; Garbutt et al., 1999).

Other Medical Interventions for AUDs and Related Conditions

Detoxification from chronic alcohol consumption is an important first step in treatment and often requires medical supervision, given the potential fatality of alcohol withdrawal symptoms (i.e., delirium tremens; see Kosten & O'Connor, 2003). Benzodiazepines have demonstrated effectiveness for managing seizures and delirium related to detoxification (Kosten & O'Connor, 2003). Further, anticonvulsants (e.g., carbamazepine) and alpha-agonists (e.g., clonidine) appear to decrease severity of withdrawal symptoms, and beta-blockers reduce cravings for alcohol (Kosten & O'Connor, 2003). Indeed, medication to manage withdrawal symptoms can be delivered on a fixed schedule or as needed, though the latter

requires more frequent monitoring yet leads to a more rapid detoxification. Without pharmacological intervention withdrawal symptoms are expected to peak within 72 hr after last use, but medications reduce these symptoms within hours of delivery (see Kosten & O'Connor, 2003 for more details).

The cornerstone of treatment for alcoholic liver disease is abstinence, as significant improvement in disease progression can be observed in as little as 3 months. However, nutrition therapy also appears to be effective, given that the severity of malnutrition related to chronic alcohol consumption is correlated with disease severity and negative outcomes (see O'Shea, Dasarathy, & McCullough, 2010). Steroid treatment for alcoholic hepatitis has received the most attention in the extant literature, and there is evidence to suggest that a tapered dose of prednisolone over 2 weeks is most effective; however, studies often exclude individuals at an advanced stage of liver disease, which limits the generalizability of these findings (O'Shea et al., 2010). Notably, alcoholic liver disease is the second most common indicator for a liver transplant; however, given the stigma related to alcoholic liver disease (i.e., a "self-induced" disease), less than 5% of patients with advanced liver disease resulting from chronic alcohol consumption are formally evaluated for candidacy for liver transplantation (O'Shea et al., 2010).

Conclusions

AUDs are among the most commonly experienced psychiatric disorders in the United States, with especially high rates among young adults. Although many individuals who experience AUDs during adolescence and young adulthood will subsequently recover, underage alcohol use is more likely to be fatal among young people than all other illegal drugs combined (Grunbaum et al., 2002). In addition to alcohol-related injuries, alcohol consumption has been linked to over 200 health conditions (WHO, 2014). Given that alcohol use is normative and problematic use is common, treatment options to increase treatment involvement and retention rates, such as MI and stepped care approaches, may be especially beneficial (see Littlefield & Sher, 2009). Further, in light of data supporting the so-called prevention paradox (i.e., most alcohol-related harm in a given population arises within drinkers that are "low risk"; see Rossow & Romelsjo, 2006) and the health perils of acute intoxication regardless of disorder status, preventing alcohol-related health problems through prevention policy efforts, should also be considered in addition to treatments aimed at individuals with AUDs (see Gruenewald, 2011).

Author Biographies

Angela K. Stevens is currently a PhD student in clinical psychology at Texas Tech University in the Department of Psychological Sciences. Broadly, her research interests include individual differences in adult problematic substance use.

Brittany E. Blanchard is currently a PhD student in clinical psychology at Texas Tech University in the Department of Psychological Sciences. Her research examines individual differences in alcohol and substance use, including use of protective behavioral strategies.

Molin Shi is currently a PhD student in clinical psychology at Texas Tech University in the Department of Psychological Sciences. Her research focuses on the influences and consequences of substance-related behaviors, with an emphasis on the familial, social, and acculturative impact of substance use in minority subgroups.

Andrew K. Littlefield is currently an assistant professor at Texas Tech University in the Department of Psychological Sciences. He obtained his predoctoral training in clinical psychology at the University of Missouri and completed his clinical internship at the University of Mississippi Medical Center. Dr. Littlefield's research focuses on addictive behaviors.

References

- Carmen, B., Angeles, M., Ana, M., & María, A. J. (2004). Efficacy and safety of naltrexone and acamprosate in the treatment of alcohol dependence: A systematic review. *Addiction*, 99(7), 811–828.
- Center for Substance Abuse Treatment, Substance Abuse and Mental Health Services Administration. (1999). *Brief interventions and brief therapies for substance abuse: Treatment improvement protocol (TIP) series* (DHHS Publication No. SMA 99-3353). Rockville, MD: Author.
- Chikritzhs, T., Stockwell, T., Naimi, T., Andreasson, S., Dangardt, F., & Liang, W. (2015). Has the leaning tower of presumed health benefits from 'moderate' alcohol use finally collapsed? *Addiction*, 110(5), 726–727.
- Dimeff, L. A., Baer, J. S., Kivlahan, D. R., & Marlatt, G. A. (Eds.) (1999). *Brief alcohol screening and intervention for college students (BASICS): A harm reduction approach*. New York, NY: Guilford Press.
- Epstein, E. E., & McCrady, B. S. (1998). Behavioral couples treatment of alcohol and drug use disorders: Current status and innovations. *Clinical Psychology Review*, 18(6), 689–711.
- Fuchs, F. D., Chambless, L. E., Whelton, P. K., Nieto, F. J., & Heiss, G. (2001). Alcohol consumption and the incidence of hypertension: The atherosclerosis risk in communities study. *Hypertension*, 37(5), 1242–1250.
- Garbutt, J. C., West, S. L., Carey, T. S., Lohr, K. N., & Crews, F. T. (1999). Pharmacological treatment of alcohol dependence: A review of the evidence. *JAMA*, 281(14), 1318–1325.
- Gruenewald, P. J. (2011). Regulating availability: How access to alcohol affects drinking and problems in youth and adults. *Alcohol Research and Health*, 34, 248–256.
- Grunbaum, J. A., Kann, L., Kinchen, S. A., Williams, B., Ross, J. G., Lowry, R., & Kolbe, L. (2002). Youth risk behavior surveillance: United States, 2001. *Journal of School Health*, 72(8), 313–328.
- Hunt, W. A. (1993). Neuroscience research: how has it contributed to our understanding of alcohol abuse and alcoholism? A review. *Alcoholism: Clinical and Experimental Research*, 17(5), 1055–1065.
- Jarosz, J., Miernik, K., Wąchal, M., Walczak, J., & Kruppl, G. (2013). Naltrexone (50 mg) plus psychotherapy in alcohol-dependent patients: A meta-analysis of randomized controlled trials. *The American Journal of Drug and Alcohol Abuse*, 39(3), 144–160.
- Kosten, T. R., & O'Connor, P. G. (2003). Management of drug and alcohol withdrawal. *New England Journal of Medicine*, 348(18), 1786–1795.
- Kownacki, R. J., & Shadish, W. R. (1999). Does Alcoholics Anonymous work? The results from a meta-analysis of controlled experiments. *Substance Use & Misuse*, 34(13), 1897–1916.
- Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H., ... Aryee, M. (2013). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: A systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*, 380(9859), 2224–2260.
- Littlefield, A. K., & Sher, K. J. (2009). Alcohol use disorders in young adulthood. In J. E. Grant & M. N. Potenza (Eds.), *Young Adult Mental Health* (pp. 292–310). New York, NY: Oxford University Press.
- Lundahl, B., & Burke, B. L. (2009). The effectiveness and applicability of motivational interviewing: A practice-friendly review of four meta-analyses. *Journal of Clinical Psychology*, 65(11), 1232–1245.
- Mark, T. L., Kassed, C. A., Vandivort-Warren, R., Levit, K. R., & Kranzler, H. R. (2009). Alcohol and opioid dependence medications: Prescription trends, overall and by physician specialty. *Drug and Alcohol Dependence*, 99(1), 345–349.

- Miller, W. R., Zweben, A., DiClemente, C. C., & Rychtarik, R. G. (1992). *Motivational enhancement therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence* (NIH Publication No. 94-3723). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.
- Montalto, M. (2016). Alcoholics Anonymous: One treatment program to rule them all? *Journal of Alcoholism & Drug Dependence*, 3(6), 1–5.
- Morgenstern, J., & Longabaugh, R. (2000). Cognitive-behavioral treatment for alcohol dependence: A review of evidence for its hypothesized mechanisms of action. *Addiction*, 95(10), 1475–1490.
- Moyer, A., Finney, J. W., Swearingen, C. E., & Vergun, P. (2002). Brief interventions for alcohol problems: A meta-analytic review of controlled investigations in treatment-seeking and non-treatment-seeking populations. *Addiction*, 97(3), 279–292.
- Murphy, J. G., Duchnick, J. J., Vuchinich, R. E., Davison, J. W., Karg, R. S., Olson, A. M., ... Coffey, T. T. (2001). Relative efficacy of a brief motivational intervention for college student drinkers. *Psychology of Addictive Behaviors*, 15(4), 373–379.
- O'Donnell, A., Anderson, P., Newbury-Birch, D., Schulte, B., Schmidt, C., Reimer, J., & Kaner, E. (2014). The impact of brief alcohol interventions in primary healthcare: A systematic review of reviews. *Alcohol and Alcoholism*, 49(1), 66–78.
- O'Leary, C., Leonard, H., Bourke, J., D'Antoine, H., Bartu, A., & Bower, C. (2013). Intellectual disability: Population-based estimates of the proportion attributable to maternal alcohol use disorder during pregnancy. *Developmental Medicine & Child Neurology*, 55(3), 271–277.
- O'Shea, R. S., Dasarathy, S., & McCullough, A. J. (2010). Alcoholic liver disease. *Hepatology*, 51(1), 307–328.
- Peters, R., Peters, J., Warner, J., Beckett, N., & Bulpitt, C. (2008). Alcohol, dementia and cognitive decline in the elderly: A systematic review. *Age and Ageing*, 37(5), 505–512.
- Petrakis, I. L., Poling, J., Levinson, C., Nich, C., Carroll, K., Rounsaville, B., & VA New England VISN I MIRECC Study Group. (2005). Naltrexone and disulfiram in patients with alcohol dependence and comorbid psychiatric disorders. *Biological Psychiatry*, 57(10), 1128–1137.
- Pitel, A. L., Segobin, S. H., Ritz, L., Eustache, F., & Beaunieux, H. (2015). Thalamic abnormalities are a cardinal feature of alcohol-related brain dysfunction. *Neuroscience & Biobehavioral Reviews*, 54, 38–45.
- Powers, M. B., Vedel, E., & Emmelkamp, P. M. (2008). Behavioral couples therapy (BCT) for alcohol and drug use disorders: A meta-analysis. *Clinical Psychology Review*, 28(6), 952–962.
- Rehm, J., Mathers, C., Popova, S., Thavorncharoensap, M., Teerawattananon, Y., & Patra, J. (2009). Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. *The Lancet*, 373(9682), 2223–2233.
- Rehm, J., Samokhvalov, A. V., & Shield, K. D. (2013). Global burden of alcoholic liver diseases. *Journal of Hepatology*, 59(1), 160–168.
- Riley, E. P., Infante, M. A., & Warren, K. R. (2011). Fetal alcohol spectrum disorders: An overview. *Neuropsychology Review*, 21(2), 73–80.
- Rollnick, S., Miller, W. R., & Butler, C. (2008). *Motivational Interviewing in health care: Helping patients change behavior*. New York, NY: Guilford Press.
- Rossow, I., & Romelsjö, A. (2006). The extent of the “prevention paradox” in alcohol problem as a function of population drinking patterns. *Addiction*, 101(1), 84–90.
- Samokhvalov, A. V., Rehm, J., & Roerecke, M. (2015). Alcohol consumption as a risk factor for acute and chronic pancreatitis: A systematic review and a series of meta-analyses. *eBioMedicine*, 2(12), 1996–2002.
- Shield, K. D., Parry, C., & Rehm, J. (2014). Chronic diseases and conditions related to alcohol use. *Alcohol Research: Current Reviews*, 35(2), 155–171.
- Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental & Behavioral Pediatrics*, 25(4), 228–238.

- Tonigan, J. S., Tosco, R., & Miller, W. R. (1996). Meta-analysis of the literature on alcoholics anonymous: Sample and study characteristics moderate findings. *Journal of Studies on Alcohol*, 57(1), 65–72.
- Witkiewitz, K., Marlatt, G. A., & Walker, D. (2005). Mindfulness-based relapse prevention for alcohol and substance use disorders. *Journal of Cognitive Psychotherapy*, 19(3), 211–228.
- World Health Organization. (2014). *Global status report on alcohol and health*. Geneva, Switzerland: World Health Organization.

Suggested Reading

- Li, T. K. (2008). Quantifying the risk for alcohol use and alcohol attributable health disorders: Present findings and future research needs. *Journal of Gastroenterology and Hepatology*, 23(s1), 2–8.
- Marlatt, G. A., & Witkiewitz, K. (2002). Harm reduction approaches to alcohol use: Health promotion, prevention, and treatment. *Addictive Behaviors*, 27(6), 867–886.
- Rehm, J., Room, R., Graham, K., Monteiro, M., Gmel, G., & Sempos, C. T. (2003). The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease: An overview. *Addiction*, 98(9), 1209–1228.
- Room, R., Babor, T., & Rehm, J. (2005). Alcohol and public health. *The Lancet*, 365(9458), 519–530.
- Shield, K. D., Parry, C., & Rehm, J. (2014). Chronic diseases and conditions related to alcohol use. *Alcohol Research: Current Reviews*, 35(2), 155–173.

Body Image Assessment

Kathryn E. Smith^{1,2}, Tyler B. Mason^{1,3},
Jason M. Lavender⁴, and Stephen A. Wonderlich^{1,2}

¹*Neuropsychiatric Research Institute, Fargo, ND, USA*

²*Department of Psychiatry and Behavioral Science, University of North Dakota School of
Medicine and Health Sciences, Grand Forks, ND, USA*

³*Department of Psychology, University of Southern California, Los Angeles, CA, USA*

⁴*Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA*

Body image is a complex, multidimensional construct that includes a number of interrelated facets (e.g., Yagil et al., 2015). As such, a wide range of assessments have been developed to measure these domains. Such measures differ in methodology, specificity and content, and in their targeted populations. It is therefore important to consider the targeted patient or sample, the specific nature of presenting body image issues, and the psychometric properties of measures when selecting a body image assessment. Although a comprehensive list and description of body image measures is beyond the scope of this chapter, below we briefly review and describe a variety of measures that differ in the characteristics noted above.

Methodology

While the majority of body image assessments are self-report measures, subscales related to body image are included in three widely used structured interviews for eating disorders: the Eating Disorder Examination (Fairburn & Cooper, 1993), the Structured Interview for Anorexia and Bulimic Disorders (Fichter, Herpertz, Quadflieg, & Herpertz-Dahlmann, 1998), and the Interview for Diagnosis of Eating Disorders (Kutlesic, Williamson, Gleaves, Barbin, & Murphy-Eberenz, 1998). Similarly, self-report measures of global eating psychopathology include subscales that assess overvaluation of shape and weight or body dissatisfaction (e.g., Eating Disorder Examination Questionnaire, Fairburn & Beglin, 1994; Eating Disorder Inventory, Garner, 2004).

Although many self-report questionnaires use items scored in a Likert-type or multiple-choice format, figural or schematic measures can also provide an index of self-discrepancy related to body image. Figure rating scales require respondents to compare their current figure with an array of figures that differ in body size; the discrepancy between one's current and ideal size provides an index of body dissatisfaction (e.g., Thomson & Gray, 1995; Williamson, Davis, Bennett, Goreczny, & Gleaves, 1989). More recently, the somatomorphic matrix was developed to assess satisfaction based on dimensions of body fat and muscularity (Gruber, Pope, Borowiecki, & Cohane, 2000) and is thought to be relevant to both males and females. In addition, *in vivo* methods have been applied to the measurement of body image, particularly with respect to perceptual domains (e.g., Video Distortion Method, Probst, Vandereycken, Van Coppenolle, & Pieters, 1995; Digital Photography Mirror-Based Technique; Shafran & Fairburn, 2002).

Content

Measures of body image also range in content and specificity and have previously been categorized based on cognitive, affective, subjective, behavioral, perceptual, and sociocultural focus (Thompson, Roehrig, Cafri, & Heinberg, 2005), although many instruments assess multiple domains. Cognitive assessments evaluate individuals' attitudes and beliefs about the importance of physical appearance (i.e., overvaluation of shape and weight) and the extent to which they experience appearance-related cognitions (e.g., Appearance Schemas Inventory, Cash & Labarge, 1996; Beliefs about Appearance Scale, Spangler & Stice, 2001). Affective and subjective measures generally assess emotional states that accompany body dissatisfaction (e.g., Situational Inventory of Body Image Dysphoria, Cash, 1994) or individuals' degree of satisfaction or concern with their body image, which may be global (e.g., Body Shape Questionnaire; Cooper, Taylor, Cooper, & Fairburn, 1987) or specific (e.g., Body Parts Satisfaction Scale; Petrie, Tripp, & Harvey, 2002). In turn, behavioral indices measure common manifestations of body dissatisfaction, such as body avoidance (e.g., Body Image Avoidance Questionnaire; Rosen, Srebnick, Saltzberg, & Wendt, 1991) or body checking (e.g., Body Checking Questionnaire; Reas, Whisenhunt, Netemeyer, & Williamson, 2002). Further, measures of perceptual components of body image (e.g., signal detection methods) provide an index of body size distortion, which is a core feature of body dysmorphic disorder and has also been observed in eating disorders.

Other measures evaluate sociocultural and interpersonal factors that have been thought to contribute to body image dissatisfaction and the internalization of thin ideals (e.g., Sociocultural Attitudes Towards Appearance Scale; Thompson, Van den Berg, Roehrig, Guarda, & Heinberg, 2004; Ideal Body Internalization Scale; Stice and Agras, 1998; Physical Appearance Related Teasing Scale; Thompson, Fabian, Moulton, Dunn, & Altabe, 1991).

Specific Populations

While body image concerns have been documented in a variety of medical and psychiatric populations, the majority of assessment measures have been developed and validated using adult Caucasian females. Thus, there remains a limited number of assessment measures for which psychometric properties have been evaluated in other samples (e.g., males, children, minority populations). Nevertheless, some advances have been made in the development of

measures that specifically assess muscular body ideals that are particularly common among males (e.g., Drive for Muscularity Scale; McCreary & Sasse, 2000; Muscle Appearance Satisfaction Scale; Mayville, Williamson, White, Netemeyer, & Drab, 2002) and developmentally appropriate measures for children and adolescents (e.g., Cragun, DeBate, Ata, & Thompson, 2013; Veron-Guidry & Williamson, 1996).

Given that body image is not a static construct and may change in response to medical status, some measures have been developed to address body image concerns that may be particularly relevant among individuals with specific health problems, such as cancer (Hopwood, Fletcher, Lee, & Al Ghazal, 2001; Kopel, Eiser, Cool, & Carter, 1998), inflammatory bowel disease (McDermott et al., 2014), and conditions associated with disfigurement (Jewett et al., 2015; Lawrence et al., 1998). While the number of body image assessments for specific medical conditions remains limited, it has been recommended that health providers consider conducting routine screenings of body image concerns, as doing so may (a) initiate conversations about issues that patients may not otherwise voice openly, (b) identify patients currently experiencing or who are at risk for the negative sequelae of body dissatisfaction, and (c) provide insight into possible predictors of health outcomes (Pruzinsky, 2004). Thus, further development of condition-specific measures, as well as the use of standardized screening processes in healthcare settings, has promising clinical utility. These efforts may offer more precise and idiographic measures of condition-specific body image concerns, which could serve to inform subsequent prevention and intervention.

Conclusions

There are a large number of existing assessments, including many that are freely available for use in research and clinical contexts that may be used to measure various aspects of body image. Brief assessments of overall body image (e.g., eight-item Body Shape Questionnaire; Pook, Tuschen-Caffier, & Brähler, 2008) and assessments developed for individuals with specific health conditions may be of greatest utility in clinical health settings. In sum, the use of accurate methods to assess and detect body image concerns is necessary to provide health professionals with opportunities to address such concerns and ameliorate risk for the development of related psychopathology.

Author Biographies

Kathryn E. Smith, PhD, is a T32 postdoctoral research fellow at the Neuropsychiatric Research Institute. She received her BA in psychology from Macalester College and her PhD in clinical psychology from Kent State University. Her primary interests include emotion regulation and co-occurring psychopathology in eating disorders and obesity.

Tyler B. Mason, PhD, is an assistant professor in the Department of Preventative Medicine at the University of Southern California. He received his PhD in applied psychological science in 2015 and BS in psychology in 2010 from Old Dominion University. His primary research interests include etiology and treatment of binge eating and obesity.

Jason M. Lavender, PhD, is an assistant research scientist in the Department of Psychiatry at the University of California, San Diego. He completed his undergraduate education at Duke University and his PhD in clinical psychology at the University at Albany, SUNY. His research addresses emotion, neurocognition, and personality in eating disorders.

Stephen A. Wonderlich, PhD, is the Chester Fritz distinguished professor and associate chairperson in the Department of Psychiatry and Behavioral Science at the University of North Dakota, chair of Eating Disorders and codirector of the Eating Disorder and Weight Management Center at Sanford, and president and scientific director of the Neuropsychiatric Research Institute.

References

- Cash, T. F. (1994). The Situational Inventory of Body-Image Dysphoria: Contextual assessment of a negative body image. *Behaviour Therapist*, 17, 133–133.
- Cash, T. F., & Labarge, A. S. (1996). Development of the Appearance Schemas Inventory: A new cognitive body-image assessment. *Cognitive Therapy and Research*, 20, 37–50.
- Cooper, P. J., Taylor, M. J., Cooper, Z., & Fairburn, C. G. (1987). The development and validation of the body shape questionnaire. *International Journal of Eating Disorders*, 6, 485–494.
- Cragun, D., DeBate, R. D., Ata, R. N., & Thompson, J. K. (2013). Psychometric properties of the body esteem scale for adolescents and adults in an early adolescent sample. *Eating and Weight Disorders*, 18, 275–282. doi:10.1007/s40519-013-0031-1
- Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders*, 16, 363–370.
- Fairburn, C. G., & Cooper, Z. (1993). The eating disorder examination. In C. G. Fairburn & G. T. Wilson (Eds.), *Binge eating: Nature, assessment and treatment* (pp. 317–360). New York, NY: Guilford Press.
- Fichter, M. M., Herpertz, S., Quadflieg, N., & Herpertz-Dahlmann, B. (1998). Structured interview for anorexic and bulimic disorders for DSM-IV and ICD-10: Updated (third) revision. *International Journal of Eating Disorders*, 24, 227–249.
- Garner, D. M. (2004). *EDI 3: Eating disorder inventory-3: Professional manual*. Lutz, FL: Psychological Assessment Resources.
- Gruber, A. J., Pope, H. G., Borowiecki, J. J., & Cohane, G. (2000). The development of the somatomorphic matrix: A bi-axial instrument for measuring body image in men and women. *Kinanthropometry*, VI, 217–231.
- Hopwood, P., Fletcher, I., Lee, A., & Al Ghazal, S. (2001). A body image scale for use with cancer patients. *European Journal of Cancer*, 37, 189–197.
- Jewett, L. R., Malcarne, V. L., Kwakkenbos, L., Harcourt, D., Rumsey, N., Körner, A., ... Heinberg, L. (2015). Development and validation of the Body Concealment Scale for Scleroderma (BCSS). *Arthritis Care and Research*. doi:10.1002/acr.22819
- Kopel, S. J., Eiser, C., Cool, P., & Carter, S. R. (1998). Brief report: Assessment of body image in survivors of childhood cancer. *Journal of Pediatric Psychology*, 23, 141–147.
- Kutlesic, V., Williamson, D. A., Gleaves, D. H., Barbin, J. M., & Murphy-Eberenz, K. P. (1998). The interview for the diagnosis of eating disorders—IV: Application to DSM-IV diagnostic criteria. *Psychological Assessment*, 10, 41–48. doi:10.1037/1040-3590.10.1.41
- Lawrence, J. W., Heinberg, L. J., Roca, R., Munster, A., Spence, R., & Fauerbach, J. A. (1998). Development and validation of the satisfaction with appearance scale: Assessing body image among burn-injured patients. *Psychological Assessment*, 10, 64–70.
- Mayville, S. B., Williamson, D. A., White, M. A., Netemeyer, R. G., & Drab, D. L. (2002). Development of the muscle appearance satisfaction scale: A self-report measure for the assessment of muscle dysmorphia symptoms. *Assessment*, 9, 351–360.
- McCreary, D. R., & Sasse, D. K. (2000). An exploration of the drive for muscularity in adolescent boys and girls. *Journal of American College Health*, 48, 297–304.
- McDermott, E., Moloney, J., Rafter, N., Keegan, D., Byrne, K., Doherty, G. A., ... Mulcahy, H. E. (2014). The body image scale: A simple and valid tool for assessing body image dissatisfaction in

- inflammatory bowel disease. *Inflammatory Bowel Diseases*, 20, 286–290. doi:10.1097/01.MIB.0000438246.68476.c4
- Petrie, T. A., Tripp, M. M., & Harvey, P. (2002). Factorial and construct validity of the body parts satisfaction scale-revised: An examination of minority and nonminority women. *Psychology of Women Quarterly*, 26, 213–221.
- Pook, M., Tuschen-Caffier, B., & Brähler, E. (2008). Evaluation and comparison of different versions of the body shape questionnaire. *Psychiatry Research*, 158, 67–73.
- Probst, M., Vandereycken, W., Van Coppenolle, H., & Pieters, G. (1995). Body size estimation in eating disorder patients: Testing the video distortion method on a life-size screen. *Behaviour Research and Therapy*, 33, 985–990. doi:10.1016/0005-7967(95)00037-X
- Pruzinsky, T. (2004). Enhancing quality of life in medical populations: A vision for body image assessment and rehabilitation as standards of care. *Body Image*, 1, 71–81.
- Reas, D. L., Whisenhunt, B. L., Netemeyer, R., & Williamson, D. A. (2002). Development of the body checking questionnaire: A self-report measure of body checking behaviors. *International Journal of Eating Disorders*, 31, 324–333.
- Rosen, J. C., Srebnik, D., Saltzberg, E., & Wendt, S. (1991). Development of a body image avoidance questionnaire. *Psychological Assessment*, 3, 32–37.
- Shafran, R., & Fairburn, C. G. (2002). A new ecologically valid method to assess body size estimation and body size dissatisfaction. *International Journal of Eating Disorders*, 32, 458–465. doi:10.1002/eat.10097
- Spangler, D. L., & Stice, E. (2001). Validation of the beliefs about appearance scale. *Cognitive Therapy and Research*, 25, 813–827. doi:10.1023/A:1012931709434
- Stice, E., & Agras, S. (1998). Predicting onset and cessation of bulimic factors during adolescence. *Behavior Therapy*, 29(2), 257–276.
- Thompson, J. K., Fabian, L. J., Moulton, D. O., Dunn, M. E., & Altabe, M. N. (1991). Development and validation of the physical appearance related teasing scale. *Journal of Personality Assessment*, 56(3), 513–521.
- Thompson, J. K., Roehrig, M., Cafri, G., & Heinberg, L. J. (2005). Assessment of body image disturbance. In J. E. Mitchell & C. B. Peterson (Eds.), *Assessment of eating disorders* (pp. 175–202). New York, NY: Guilford Press.
- Thompson, J. K., van den Berg, P., Roehrig, M., Guarda, A. S., & Heinberg, L. J. (2004). The Sociocultural Attitudes Towards Appearance Scale-3 (SATAQ-3): Development and validation. *International Journal of Eating Disorders*, 35, 293–304.
- Thompson, M. A., & Gray, J. J. (1995). Development and validation of a new body-image assessment scale. *Journal of Personality Assessment*, 64, 258–269.
- Veron-Guidry, S., & Williamson, D. A. (1996). Development of a body image assessment procedure for children and preadolescents. *International Journal of Eating Disorders*, 20, 287–293. doi:10.1002/(SICI)1098-108X(199611)20:3<287::AID-EAT8>3.0.CO;2-K
- Williamson, D. A., Davis, C. J., Bennett, S. M., Goreczny, A. J., & Gleaves, D. H. (1989). Development of a simple procedure for assessing body image disturbances. *Behavioral Assessment*, 11, 433–446.
- Yagil, Y., Geller, S., Sidi, Y., Tirosh, Y., Katz, P., & Nakache, R. (2015). The implications of body-image dissatisfaction among kidney-transplant recipients. *Psychology, Health & Medicine*, 20, 955–962.

Anxiety and Skin Disease

Laura J. Dixon and Sara M. Witcraft

Department of Psychology, University of Mississippi, Oxford, MS, USA

Skin represents the largest organ of the human body, and collectively, skin (dermatology) diseases are among the most widespread illnesses in the world. Skin disease affects all ages and encompasses many conditions, including eczema, psoriasis, acne, alopecia, hyperhidrosis, urticaria (hives), fungal skin diseases, cellulitis, viral warts, skin cancer, and rosacea. Given that dermatology is seldom linked to fatality, the impact of skin disease is often underestimated. However, skin diseases represent the fourth leading global cause of nonfatal burden and are associated with significant psychosocial impairment and high rates of psychological comorbidity (Dalgard et al., 2015; Hay et al., 2014). Perhaps unsurprisingly, given the well-known link between the mind and skin, many skin diseases have a psychological component. Albeit simplified, skin and psychological reactions are posited to be connected through multifaceted responses from the psychoneuroimmunological systems. Psychodermatology addresses this skin–mind connection, and although it is a relatively new subfield, psychodermatology has received increased attention over the past two decades.

Psychodermatological (psychocutaneous) disorders have been classified into three categories—psychophysiologic disorders, primary psychiatric disorders, and secondary psychiatric disorders (Koo & Lee, 2003). Psychophysiologic disorders characterize skin conditions (e.g., acne, eczema) that may be triggered or worsened by psychological factors, such as stress and anxiety. In primary psychiatric disorders, psychopathology contributes to the manifestation of skin symptoms. Examples of primary psychiatric disorders include trichotillomania and delusions of parasitosis. Lastly, individuals affected by secondary psychiatric disorders experience emotional problems as a result of the disfigurement and impairment associated with skin disease. For instance, an individual with vitiligo or alopecia areata may experience depression or social anxiety symptoms due to their visible skin symptoms.

Psychological conditions are catalogued by the Diagnostic and Statistical Manual of Mental Health Disorders (DSM) (American Psychiatric Association [APA], 2013). Of the mental health disorders, anxiety represents one of the most common mental health problems among dermatology patients (Dalgard et al., 2015; Picardi, Amerio, et al., 2004). Anxiety is an

adaptive component of the normal human experience, but it becomes maladaptive when it is persistent, distressing, pervasive, and interferes with everyday life. According to recent studies, as many as 52–70% of dermatology patients report moderate to severe clinical anxiety symptoms (Gascón et al., 2012; Rasouljan, Ebrahimi, Zare, & Taherifar, 2010), and the prevalence rate of anxiety disorders ranges between 15 and 17% in dermatology patients (Dalgard et al., 2015; Picardi, Abeni, et al., 2004). Furthermore, studies have found significantly higher anxiety symptoms in dermatology samples compared with controls (Fleming et al., 2017; Pärna, Aluoja, & Kingo, 2015). In addition to worsening symptoms of skin disease, anxiety symptoms have been associated with a number of adverse outcomes for dermatology patients, such as the use of poor coping strategies, low quality of life, loss of life meaning, and poor social functioning (Leibovici et al., 2010; Mazzotti et al., 2012). While it is possible that anxiety symptoms may be separate and unrelated to skin symptoms, it is likely that in many cases, these anxiety symptoms may operate within the framework of psychodermatological disorders. In particular, anxiety may be integral in psychophysiological disorders, with the cognitive, emotional, and physiological symptoms of anxiety exacerbating or triggering skin symptoms, or to secondary psychiatric disorders, such as an individual experiencing social or health anxiety symptoms in response to their dermatological condition.

Though anxiety refers to a broad syndrome, the most recent editions of the DSM have recognized different types of clinical anxiety including social anxiety disorder (SAD), generalized anxiety disorder (GAD), health anxiety, posttraumatic stress disorder, obsessive-compulsive disorder (OCD), and panic disorder/agoraphobia. Given the high prevalence rates and costs of clinical anxiety in dermatology patients, it is worthwhile to further explore these disorders in relation to skin disease.

Social Anxiety Disorder (Social Phobia)

The primary feature of SAD (formally known as social phobia) is the fear of being judged, rejected, or negatively evaluated in social situations (APA, 2013). Individuals with SAD experience anxiety in situations or in anticipation of a wide range of situations where they perceive that they may act, speak, or appear foolish, silly, stupid, or incompetent in front of others. SAD is one of the most prevalent and costly mental disorders, affecting 7.4% of the US population each year (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Given the visible nature of skin conditions and potential disfigurement, dermatology patients may be highly susceptible to social anxiety and fear of negative evaluation. Indeed, 33.4% of dermatology outpatients reported clinically significant social anxiety (Montgomery, Norman, Messenger, & Thompson, 2016), and dermatology patients report significantly greater social anxiety symptoms compared with healthy adults (Salman, Kurt, Topcuoglu, & Demircay, 2016). In addition, symptoms of SAD, such as blushing, sweating, and trembling, overlap with various skin diseases. For instance, Davidson, Foa, Connor, and Churchill (2002) found that 24.8–32.3% of individuals with SAD met criteria for hyperhidrosis (excessive sweating).

Social anxiety symptoms may contribute to positive feedback loop among individuals with certain skin diseases, such as rosacea, eczema, acne, or hyperhidrosis. For instance, anxiety in social situations may trigger or aggravate skin symptoms, and then an individual becomes anxious or embarrassed about experiencing visible symptoms, which further exacerbates skin symptoms. Conversely, in long-term skin conditions like vitiligo, the skin changes over time, and situational social anxiety may not have a strong impact on the course of the disease. Nevertheless, individuals with vitiligo may experience embarrassment, shame, and social

anxiety due to the visible distortions of their skin. Studies have found that social anxiety symptoms are associated with greater impairment and disability among patients with hyperhidrosis (Lessa Lda et al., 2014), vitiligo (Salman et al., 2016), psoriasis (Schneider, Heuft, & Hockmann, 2013), and acne (Salman et al., 2016). Despite the clear connections between SAD and skin disease, there is remarkably limited research in this area. Further research examining prevalence rates, mechanisms, and treatments for social anxiety among dermatology patients is warranted as this work could improve the identification and treatment of SAD and ultimately, prevent long-term personal and societal costs of this mental disorder.

Generalized Anxiety Disorder

GAD is characterized by persistent, uncontrollable, excessive worry across multiple domains (e.g., health, finances, daily routine; APA, 2013). Individuals with GAD may worry about their health symptoms, but as compared with health anxiety (see below), they also experience pathological worry in other areas in their life. GAD is a relatively common anxiety disorder, presenting in 6.2% of the general population (Kessler et al., 2012). Yet, higher rates of GAD have been observed in dermatology samples. For instance, Woodruff, Higgins, Du Vivier, and Wessely (1997) examined medical records of dermatology patients referred to a psychiatrist and found that 24.8% met criteria for mild GAD and 8.7% met criteria for severe GAD. Although the psychology–dermatology literature is composed of many studies examining “general anxiety” or broadly defined symptoms of distress and anxiety, fewer studies have specifically examined the GAD syndrome. In particular, research in this area has primarily focused on the role of GAD and pathological worry in psoriasis. One study demonstrated excessive worry symptoms were evidenced by 38% of patients with psoriasis, while 25% of the entire sample reported symptom levels consistent with a clinical diagnosis of GAD (Fortune, Richards, Main, & Griffiths, 2000). Beyond the high prevalence rates of GAD in psoriasis, pathological worry has been associated with greater burden of physiological and psychological symptoms. Pathological worry has been found to negatively affect the body and, specifically, has been associated with slower clearance of psoriasis symptoms (Fortune, Richards, Kirby, et al., 2003). Finally, in addition to direct associations with disease symptoms, GAD symptoms have been linked to worse illness-related beliefs, greater disability, and worse coping strategies in psoriasis patients (Fortune et al., 2000; Fortune, Richards, Corrin, et al., 2003). Given the common occurrence of GAD in the general population coupled with the findings revealed by the psoriasis literature, additional research examining GAD and pathological worry in other skin diseases is necessary.

Health Anxiety

Health anxiety, also known as illness anxiety disorder and hypochondriasis, is a preoccupation having or acquiring a serious illness. These concerns are accompanied by either avoidance of medical visits altogether or, at the other extreme, excessive medical visits and tests despite being told that there is nothing physically wrong (APA, 2013). A recent epidemiological study revealed the lifetime prevalence rate of illness anxiety disorder was 5.7% (Sunderland, Newby, & Andrews, 2013). Unfortunately, health anxiety is often unidentified in medical settings, and medical providers may ineffectively, albeit unintentionally, respond to patients’ anxieties about their health problems by offering reassurance and conducting unnecessary medical tests (Tyrer,

Eilenberg, Fink, Hedman, & Tyrer, 2016). Very few studies have investigated health anxiety in dermatology patients, and to obtain more information about this phenomena, Long and Elpern (2017) conducted a study examining health anxiety in dermatology outpatients. They found that 31% of dermatology patients reported that they worried “a lot” about a health problem, 17% reported that they worried the problem was more serious than a doctor described, and a total of 46% expressed some degree health anxiety. The findings from this study suggest that health anxiety is particularly relevant for dermatology patients, and given these staggering numbers, further research is needed to explicate health anxiety in dermatology samples.

Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) is characterized by anxiety and distress following a potentially life-threatening event such as a car wreck, natural disaster, or a physical or sexual assault. Further, the anxiety is associated with recurrent, involuntary, and distressing memories of the trauma; attempts to avoid cues that remind the individual of the trauma and physiological arousal (e.g., hypervigilance, exaggerated startle reaction, panic symptoms); and increased negative mood and cognitions (APA, 2013). Despite the fact that many individuals will experience at least one potentially traumatic event in their lifetime, PTSD has a relatively low lifetime prevalence rate of 8.0% in the general population (Kessler et al., 2012). Interestingly, skin disease has been found to be higher among individuals with PTSD compared with those without PTSD. A recent review of co-occurring PTSD and dermatology suggested that skin conditions are common among individuals with PTSD due to the increased autonomic and sympathetic arousal, which activates skin symptoms (e.g., sweating; Gupta, Jarosz, & Gupta, 2017). Notably, in a large case–control trial, patients commonly reported psoriasis, alopecia, skin allergies, and other skin diseases, and individuals with PTSD (versus without PTSD) were twice as likely to have dermatology conditions (Britvić et al., 2015).

Obsessive–Compulsive Disorder

OCD is composed of two components—(a) uncontrollable, unwanted, recurrent thoughts that cause distress (obsessions) and (b) mental acts or ritualistic behaviors that are instigated by obsessions and are conducted to reduce anxiety (compulsions; APA, 2013). OCD is a chronic, though relatively rare, disorder, with a lifetime prevalence rate of 2.7% (Kessler et al., 2012). Nevertheless, the prevalence of OCD in dermatology patients is much higher, with studies finding that between 9.1 and 24.7% of patients with skin disease meet criteria for OCD (Demet et al., 2005; Sheikhmoonesi, Hajheidari, Masoudzadeh, Mohammadpour, & Mozaffari, 2014). Interestingly, whereas most of the empirical work examining anxiety disorders has been conducted within specific dermatology conditions (e.g., psoriasis), studies investigating OCD have done so with an unselected dermatological sample. OCD symptoms can interact with skin symptoms in several ways, and within OCD, obsessions or compulsions may relate to or be in response to skin symptoms. The most common obsessions reported by dermatology patients include somatic obsessions, pathological doubt, and contamination (Demet et al., 2005; Ebrahimi, Salehi, & Tafti, 2007). These obsessions may interfere with management of skin symptoms (e.g., doubt about taking medication) and may increase distress about the status of skin symptoms (e.g., intrusive thoughts about the severity of a skin lesion or

breakout). The most common compulsions reported by dermatology patients include washing and checking (Demet et al., 2005). Chronic washing behaviors may lead to dry or raw skin and even abrasions, which may directly worsen skin symptoms and contribute to excoriation and skin picking. Similarly, though the study did not specify the type of checking behaviors, repeated checking of skin symptoms may result in decreased memory confidence and greater reactivity to perceived changes in skin status. Given the focus on skin and somatic symptoms, these individuals often present to dermatology or primary care clinics rather than seeking psychological or psychiatric services.

Panic and Agoraphobia

A panic attack is a sudden surge of anxiety and distress that peaks within 10 min and includes at least four of the following symptoms: palpitations/pounding heart, sweating, trembling or shaking, sensations of shortness of breath or smothering, feeling of choking, chest pain or discomfort, feeling dizzy or faint, nausea or abdominal distress, derealization/depersonalization, fear of losing control or going crazy, numbness or tingling sensations, and chills or hot flashes. Panic attacks may be cued by a specific event or environment (e.g., stressful event, needles, crowded area) or may occur “out of the blue” and for no apparent reason. Epidemiological work has estimated that 28.3% of individuals experience one panic attack at some point in their life (Kessler et al., 2006). Although panic attacks are not inherently tied to pathology, individuals who worry about having another panic attack or change their life to avoid having a panic attack may meet criteria for panic disorder. In addition, individuals with other anxiety disorders may experience panic attacks when they come in contact with a feared situation (e.g., social situations in SAD) and individuals with agoraphobia fear situations (e.g., public transportation, being in a crowd) because they are afraid they may not be able to escape or get help if they develop panic-like symptoms (APA, 2013). There is little literature exploring panic attacks, panic disorder, and agoraphobia in dermatology patients. One existing study found that patients with chronic skin conditions experienced significantly greater panic and agoraphobia symptoms than individuals in the control group (Pärna et al., 2015). Future research is required to understand panic and agoraphobia in dermatology. For instance, the avoidance and physiological symptoms characteristic of these disorders may amplify impairment and skin symptoms for individuals with skin disease. Moreover, it is likely that symptoms of these disorders may co-occur with other anxiety-related disorders as is commonly observed in the general population.

Future Directions

As the empirical literature of and clinical services for psychodermatology continue to evolve, anxiety disorders represent an important area for further development. Although anxiety disorders are common and known to contribute to worse outcomes in skin disease, there is a significant unmet need for treatment for individuals with co-occurring dermatological and anxiety conditions, as well as large gaps in the empirical literature. Regarding clinical services, psychiatric symptoms are often unrecognized in dermatology patients (Picardi, Abeni, et al., 2004), and even fewer patients receive psychotherapeutic interventions in routine dermatology care (Fritzsche et al., 2001). Several studies have shown promising outcomes for the inclusion of psychosocial treatments in standard dermatology care, and in particular, adjunctive cognitive

behavioral treatments have been found to reduce the severity of dermatological conditions, enhance quality of life, and decrease anxiety symptoms (Lavda, Webb, & Thompson, 2012). With regard to research, studies investigating the prevalence rates and risk factors for the different anxiety disorders across skin disease would be beneficial for improving the awareness and identification of anxiety disorders in routine care. Moreover, investigating mechanisms contributing to the interaction between anxiety and skin symptoms would enhance the understanding of psychodermatology processes and has the potential to inform the development of effective interventions to prevent and reduce anxiety in dermatology patients.

Cross References

Psychoneuroimmunology: Immune markers of psychopathology
Generalized Anxiety Disorder (GAD) and health
Posttraumatic stress disorder (PTSD)
Cognitive Behavior Therapy (CBT), sleep, mood disorders, and health

Author Biographies

Laura J. Dixon, PhD, is assistant professor at the University of Mississippi, director of the Health and Anxiety Research and Treatment Laboratory, and licensed clinical psychologist. Dr. Dixon has published numerous scientific articles on the etiology and treatment of anxiety-related disorders and received an ADAA early career award.

Sara M. Witcraft is a clinical psychology doctoral student at the University of Mississippi. Sara has published several peer-reviewed articles and book chapters on the treatment, understanding, and underlying factors of anxiety and related disorders. Her current research focuses on transdiagnostic vulnerabilities that underlie anxiety disorders.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)* (Vol. 5). Washington, DC: American Psychiatric Publishing.
- Britvić, D., Antičević, V., Kaliterna, M., Lušić, L., Beg, A., Brajević-Gizdić, I., ... Pivac, N. (2015). Comorbidities with Posttraumatic Stress Disorder (PTSD) among combat veterans: 15 years postwar analysis. *International Journal of Clinical and Health Psychology, 15*, 81–92.
- Dalgard, F. J., Gieler, U., Tomas-Aragones, L., Lien, L., Poot, F., Jemec, G. B., ... Kupfer, J. (2015). The psychological burden of skin diseases: A cross-sectional multicenter study among dermatological out-patients in 13 European countries. *Journal of Investigative Dermatology, 135*, 984–991.
- Davidson, J. R., Foa, E. B., Connor, K. M., & Churchill, L. E. (2002). Hyperhidrosis in social anxiety disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 26*, 1327–1331.
- Demet, M. M., Devci, A., Taskin, E. O., Ermertcan, A. T., Yurtsever, F., Deniz, F., ... Ozturkcan, S. (2005). Obsessive-compulsive disorder in a dermatology outpatient clinic. *General Hospital Psychiatry, 27*, 426–430.
- Ebrahimi, A. A., Salehi, M., & Tafti, K. A. (2007). Obsessive-compulsive disorder in dermatology outpatients. *International Journal of Psychiatry in Clinical Practice, 11*, 218–221.
- Fleming, P., Bai, J. W., Pratt, M., Sibbald, C., Lynde, C., & Gulliver, W. P. (2017). The prevalence of anxiety in patients with psoriasis: A systematic review of observational studies and clinical trials. *Journal of the European Academy of Dermatology and Venereology: JEADV, 31*, 798–807.

- Fortune, D. G., Richards, H. L., Corrin, A., Taylor, R. J., Griffiths, C. E. M., & Main, C. J. (2003). Attentional bias for psoriasis-specific and psychosocial threat in patients with psoriasis. *Journal of Behavioral Medicine*, 26, 211–224.
- Fortune, D. G., Richards, H. L., Kirby, B., McElhone, K., Markham, T., Rogers, S., ... Griffiths, C. E. (2003). Psychological distress impairs clearance of psoriasis in patients treated with photochemotherapy. *Archives of Dermatology*, 139, 752–756.
- Fortune, D. G., Richards, H. L., Main, C. J., & Griffiths, C. E. (2000). Pathological worrying, illness perceptions and disease severity in patients with psoriasis. *British Journal of Health Psychology*, 5, 71–82.
- Fritzsche, K., Ott, J., Zschocke, I., Scheib, P., Burger, T., & Augustin, M. (2001). Psychosomatic liaison service in dermatology. *Dermatology*, 203, 27–31.
- Gascón, M. R. P., Ribeiro, C. M., Bueno, L. M. d. A., Benute, G. R. G., Lucia, M. C. S. d., Rivitti, E. A., & Festa Neto, C. (2012). Prevalence of depression and anxiety disorders in hospitalized patients at the dermatology clinical ward of a university hospital. *Anais Brasileiros de Dermatologia*, 87, 403–407.
- Gupta, M. A., Jarosz, P., & Gupta, A. K. (2017). Posttraumatic stress disorder (PTSD) and the dermatology patient. *Clinics in Dermatology*, 35, 260–266.
- Hay, R. J., Johns, N. E., Williams, H. C., Bolliger, I. W., Dellavalle, R. P., Margolis, D. J., ... Wulf, S. K. (2014). The global burden of skin disease in 2010: An analysis of the prevalence and impact of skin conditions. *Journal of Investigative Dermatology*, 134, 1527–1534.
- Kessler, R. C., Chiu, W. T., Jin, R., Ruscio, A. M., Shear, K., & Walters, E. E. (2006). The epidemiology of panic attacks, panic disorder, and agoraphobia in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 63, 415–424.
- Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H. U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research*, 21, 169–184.
- Koo, J. Y., & Lee, C. S. (2003). General approach to evaluating psychodermatological disorders. *Basic and Clinical Dermatology*, 25, 1–12.
- Lavda, A. C., Webb, T. L., & Thompson, A. R. (2012). A meta-analysis of the effectiveness of psychological interventions for adults with skin conditions. *British Journal of Dermatology*, 167, 970–979.
- Leibovici, V., Canetti, L., Yahalomi, S., Cooper-Kazaz, R., Bonne, O., Ingber, A., & Bachar, E. (2010). Well-being, psychopathology and coping strategies in psoriasis compared with atopic dermatitis: A controlled study. *Journal of the European Academy of Dermatology and Venereology: JEADV*, 24, 897–903.
- Lessa Lda, R., Luz, F. B., De Rezende, R. M., Duraes, S. M., Harrison, B. J., De Menezes, G. B., & Fontenelle, L. F. (2014). The psychiatric facet of hyperhidrosis: Demographics, disability, quality of life, and associated psychopathology. *Journal of Psychiatric Practice*, 20, 316–323.
- Long, V., & Elpern, D. J. (2017). Health anxiety in dermatology. *International Journal of Dermatology*, 56, 968–971.
- Mazzotti, E., Mastroeni, S., Lindau, J., Lombardo, G., Farina, B., & Pasquini, P. (2012). Psychological distress and coping strategies in patients attending a dermatology outpatient clinic. *Journal of the European Academy of Dermatology and Venereology: JEADV*, 26, 746–754.
- Montgomery, K., Norman, P., Messenger, A. G., & Thompson, A. R. (2016). The importance of mindfulness in psychosocial distress and quality of life in dermatology patients. *British Journal of Dermatology*, 175, 930–936.
- Pärna, E., Aluoja, A., & Kingo, K. (2015). Quality of life and emotional state in chronic skin disease. *Acta Dermato-Venereologica*, 95, 312–316.
- Picardi, A., Abeni, D., Mazzotti, E., Fassone, G., Lega, I., Ramieri, L., ... Pasquini, P. (2004). Screening for psychiatric disorders in patients with skin diseases. *Journal of Psychosomatic Research*, 57, 219–223.

- Picardi, A., Amerio, P., Baliva, G., Barbieri, C., Teofoli, P., Bolli, S., ... Abeni, D. (2004). Recognition of depressive and anxiety disorders in dermatological outpatients. *Acta Dermato-Venereologica*, 84, 213–217.
- Rasoulzadeh, M., Ebrahimi, A. A., Zare, M., & Taherifar, Z. (2010). Psychiatric morbidity in dermatological conditions. *International Journal of Psychiatry in Clinical Practice*, 14, 18–22.
- Salman, A., Kurt, E., Topcuoglu, V., & Demircay, Z. (2016). Social anxiety and quality of life in vitiligo and acne patients with facial involvement: A cross-sectional controlled study. *American Journal of Clinical Dermatology*, 17, 305–311.
- Schneider, G., Heuft, G., & Hockmann, J. (2013). Determinants of social anxiety and social avoidance in psoriasis outpatients. *Journal of the European Academy of Dermatology and Venerology: JEADV*, 27, 383–386.
- Sheikhmoonesi, F., Hajheidari, Z., Masoudzadeh, A., Mohammadpour, R. A., & Mozaffari, M. (2014). Prevalence and severity of obsessive-compulsive disorder and their relationships with dermatological diseases. *Acta Medica Iranica*, 52, 511.
- Sunderland, M., Newby, J. M., & Andrews, G. (2013). Health anxiety in Australia: Prevalence, comorbidity, disability and service use. *British Journal of Psychiatry*, 202, 56–61.
- Tyrer, P., Eilenberg, T., Fink, P., Hedman, E., & Tyrer, H. (2016). Health anxiety: The silent, disabling epidemic. *British Medical Journal*, 353, i2250.
- Woodruff, P., Higgins, E., Du Vivier, A., & Wessely, S. (1997). Psychiatric illness in patients referred to a dermatology-psychiatry clinic. *General Hospital Psychiatry*, 19, 29–35.

Suggested Reading

- Bewley, A., Taylor, R. E., & Reichenberg, J. S. (2014). In M. Magid (Ed.), *Practical psychodermatology*. Oxford, UK: Wiley.
- Gupta, M. A., & Gupta, A. K. (2013). A practical approach to the assessment of psychosocial and psychiatric comorbidity in the dermatology patient. *Clinics in Dermatology*, 31, 57–61.
- Lavda, A. C., Webb, T. L., & Thompson, A. R. (2012). A meta-analysis of the effectiveness of psychological interventions for adults with skin conditions. *British Journal of Dermatology*, 167, 970–979.

Insomnia

Todd A. Smitherman and Daniel G. Rogers

Department of Psychology, University of Mississippi, Oxford, MS, USA

Diagnosis and Epidemiology

Insomnia is sleep disorder broadly characterized by dissatisfaction with sleep amount or quality as manifested by recurrent difficulty initiating or maintaining sleep and causing significant distress or daytime impairment (e.g., fatigue, difficulty concentrating, irritability) (Morin & Benca, 2012; Rybarczyk, Lund, Garroway, & Mack, 2013). Sleep maintenance difficulties include waking frequently during sleep or being unable to quickly return back to sleep after waking. Both the International Classification of Sleep Disorders—Third Edition (ICSD-3) (American Academy of Sleep Medicine, 2014) and the Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition (DSM-5) (American Psychiatric Association, 2013) also require that sleep difficulties occur despite ample opportunity for sleep, have a frequency of at least 3 days/week for at least 3 months, and are not attributable to another medical or mental disorder. In research settings, regularly obtaining <6 hr sleep per night and being awake for >30 min while trying to fall or stay asleep are commonly employed as diagnostic thresholds.

Prevalence and Impact

Insomnia is one of the most common medical complaints in primary care settings. Over 30% of adults report at least one insomnia-related sleep complaint, 10–15% report daytime impairment resulting from poor sleep, and 6–10% of adults have symptoms that meet formal diagnostic criteria for insomnia (Morin & Jarrin, 2013). Without intervention, 40–60% of individuals with chronic insomnia report similar symptoms a year later (Pillai, Roth, & Drake, 2015).

Complaints of poor sleep are more common in women than men (Zhang & Wing, 2006) and among older adults (Morphy, Dunn, Lewis, Boardman, & Croft, 2007). Older adults experience more fragmented sleep and earlier awakening, awaken to external stimuli more easily, and are more prone to disruptions of normal sleep than young adults (Fetveit, 2009).

Older adults also are more likely to experience comorbid medical conditions or disturbed sleep as a side effect of medication, which obfuscates the role of normal aging in sleep impairment (Vitiello, 2006).

Insomnia is associated with worsened cognitive functioning, particularly deficits in working memory, episodic memory, and executive functioning (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012). Insomnia results in tens of billions of dollars of lost productivity in the United States annually, mostly as a result of impaired work performance (Kessler et al., 2011), as well as significant direct and indirect costs to individuals (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009).

Changes in Conceptualization of Insomnia

The most recent editions of the DSM and ICSD eliminated nonrestorative sleep as a symptom of insomnia (termed “insomnia disorder” in DSM-5 and “chronic insomnia” in ICSD-3) as this symptom is often poorly defined and not specific to insomnia. Classification task forces collaborated to ensure similar diagnostic criteria, including frequency and duration of insomnia, across diagnostic manuals. Epidemiological studies adhering to these updated criteria are needed.

Previous editions of insomnia diagnostic manuals made distinctions between “primary” and “secondary” insomnia, the latter of which referred to insomnia comorbid with another medical or psychiatric disorder. This distinction proved problematic in several ways: (a) limited understanding of mechanistic pathways prevents conclusions about the direction of causality, (b) use of the term “secondary insomnia” may contribute to potential for inadequate treatment, and (c) regardless of terminology, various insomnia presentations share numerous behavioral and cognitive characteristics and similar treatment approaches (NIH, 2005). As such, recent nosologies have eliminated this distinction.

Risk Factors and Comorbidities

Onset of insomnia is often conceptualized from a diathesis–stress perspective, in which inherited predisposing factors interact with precipitating events to disrupt biological processes involved in sleep. The sleep–wake cycle is largely determined by circadian and homeostatic processes, the former being regulated by external light cycles and the latter as a function of a sleep drive that increases the longer an individual remains awake. Diatheses involved in insomnia include genetically transmitted factors that predispose one to physiological hyperarousal throughout the day and night (e.g., family history of sleep disorders, negative affectivity, high susceptibility to stress; Riemann et al., 2010; Vgontzas et al., 2001).

Precipitating events among susceptible individuals include a broad range of medical, environmental, and psychological factors. Medical conditions such as chronic pain, heart disease, chronic obstructive pulmonary disease (COPD), and movement disorders can interfere with sleep onset or maintenance (Smith & Haythornthwaite, 2004). Environmental factors include those in the immediate sleep environment (e.g., excessive light, noise, uncomfortable ambient temperature) and more distal external variables (e.g., life stressors such as a new job or deadlines, changes in an individual’s sleep schedule such as from jet lag). Psychological factors encompass comorbid psychiatric disorders and both behaviors and cognitions that contribute to disturbed sleep. Individuals with insomnia are up to five times more likely than those without insomnia to experience symptoms of anxiety and depression, both of which disrupt normal sleep patterns (Pearson, Johnson, & Nahin, 2006). This relationship appears

bidirectional in nature, such that insomnia may develop before or after the psychiatric disorder. Use of various substances can also interfere with sleep (Roth et al., 2006): nicotine and caffeine can delay sleep onset, and alcohol can disrupt sleep maintenance and quality. Sleep disturbance may also result from use or withdrawal of controlled substances such as alcohol, anxiolytics, opioids, sedatives, and stimulants.

Beyond their role in the onset of acute episodes of insomnia, psychological factors operate also to maintain insomnia, such that an individual's response to difficulty sleeping may inadvertently perpetuate dysregulated sleep and contribute to development of chronic insomnia (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Individuals experiencing insomnia often begin to worry about their ability to sleep and the functional consequences of too little sleep, and this preoccupation with sleep contributes to physiological arousal that interferes with sleep. Similarly, compensatory behavioral responses are generally designed to increase opportunity for sleep (e.g., remaining in bed if unable to sleep, sleeping in on weekends, utilizing daytime napping) but inadvertently maintain insomnia by conditioning the individual to be awake at night while attempting to sleep. Ultimately, chronification of insomnia thus often occurs through repeated pairing of cognitive and cortical arousal with sleep-related stimuli.

Assessment

Assessment of insomnia focuses principally on gathering information about sleep-related behaviors and cognitions, symptoms and functional impairment, and perpetuating factors that may maintain insomnia. In most cases, gathering this information via interview and daily sleep diaries is sufficient for making the diagnosis and developing a treatment plan.

Clinical Interview

Existing structured diagnostic interviews for insomnia are few in number, not validated using recent insomnia diagnostic criteria, and time consuming, and thus infrequently used outside of research settings or sleep clinics. The clinical interview focuses principally on gathering detailed information about one's sleep history and screening for the aforementioned comorbid conditions. In addition to querying specific diagnostic criteria and functional impairment, detailed information is gathered about the history of the sleep problem (onset, course, duration), typical sleep-wake pattern, and perpetuating factors. The latter includes identifying behaviors (e.g., daytime napping, staying in bed when unable to sleep, non-sleep activities while in bed), cognitions (e.g., preoccupation with sleep, catastrophizing about the effects of poor sleep), and medications/substances that condition physiological arousal when attempting to sleep.

Daily Sleep Diaries

Self-monitoring via use of daily sleep diaries (logs) is invaluable for quantifying one's sleep schedule at baseline and throughout treatment. At a minimum, sleep diaries should solicit information pertaining to time in bed (TiB), time out of bed, time to fall asleep (sleep latency [SL]), total sleep time (TST), and time spent napping. Some diaries include entries for bed-time, wake time after sleep onset (WASO), medication/alcohol consumption, lights on/off times, and ratings of sleep quality. From these self-monitoring data, sleep efficiency (SE) is calculated ($\text{average TST} / \text{average TiB} \times 100$), representing the percentage of time in bed that

one is asleep. Patients are instructed to complete entries shortly after awakening each morning, and a minimum of 2 weeks of self-monitoring is recommended for each assessment period.

Other Means of Assessment

In most cases a thorough clinical interview and daily sleep diaries provide ample data to inform diagnosis and treatment, but other methods of assessment may be used to supplement the interview and diaries.

Questionnaires

The most widely researched sleep questionnaire is the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1998), which inquires about one's sleep pattern over the last month and solicits Likert-type ratings of various sleep disturbances. Items are aggregated into 7 component scores and then into a total score that ranges from 0 to 21; scores >5 are indicative of poor sleep quality. However, scoring the PSQI can be difficult, and, as it is not specific to insomnia, elevated scores can reflect difficulties stemming from other sleep disorders.

The Insomnia Severity Index (ISI) (Morin, Belleville, Bélanger, & Ivers, 2011) is the most widely used questionnaire specific to insomnia. This seven-item Likert-type measure quantifies severity of insomnia as a function of symptoms and impairment experienced over the last month. Total scores range from 0 to 28, with higher scores indicative of greater insomnia. The ISI is sensitive to treatment effects and can be utilized to identify insomnia in population samples.

Polysomnography

Polysomnography involves overnight recording of physiological variables during sleep (respiration, electrical activity of the brain) and is usually conducted in a sleep clinic. This form of assessment is not indicated in the overwhelming majority of insomnia presentations. Polysomnography is useful when there is strong reason to suspect breathing (sleep apnea) or movement disorders (e.g., restless leg syndrome) as the cause of poor sleep or when standard treatments fail without a clear reason (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008).

Actigraphy

Actigraphs are ambulatory digital monitoring devices that are worn on the wrist and quantify sleep as a function of movement (via accelerometry) and ambient light. Actigraphy is often used to supplement data from sleep diaries, as the former provides more objective data, or as an alternative to overnight polysomnography (Morganthaler et al., 2007). Several companies produce actigraphs and use proprietary computer algorithms to derive sleep-wake variables.

Treatment

Psychological Interventions

Cognitive behavioral therapy for insomnia (CBT-I) is grounded in principles of learning theory and seeks to modify behaviors and cognitions that inadvertently perpetuate conditioned arousal. CBT-I is a multicomponent intervention that includes some combination of stimulus control, sleep restriction, sleep hygiene education, relaxation training, and cognitive therapy. Stimulus control and sleep restriction appear to be the most potent components of

CBT-I (Schutte-Rodin et al., 2008). Stimulus control involves techniques to reassociate the bedroom with sleep using classical and operant conditioning principles. These include instructions in using the bed only for sleep and sexual activity (e.g., eliminating other activities in bed such as eating or watching TV), eliminating daytime naps, and getting out of bed if unable to fall asleep within 20–30 min. Sleep restriction focuses on consolidating one's sleep by restricting one's TiB to the actual time spent sleeping and then gradually increasing TiB as SE reaches 85% or higher as indexed by daily sleep diaries.

Sleep hygiene is a psychoeducational intervention focused on general habits that promote sleep (e.g., limiting caffeine/alcohol, keeping a comfortable sleep environment and consistent sleep–wake schedule) but is not an efficacious monotherapy (Edinger et al., 2009; Schutte-Rodin et al., 2008). Relaxation training endeavors to reduce contributing physiological arousal and is often used for those who have difficulty relaxing at bedtime or in place of sleep restriction for individuals who have bipolar disorder, in whom sleep restriction is contraindicated due to risk of inducing mania. Finally, cognitive therapy techniques may be included to address dysfunctional cognitions that perpetuate insomnia, such as chronic worries that prevent sleep or unrealistic expectations about sleep.

CBT-I is the most frequently researched and well-established psychological intervention for insomnia. The most recent comprehensive review of its efficacy in adults was provided in a meta-analysis conducted by the Agency for Healthcare Research and Quality (AHRQ) (Brasure et al., 2015). Eighteen randomized controlled trials provided data for pooled outcomes regarding CBT-I versus passive control interventions (e.g., attention control, treatment as usual, wait list, sham control) among the general adult population. Most studies utilized individual CBT-I, though some included alternative modes of delivery (e.g., group or phone administration). On average, CBT-I was associated with a tripled rate of remission (61 vs. 18% for control); substantially greater reductions on both the PSQI and ISI; and superior improvements in SL, TST, WASO, SE, and sleep quality. Many treatment gains appeared to be maintained for months after cessation of treatment. CBT-I also produced favorable outcomes versus control treatments among older adults and among those with comorbid pain conditions, though the evidence was of lower quality as there were fewer trials pertinent to these specific groups.

Other recent meta-analytic reviews have reached similar conclusions regarding the short- and long-term efficacy of CBT-I for insomnia, including insomnia comorbid with other medical and psychiatric conditions (Geiger-Brown et al., 2015; Trauer, Qian, Doyle, Rajaratnam, & Cunningham, 2015; Wu, Appleman, Salazar, & Ong, 2015). Advances in treatment delivery and information technology have facilitated efforts to condense CBT-I into as few as two to four treatment sessions so that it can be more easily integrated into routine medical practice settings (Edinger et al., 2009; Edinger & Sampson, 2003), as well as delivered via the Internet to enhance access and utilization (Zachariae, Lyby, Ritterband, & O'Toole, 2016).

There is insufficient evidence for complementary and alternative medicine approaches (e.g., acupuncture, homeopathy, valerian root) to suitably assess their efficacy for treatment of insomnia (Brasure et al., 2015).

Pharmacotherapy

Pharmacological therapies for insomnia can be broadly delineated into over-the-counter (OTC) medications, prescription medications approved by the Food and Drug Administration (FDA) for treatment of insomnia, and prescription medications with sedating properties that are used “off-label.” In general, the following medications have shown efficacy for short-term treatment of insomnia, though few studies on long-term efficacy or safety exist.

OTC medications

Common OTC medications for insomnia include antihistamines (e.g., hydroxyzine, diphenhydramine, doxylamine succinate) and exogenous melatonin. Antihistamines are frequently used for insomnia despite limited evidence of efficacy, rapid development of tolerance with daily usage, and risk of rebound insomnia following discontinuation. Melatonin has been increasingly used to aid sleep since sedative properties were discovered in the 1970s. Melatonin has demonstrated significant effects in reducing sleep onset latency and increasing TST and sleep quality compared with placebo across several studies (Ferracioli-Oda, Qawasmi, & Bloch, 2013). However, the clinical significance of these effects appears modest at best, with sleep onset latency and TST improved by less than 10 min on average as compared with placebo.

Prescription medications approved for insomnia

The most common medications approved for the treatment of insomnia are benzodiazepine (e.g., estazolam) and non-benzodiazepine hypnotics (e.g., zolpidem [Ambien], eszopiclone [Lunesta]), the latter of which have the strongest evidence among adults in the general population. Both classes of agents are GABA agonists, though the non-benzodiazepines appear to have more selective action at the GABA_A receptor and perhaps better safety profiles. Non-benzodiazepines have been shown to reduce SL and WASO and to increase SE and sleep quality (Becker & Somiah, 2015; Buscemi et al., 2007). Evidence for benzodiazepines is less robust as a function of trial design limitations and significant improvements evidenced only in sleep onset latency (Brasure et al., 2015). The efficacy of hypnotic agents has been called into question relative to risks associated with both short-term and long-term usage. Short-term side effects include drowsiness and fatigue, cognitive impairment, and motor problems, which can be particularly relevant among older adults (Glass, Lancôt, Herrmann, Sproule & Busto, 2005). Long-term use is associated with risk for dependence, misuse, and rebound insomnia, as well as higher rates of dementia and fractures in observational studies (Brasure et al., 2015).

Other approved medications include the melatonin receptor agonists (MRA) (e.g., ramelteon), orexin receptor antagonists (ORA) (e.g., suvorexant), and tricyclic antidepressants (TCA) (e.g., doxepin). Ramelteon does not appear to produce clinically meaningful sleep improvements, similar to conclusions reached regarding exogenous melatonin (Brasure, Fuchs, & Macdonald, 2016). Both ORAs and TCAs have demonstrated efficacy in improving sleep time and reducing WASO, though evidence for TCAs is weaker (Brasure et al., 2015).

Prescription medications not approved for insomnia

In addition to FDA-approved medications for insomnia, several antidepressants (e.g., trazodone, mirtazapine), anticonvulsants (e.g., tiagabine, pregabalin), and antipsychotics have sedating properties and thus are commonly prescribed. In general, the evidence for off-label use of these medications is insufficient as compared with the aforementioned approved prescription medications, and risk of adverse events is high. As such, these agents are recommended only when insomnia is comorbid with another condition for which they are indicated (Buysse, John Rush, & Reynolds, 2017).

CBT-I Versus Pharmacotherapy for Insomnia

The AHRQ review concluded that there was insufficient evidence to directly compare CBT-I and pharmacotherapy but noted that CBT-I carries considerably less risk for adverse effects and physical harm (Brasure et al., 2015). Accordingly, the American College of Physicians'

most recent clinical practice guideline recommended CBT-I as the frontline treatment for adults with insomnia, with medication as a secondary option for those who do not respond to an initial trial of CBT-I (Qaseem, Kansagara, Forcica, Cooke, & Denberg, 2016). As pharmacotherapy is only recommended for short-term use, most drug trials have been only 4–6 weeks in duration, and thus serious long-term adverse effects directly associated with use of pharmacological agents for sleep remain largely unknown.

See Also

Adherence to Behavioral and Medical Regimens; Depression and Comorbidity in Health Contexts; Older Adults and Perspectives for Researchers and Clinicians Working in Health Psychology and Behavioral Medicine; Pain; Treatment: Anxiety and Stress with Chronic Diseases

Author Biographies

Todd A. Smitherman, PhD, is associate professor and director of Clinical Training at the University of Mississippi in Oxford, Mississippi. His research focuses on psychological factors in and behavioral interventions for chronic medical conditions such as migraine and insomnia.

Daniel G. Rogers, BAS, is a graduate student in the University of Mississippi Clinical Psychology Program in Oxford, Mississippi.

References

- American Academy of Sleep Medicine. (2014). *International classification of sleep disorders: Diagnostic and coding manual* (3rd ed.). Darien, IL: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.
- Becker, P. M., & Somiah, M. (2015). Non-benzodiazepine receptor agonists for insomnia. *Sleep Medicine Clinics*, 10, 57–76.
- Brasure, M., Fuchs, E., & Macdonald, R. (2016). Psychological and behavioral interventions for managing insomnia. *Annals of Internal Medicine*, 117, 1–13.
- Brasure, M., MacDonald, R., Fuchs, E., Olson, C. M., Carlyle, M., Diem, S., ... Wilt, T. J. (2015). *Management of insomnia disorder. Comparative effectiveness review 159. (Prepared by the Minnesota evidence-based practice center under contract 290-2012-00016-I.) AHRQ Publication no.15(16)-EHC027-EF*. Rockville, MD: Agency for Healthcare Research and Quality. <https://www.effectivehealthcare.ahrq.gov/topics/insomnia/research>
- Buscemi, N., Vandermeer, B., Friesen, C., Bialy, L., Tubman, M., Ospina, M., ... Witmans, M. (2007). The efficacy and safety of drug treatments for chronic insomnia in adults: A meta-analysis of RCTs. *Journal of General Internal Medicine*, 22, 1335–1350.
- Buysse, D. J., John Rush, A., & Reynolds, C. F. (2017). Clinical management of insomnia disorder. *JAMA*, 318, 1973–1974.
- Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1998). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28, 193–213.

- Daley, M., Morin, C. M., LeBlanc, M., Grégoire, J.-P. P., & Savard, J. (2009). The economic burden of insomnia: Direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep*, 32, 55–64.
- Edinger, J. D., Olsen, M. K., Stechuchak, K. M., Means, M. K., Lineberger, M. D., Kirby, A., & Carney, C. E. (2009). Cognitive behavioral therapy for patients with primary insomnia or insomnia associated predominantly with mixed psychiatric disorders: A randomized clinical trial. *Sleep*, 32, 499–510.
- Edinger, J. D., & Sampson, W. S. (2003). A primary care “friendly” cognitive behavioral insomnia therapy. *Sleep*, 26, 177–182.
- Ferracioli-Oda, E., Qawasmi, A., & Bloch, M. H. (2013). Meta-analysis: Melatonin for the treatment of primary sleep disorders. *PLoS One*, 8, e63773.
- Fetveit, A. (2009). Late-life insomnia: A review. *Geriatrics and Gerontology International*, 9, 220–234.
- Fortier-Brochu, É., Beaulieu-Bonneau, S., Ivers, H., & Morin, C. M. (2012). Insomnia and daytime cognitive performance: A meta-analysis. *Sleep Medicine Reviews*, 16(1), 83–94.
- Geiger-Brown, J. M., Rogers, V. E., Liu, W., Ludeman, E. M., Downton, K. D., & Diaz-Abad, M. (2015). Cognitive-behavioral therapy in persons with comorbid insomnia: A meta-analysis. *Sleep Medicine Reviews*, 23, 54–67.
- Glass, J., Lancôt, K. L., Herrmann, N., Sproule, B. A., & Busto, U. E. (2005). Sedative hypnotics in older people with insomnia: Meta-analysis of risks and benefits. *BMJ*, 331, 1169.
- Kessler, R. C., Berglund, P. A., Coulouvrat, C., Hajak, G., Roth, T., Shahly, V., ... Walsh, J. K. (2011). Insomnia and the performance of US workers: Results from the America Insomnia Survey. *Sleep*, 34, 1161–1171.
- Morganthaler, T., Alessi, C., Friedman, L., Owens, J., Kapur, V., Boehlecke, B., ... Swick, T. J. (2007). Practice parameters for the use of actigraphy in the assessment of sleep and sleep disorders: An update for 2007. *Sleep*, 30, 519–529.
- Morin, C., & Jarrin, D. (2013). Epidemiology of insomnia: Prevalence, course, risk factors, and public health burden. *Sleep Medicine Clinics*, 8, 281–297.
- Morin, C. M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The insomnia severity index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, 34, 601–608.
- Morin, C. M., & Benca, R. (2012). Chronic insomnia. *The Lancet*, 379(9821), 1129–1141.
- Morphy, H., Dunn, K. M., Lewis, M., Boardman, H. F., & Croft, P. R. (2007). Epidemiology of insomnia: A longitudinal study in a UK population. *Sleep*, 30, 274–280.
- NIH. (2005). NIH state-of-the-science conference statement on manifestations and management of chronic insomnia in adults. *NIH Consensus and State-of-the-Science Statements*, 22, 1–30.
- Pearson, N. J., Johnson, L. L., & Nahin, R. L. (2006). Insomnia, trouble sleeping, and complementary and alternative medicine: Analysis of the 2002 National Health Interview Survey Data. *Archives of Internal Medicine*, 166, 1775–1782.
- Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R., & Wyatt, J. K. (1997). Psychophysiological insomnia: The behavioural model and a neurocognitive perspective. *Journal of Sleep Research*, 6, 179–188.
- Pillai, V., Roth, T., & Drake, C. L. (2015). The nature of stable insomnia phenotypes. *Sleep*, 38, 127–138.
- Qaseem, A., Kansagara, D., Forcica, M. A., Cooke, M., & Denberg, T. D. (2016). Management of chronic insomnia disorder in adults: A clinical practice guideline from the American College of Physicians. *Annals of Internal Medicine*, 165, 125–133.
- Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews*, 14, 19–34.
- Roth, T., Jaeger, S., Jin, R., Kalsekar, A., Stang, P. E., & Kessler, R. C. (2006). Sleep problems, comorbid mental disorders, and role functioning in the national comorbidity survey replication. *Biological Psychiatry*, 60(12), 1364–1371.

- Rybarczyk, B., Lund, H. G., Garroway, A. M., & Mack, L. (2013). Cognitive behavioral therapy for insomnia in older adults: background, evidence, and overview of treatment protocol. *Clinical Gerontologist, 36*(1), 70–93.
- Schutte-Rodin, S., Broch, L., Buysse, D., Dorsey, C., & Sateia, M. (2008). Clinical guideline for the evaluation and management of chronic insomnia in adults. *Journal of Clinical Sleep Medicine, 4*, 487–504.
- Smith, M. T., & Haythornthwaite, J. A. (2004). How do sleep disturbance and chronic pain inter-relate? Insights from the longitudinal and cognitive-behavioral clinical trials literature. *Sleep Medicine Reviews, 8*, 119–132.
- Trauer, J. M., Qian, M. Y., Doyle, J. S., Rajaratnam, S. M. W., & Cunnington, D. (2015). Cognitive behavioral therapy for chronic insomnia: A systematic review and meta-analysis. *Annals of Internal Medicine, 163*, 191–204.
- Vgontzas, A. N., Bixler, E. O., Lin, H.-M., Prolo, P., Mastorakos, G., Vela-Bueno, A., ... Chrousos, G. P. (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: Clinical implications. *The Journal of Clinical Endocrinology & Metabolism, 86*, 3787–3794.
- Vitiello, M. V. (2006). Sleep in normal aging. *Sleep Medicine Clinics, 7*, 539–544.
- Wu, J. Q., Appleman, E. R., Salazar, R. D., & Ong, J. C. (2015). Cognitive behavioral therapy for insomnia comorbid with psychiatric and medical conditions: A meta-analysis. *JAMA Internal Medicine, 175*, 1461–1472.
- Zachariae, R., Lyby, M. S., Ritterband, L. M., & O'Toole, M. S. (2016). Efficacy of internet-delivered cognitive-behavioral therapy for insomnia: A systematic review and meta-analysis of randomized controlled trials. *Sleep Medicine Reviews, 30*, 1–10.
- Zhang, B., & Wing, Y.-K. (2006). Sex differences in insomnia: A meta-analysis. *Sleep, 29*, 85–93.

Suggested Reading

- Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., ... Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analysis of longitudinal epidemiological studies. *Journal of Affective Disorders, 135*, 10–19.
- Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive-behavioral therapy approach (therapist guide)*. New York, NY: Oxford University Press.
- Riemann, D., Nissen, C., Palagini, L., Otte, A., Perlis, M. L., & Spiegelhalder, K. (2015). The neurobiology, investigation, and treatment of chronic insomnia. *The Lancet Neurology, 14*, 547–558.

Index

- 6-minute walk (6MW) performance 146
 - compromise 143
- acamprosate (Campral), usage 440
- acceptance and commitment therapy (ACT) 248
- accountable care organization (ACO), coordinated/
 - colocated systems 287
- acetaminophen (paracetamol), usage 134
- acne 451, 452
- actigraphy, usage 462
- active mental model, areas 304
- activities of daily living (ADLs), disruption 98
- acupoints 251
- acupuncture 251–252, 429
 - efficacy 134
 - migraine headache usage 137
 - tobacco use cessation 230
- acute exercise, impact 409–410
- acute illness adjustment, chronic illness adjustment
 - (contrast) 374
- acute stress, counteracting 357–358
- addictions, medical resident stigma-related
 - concerns 59
- adherence. *See* medication adherence; patient adherence
 - defining 375
- adolescence, development/health 332
- adolescents
 - adolescent-focused therapy, family-based
 - treatment (comparison) 123–124
 - alcohol consumption (reduction), self-affirmation
 - implementation intention intervention
 - (development) 49
- alcohol use/abuse, longitudinal pathways
 - (exploration) 312
- anorexia nervosa, family therapy (usage) 123–124
- CAM approaches 246
- medication regimens, nonadherence 376
- sexual orientation/gender minority,
 - identification 419
- stress responses 374
- adulthood, sexual trauma (negative health
 - consequences) 154
- adults, depression screening (USPSTF
 - recommendation) 197–198
- Adverse Childhood Experiences Study 154
- advertising (psychologists) 431–432
- aerobic exercises, participation (impact) 409
- affective forecasting 293
 - bias 295
 - difference variables, impact 295
 - errors 295
 - impact 297
 - focus 295
 - generalized affective forecasts,
 - importance 298–299
 - health-related examples 296
 - impact 297
 - study 294
 - quality, determination 299
 - research 295–296
 - role 296–297
- affective reactions, gender differences 23
- Affordable Care Act. *See* Patient Protection and Affordable Care Act

- Agency for Healthcare Research and Quality (AHRQ), CBT-I efficacy review 463
- aging 301
- agoraphobia, panic (relationship) 455
- alcohol
- abuse diagnosis, establishment 67–68
 - alcohol-related birth defects/neurodevelopmental disorders 438
 - alcohol-related diagnoses, classification (DSM change) 67
 - alcohol-related Korsakoff's syndrome, results 437
 - alcohol-related motor vehicle accidents, NHTSA estimates 70
 - alcohol-related NCD 95
 - blood alcohol concentration (BAC), increase 69
 - hepatitis 438
 - reaction time/decision making impairment 70
- alcohol consumption
- acute alcohol consumption, impact 69–70
 - acute effects 69–70
 - breast cancer risk, increase (relationship) 48
 - chronic alcohol consumption, impact 437
 - definitions 69
 - detrimental outcomes 438
 - excess, impact 437–438
 - moderation, health benefits 438
 - prediction 68–69
- Alcohol Dependence Scale (ADS) 238
- alcoholic liver disease, treatment 441
- Alcoholics Anonymous (AA), usage 440
- alcoholism 67
- alcohol use
- avoidance 40
 - longitudinal pathways, exploration 312
 - long-term negative health outcomes 437–438
 - social cognitive theories 68–69
- alcohol use disorder (AUD) 67
- acamprosate (Campral), usage 440
 - Alcoholics Anonymous (AA), usage 440
 - behavioral couples therapy (BCT) 439–440
 - biopsychosocial framework, usage 68
 - cognitive behavioral (CB) approaches/interventions 439
 - development 68
 - disulfiram, usage 440
 - etiological influences 68–70
 - interventions 439
 - long-term consequences 437
 - maintenance 68
 - medical interventions 440–441
 - motivational interviewing (MI), usage 439
 - naltrexone (ReVia/Vivitrol), usage 440
 - onset 68
 - pharmacotherapy 440
 - prevalence 68
 - psychological factors, integration 68–69
 - relapse processes 68
 - support groups 440
 - treatment approaches 439–441
 - withdrawal symptoms, severity (decrease) 440–441
- allostatic load, manifestation 156
- allostatic mediators, production/dissemination 156
- alopecia 451
- alternative health techniques, ethics issues 427
- American Association of Retired Persons (AARP), family caregiver report 9
- American Board of Professional Psychology (ABPP), certification 347, 360
- American College of Cardiology Foundation, CAC report 84
- American Congress of Obstetricians and Gynecologists (ACOG), Committee on Obstetric Practice (depression screening recommendation) 197
- American Heart Association
- Strategic Impact Goal 90
 - Task Force on Practice Guidelines, CAC report 84
- American Medical Association (AMA), Code of Ethics 289
- American Music Therapy Association, music therapy practitioner certification 253
- American Psychological Association (APA)
- Ethics Code 428, 431
 - Standard 5.05 432
 - “Guidelines for Psychological Practices in Health Care Delivery Systems” 345
 - mission statement, amendment 355
 - Practice Guidelines 124
 - Prevention Guidelines 37
- American Psychosomatic Society, mission 357
- American Sexual Health Association (ASHA), oral contraceptive effectiveness data 387
- Americans with Disabilities Act (ADA) 60
- passage 273
- anabolic-androgenic steroid use, impact 412, 422
- anhedonia 87, 163–164
- schizophrenia, relationship 164
 - self-reported anhedonia, emotion regulation deficits (impact) 164
- anorexia nervosa (AN) 4, 117, 396
- bone mass issues, association 121
 - family therapy 123–124
 - Maudsley model of AN treatment for adults (MANTRA) 123
 - psychotherapy, usage 122–124
 - reproductive consequences, association 121
 - specialist supportive clinical management (SSCM) 123

- treatment, APA Practice Guidelines 124
- weight restoration 123
- antiretroviral (ARV) medications 387
 - adherence, enhancement 387
- anxiety
 - aromatherapy, usage 249
 - disorders
 - medical disorders, comorbidity 237
 - primary care health settings screening 237
 - rates, elevation 122
 - health anxiety 452, 453–454
 - levels, increase 335
 - pediatric cancer, relationship 336
 - physical manifestations, reduction 368
 - physician screening (ACOG recommendations) 197–198
 - prevention/addressing 98
 - reduction 454
 - physical activity, impact 408
 - sensitivity 87
 - skin disease, relationship 451
 - SSRIs/SNRIs, usage 134
 - treatment, biofeedback (usage) 252
- Appearance Schemas Inventory 446
- appetite, change 193
- aromatherapy, usage 247, 249
- arrhythmias, bulimia nervosa (association) 121
- art therapy, usage 246
- Ashtanga yoga 250
- assent, implication 349
- assisted reproductive technology (ART), usage 28
- asthma 327
- athleticism, impact 412
- attentional resources, regulation 14
- attention deficit hyperactivity disorder (ADHD) 236
 - biofeedback, usage 252–253
 - medication, pilot usage 350
- attention, sustaining 60
- attention training 276
- autism spectrum disorders 236
- autonomic nervous system (ANS), activation 75
- autonomy supportive characteristic (MI) 267
- avolition 164–165
- Ayurveda 245, 246
- back pain (treatment)
 - acupuncture, usage 252
 - chiropractic, usage 248
- bacterial vaginosis (BV) 385
- bariatric surgery, usage 177–178
- Beck Anxiety Inventory (BAI) 237, 239
- Beck Depression Inventory (BDI) 208, 239
- Beck Depression Inventory for Primary Care (BDI-PC) 237
- Beck Depression Inventory, Second Edition (BDI-II) 237
- behavior
 - change, transtheoretical model 39–40
 - proximate/ultimate explanations 323
 - psychological mechanisms/processes, knowledge 324–325
- behavioral activation, impact 102
- behavioral adjustment 238
- behavioral care 13
- behavioral couples therapy for AUD (ABCT) 439–440
- behavioral health, behavioral medicine (contrast) 358
- behavioral health providers (BHPs)
 - administrative issues, types 284
 - appointment structure 285–286
 - billing/reimbursement 287–288
 - colleagues, interaction 289
 - confidentiality, management 288
 - documentation 286
 - general clinic supports 285
 - general practice, organization 284–285
 - integration, management
 - considerations 284–286
 - multiple relationships, management 288–290
 - non-colleague patients, interaction 289–290
 - office/clinic space 285
 - primary care integration 283
 - referral management 286–287
- behavioral medicine 358
- behavioral medicine, trends 311
 - biological issues 314–315
 - chronic diseases 312–313
 - chronic psychopathology, health problems/diseases (comorbidity) 316
 - diversity/inclusiveness 313–314
 - healthy psychology knowledge, translation 315
 - methodological sophistication 311–312
 - social justice 316–317
- behavioral regimens, adherence 217
- behavioral theories 39
- behavioral therapy (tobacco use disorder) 228–229
- Beliefs about Appearance Scale 446
- benefit finding (BF) 11
- Benson, Herbert 357
- benzodiazepines, effectiveness 440
- bereavement, impact 388
- Best Practices in Psychology* 36
- Bikram yoga 250
- biliopancreatic diversion, duodenal switch (usage) 178
- binge drinking (SAMHSA definition) 69
- binge eating disorder (BED) 4, 118, 396
 - health complications, association 121

- binge eating, health risk behaviors 155
- biofeedback, usage 137, 252–253
- biologically based practices 247
- biological measures, examples 314–315
- biomedical reductionism 184
- biopsychosocial (BPS) approach 184
 - evolution 321–323
- biopsychosocial (BPS) functioning 322
- biopsychosocial (BPS) model 147, 184–185, 321, 355
 - focus 185
- biopsychosocial (BPS) perspective (suicide) 204–205
- biopsychosocial (BPS) perspectives 323–325
- biopsychosocial (BPS) practice (health psychology) 321
- biopsychosocial (BPS) term, usage 323
- bipobia, behaviors 418
- birth defects 30
- blood alcohol concentration (BAC), increase 69
- body
 - body-based practices 247
 - dissatisfaction, risk factor 400
 - fat/muscularity, dimensions (satisfaction) 446
 - satisfaction/acceptance 409
- Body Checking Questionnaire 446
- body image 1
 - assessment 445
 - methodology 445–446
 - changes 447
 - clinical health psychology, relationship 2
 - concerns
 - correlates/consequences 3–5
 - risk factors 2–3
 - demographic/sociocultural/psychosocial variables, study 2–3
 - disorder/dissatisfaction 396
 - eating-related behaviors 4
 - health behaviors 4
 - manifestation 2
 - measures, content/specificity (range) 446
 - mental health problems 3
 - populations, assessment 446–447
 - psychiatric conditions 4
 - psychological functioning, problems 3
 - sexual health, relationship 4–5
 - sport/exercise, relationship 409
 - weight-related behaviors 4
- Body Image Avoidance Questionnaire 446
- body mass index (BMI)
 - levels 171
 - obesity definition 172
 - premature death/disability, association 172
 - quality of life, reduction 172
- Body Parts Satisfaction Scale 446
- Body Shape Questionnaire 446
- bone mass issues, anorexia nervosa
 - (association) 121
- boundaries, rules/structures 430–431
- bradycardia, bulimia nervosa (association) 121
- brain
 - activity, suppression 135
 - schizophrenia 161
- breast cancer
 - diagnosis, psychological adjustment (trajectories) 77
 - patients, behavioral activation (impact) 102
- bright light therapy, usage 102
- Bronfenbrenner, Urie 327
- bulimia nervosa (BN) 4, 117–118, 396
 - cardiovascular issues, association 121
 - dialectical behavior therapy (DBT), usage 125
 - psychotherapy, usage 124–125
 - treatment, CBT (superiority) 124
- bullying, persistence 421
- bupropion, usage 176
- burnout, prevention 42
- cancer
 - cancer-specific coping processes, psychological adjustment predictors 78
 - childhood cancer 335
 - chronic alcohol misuse, link 438
 - death rates, contributors 73
 - depressed cancer patients, treatment options (evaluation) 102
 - depression, association 101–103, 236
 - development, personality type impact (belief) 74–75
 - diagnosis, psychosocial aspects (improvement) 102
 - early detection, screening 75–76
 - early-stage breast cancer, group therapy (impact) 101–102
 - existence, negative effect 295–296
 - experience, characterization 77
 - impact, studies 102–103
 - initiation/progression, behavioral/psychosocial factors 74–75
 - initiation, risk (reduction) 74
 - long-term survivorship 76
 - mental health, deterioration 77
 - mortality, decline 73
 - outcomes, improvement 74
 - pediatric cancer 335–340
 - psycho-oncology, relationship 73
 - psychosocial adjustment 76–77
 - demographic risk factors 78
 - predictors 77–78
 - recurrence, fear 241

- reentry phase 76–77
- screening
 - barriers, contextual/individual factors 75
 - individual-level factors 76
 - USPSTF recommendations 75
- survivorship periods 240
- trajectory, adjustment 76–77
- Cancer by the Carton* (Reader's Digest) 224
- capacity, implication 349
- cardiac disease, Framingham Risk Equation 85
- cardiac events
 - Framingham Risk Equation 85
 - reduction 90
- cardiology, primary care setting 239
- cardiovascular disease 327
 - mortality (reduction), Strategic Impact Goal (AHA) 90
- cardiovascular issues, bulimia nervosa (association) 121
- caregivers
 - assistance, technological forms 12
 - burden 10
 - caregiver-focused, problem-solving training 13
 - caregiver-oriented interventions 276
 - caregiving class, coping 13
 - clinical health psychology, relationship 9
 - dementia caregivers, burden/depression (alleviation) 13–14
 - duties, break 12
 - enhanced care 13
 - enhanced support group 13
 - environmental skill building program 13
 - female caregivers, financial burden 10
 - information/referral, usage 13
 - interventions 13, 375
 - meditative-based interventions 13–14
 - negative psychosocial sequelae 11
 - positive psychosocial sequelae 11–12
 - post-caregiving 14–15
 - problem-solving skills training (PSST) 375
 - psychosocial interventions 13–14
 - utilization, barriers 14
 - PTG, patient-related factors (relationship) 11
 - respite 12
 - stressors, responses 13
 - support 12
- care-recipient-focused behavior management skill training 13
- cellulitis 451
- Centers for Epidemiologic Studies—Depression (CES-D) scale 88–89, 89
- central nervous system (CNS), demyelinating disease 143
- cervix, narrowing 28
- change
 - fear 277
 - patient language 266
 - resistance, problem 266
 - talk 266, 267
- changes, self-affirmation (impact) 52–53
- Chantix (varenicline tartrate) 228
- childhood
 - cancer 335
 - obesity, public health problem 373
 - sexual behaviors 391
- children
 - adolescence, development/health 332
 - anxiety, at-risk range 336
 - asthma, suffering 373
 - CAM approaches 246
 - chronic illnesses, interventions (development) 373
 - community-based programs, care 377
 - coping strategies (reduction), interventions (impact) 374–375
 - culture, impact 330
 - depression levels, increase 336
 - discrimination 331
 - early childhood, attachment security (importance) 332
 - ethnic minorities, healthcare (relationship) 329–331
 - exosystem 328
 - health 327
 - disparities 331
 - impact 328
 - healthcare access, disparities 330–331
 - hemoglobin A1c levels (reduction), telemedicine (impact) 377
 - language, disparities 331
 - macrosystem 328
 - maltreatment 327
 - medication adherence 337–339, 375–376
 - barriers 375–376
 - mesosystem 327–328
 - microsystem 327–328
 - middle childhood, developmental milestones 332
 - negative health status, impact 328
 - outcomes, perinatal depression development timing (impact) 196
 - pediatric cancer 335–340
 - poverty, long-term impact 329
 - protective/risk factors 331–332
 - sexual attraction (pedophilic disorder) 390
 - sociodemographic disadvantage 337
 - socioeconomic status (SES), impact 329
 - stress reduction, interventions (development) 374–375
 - stress responses 374
- chiropractic therapy, usage 133

- chiropractic, usage 247, 248–249, 429
- chlamydia 385
 - infections, reduction 386
- chronic alcohol consumption, impact 437
- chronic alcohol misuse, link 438
- chronic disease
 - long-term alcohol consumption, impact 437
 - management, preference-sensitive decisions 297
 - physical/psychological symptoms 147
 - psychosocial variables, emphasis 312–313
- chronic health problems/diseases, biological factors 314–315
- chronic illnesses
 - adjustment, acute illness adjustment (contrast) 374
 - treatment, advances 374
- chronic physical health problems, depression management 369–370
- chronic physical illness, impact 365
- chronic psychopathology, health problems/diseases (comorbidity) 316–317
- chronic stress
 - body system, burden 156
 - counteracting 357–358
- chronic stressors 418–419
- chronic tension-type headache (CTTH) 131
 - acupuncture, efficacy 134
 - pain threshold, reduction 132
 - SSRIs/SNRIs, usage 134
- cigarette smoking, impact 87
- clinical competence (psychologists) 428–429
- clinical health psychology
 - ABPP recognition 359
 - APA Division of Health Psychology
 - definition 356
 - application/focus 355–356
 - body image, relationship 2
 - caregivers, relationship 9
 - competencies 359–360
 - education/training 359–361
 - future 361
 - history 355
 - problem areas, psychologist examination 356
 - professional organizations, formation 357–361
 - specialization 360
- Clinical Health Psychology Specialty Taxonomy, CCHPTP development 360
- clinical health psychology, trends 311
 - biological issues 314–315
 - chronic diseases 312–313
 - chronic psychopathology, health problems/diseases (comorbidity) 316
 - diversity/inclusiveness 313–314
 - health psychology knowledge, translation 315
 - methodological sophistication 311–312
 - social justice 316–317
- clinical hypnosis 257–258
- close relationships 19
- cluster headaches 138
- coaches, transformational leadership qualities 411
- Cochrane Reviews 245, 260
- Code of Ethics (AMA) 289
- cognition, social problem solving/work skills (association) 162
- cognitive behavioral therapy (CBT) 112, 198
 - benefits 109
 - efficacy 133
 - interventions 29
 - superiority 124
 - third-wave CBT 125
 - usage 123
- cognitive behavioral therapy for insomnia (CBT-I) 462–463
 - AHRQ efficacy review 463
- cognitive behavior therapy (CBT) 370
 - adherence, improvement 315
- cognitive decline, impact 98
- cognitive deficits 162–163
- cognitive expectancies 68
- Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia (CNTRICS) 163
- cognitive processing, types 324–325
- cognitive remediation (DR) 163
- cognitive therapy (CG) 462
 - benefits 109
 - mindfulness version 109
- Cognitive Therapy for Suicide Prevention (The Joint Commission) 211
- Collaborative Assessment and Management of Suicide (The Joint Commission) 211
- commonsense model of self-regulation (CSM)
 - construct 305
 - framework 303–304
- Community Integration Questionnaire (CIQ) 238–239
- community, promotion 38
- comorbid depression, impact 87–88
- comorbidities
 - depressive comorbidity, studies 102
 - detection/management 97
 - eating disorders 121–122
 - health context 101
 - insomnia 460–461
 - perinatal depression 197
 - psychiatric comorbidity 227
- complementary and alternative medicine (CAM) 199, 245, 427
 - approaches (children/adolescents) 246
 - boundaries, rules/structures 430–431

- domains, NCCIH perspective 246–247
- integration 433–434
- modalities
 - efficacy (NCCIH information) 245–246
 - integration 430–431
 - psychologist knowledge, necessity 429
 - usage, benefits 429
- presence (US) 246
- prevalence 428
- services, offering 432
- complementary and alternative treatments 245
 - tobacco use disorder 229–231
- complementary health techniques, ethics issues 427
- complicated grief, occurrence 31
- condom use practices, increase 386
- confidentiality
 - APA standards 288
 - limits, controls 348
 - psychologist confidentiality 348
- confusion, increase 97
 - dehydration, impact 97
- conscientiousness 37
- consent, implication 349
- Consortium on the Genetics of Schizophrenia (COGS) 163
- constipation, prevention 97
- contraceptive methods 387–388
- co-occurring disease-related stigmas, concern 62
- coping
 - behaviors 410
 - concept, utility (question) 421
 - mechanisms, taxing 418–419
 - skills 204, 238
 - strategies (reduction), interventions (development) 374–375
- coronary artery calcification (CAC)
 - depression, relationship 86, 88–90
 - lower-risk/higher-risk, odds ratios 89
 - persistent/recurrent depression prediction ability 86
 - prevalence/progression, smoking (dose-dependent predictor) 87
 - progression limitation/development prevention 90
 - smoking, relationship 88–90
 - subclinical measure 84
 - Whitehall II study 86
- Coronary Artery Risk Development in Young Adults (CARDIA) study 88
- coronary heart disease (CHD), prevalence 239
- Council of Clinical Health Psychology Training Programs (CCHPTP), Clinical Health Psychology Specialty Taxonomy development 360
- couples, serious/chronic illnesses 20
- Craig Handicap Assessment and Reporting Technique (CHART) 238–239
- cramping (miscarriage symptom) 30
- CREATE randomized controlled trials 313
- cross-dressing, sexual arousal 390
- culture
 - impact 330
 - medical culture, psychologists (interactions) 345–346
- Current Procedural Terminology (CPT) codes, usage 287–288
- cystic fibrosis (CF)
 - psychologist treatment 352
 - services 350
- daily caloric intake, goal setting 174
- daily sleep diaries, usage 461–462
- dance movement therapy (DMT) 246, 247, 251
- death
 - causes
 - biopsychosocial (BPS) perspective 323–325
 - ranking 324
 - risk (reduction), suicide (impact) 208
 - thoughts 193
- decisional bias (mitigation), disability paradox/affective forecasting errors (impact) 297
- decision making
 - affective forecasts, impact (study) 294
 - processes, alcohol-related expectancies (interaction) 58
- delayed ejaculation 388
- delirium
 - benzodiazepines, usage (effectiveness) 440
 - recognition/management 98
- dementia caregivers, burden/depression (alleviation) 13–14
- dementia, recognition/management 98
- depressed smokers, treatment 103
- depression 83
 - cancer, association 101–103
 - chronic
 - health problems, assessment/follow-up 112
 - chronicity, longitudinal effect (modeling attempt) 86
 - comorbid depression, impact 87–88
 - concepts, combination 108
 - coping skills, education 111
 - coronary artery calcification, relationship 86, 88–90
 - definition 107–108
 - depression-smoking comorbidity, recognition/treatment 90
 - evidence-based therapies 112–113
 - health context 101, 107
 - impacts, prediction 84–85

- depression (*cont'd*)
 - interpersonal psychotherapy (IPT), usage 108–109
 - ischemic heart disease (relationship), smoking (role) 87–90
 - levels, increase 335
 - management (older adults) 369–370
 - mechanisms, impact (hypothesis) 85
 - mothers, pregnancy (depression) 194
 - negative effects 84–86
 - nonclinical depression, reduction 408–409
 - patient social network, evaluation/ intervention 112
 - pediatric cancer, relationship 336
 - perinatal depression 193
 - prevalence 107–108
 - prevention/addressing 98
 - psychological manifestations 144
 - psychosocial variables, behavioral activation (usage) 102
 - relapse, relationship 1097
 - risk, medical illnesses (association) 236
 - screening 236–237
 - value, potential 237
 - smoking, comorbidity 87
 - subtypes, differential associations 89–90
 - term, usage 193
 - tobacco smoking/ischemic heart disease, relationship 88
 - tobacco smoking, relationship 83–84
 - treatment
 - acupuncture, usage 252
 - acute phase, IPT interventions 109
 - effects 111
 - SSRIs/SNRIs, usage 134
 - walking
 - bidirectional association 144–145
 - relationship 143
 - Whitehall II study 86
- depression, relapse prevention
 - enhancement 110
 - mindfulness-based therapy, potential 109
 - research, growth 111
 - strategies 111–113
 - patient treatment preferences, consideration 113–114
 - studies 108–109
- depressive comorbidity
 - challenge, consideration 104
 - diversity, relationship 103–104
 - studies 102
- depressive disorders risk (increase), multi-morbidity (impact) 366
- depressive relapse
 - chronic health problems, assessment/ follow-up 112
 - examples 109–110
 - research 110
- depressive symptomatology, clusters (differential associations) 89–90
- depressive symptomatology
 - risk 148
 - walking impairment, cyclical associations 144
- depressive symptoms
 - behavioral approaches 148
 - chronic exposure/accumulation, impact 86
 - clinical elevation 88–89
 - disordered eating symptoms, association 122
 - dysphoria, change (synergistic effect) 103
 - psychological manifestations 144
 - severe clinical depression, clarifications/ distinctions 111
 - walking (bidirectional, cyclical association) 145–146
 - walking impairment, association
 - longitudinal associations, tested model 147
 - possibility 145
 - studies 146–147
 - walking impairment changes, impact 148
- diabetes
 - depression, association 236
 - pharmacotherapy, immigrant status (associations) 314
- diabetes mellitus, development (likelihood) 438
- Diabetes Prevention Program (DPP) lifestyle treatment effects demonstration 175
- Diagnostic Interview with Structures Hamilton (DISH) 239
- dialectical behavior therapy (DBT), usage 125, 248
- Diamond, David/Martha 29
- did not keep (DNK) appointments, allowance 346
- dietary supplements, usage 247
- dieting, risk factor 400
- difference variables, impact 295
- dilation and curettage (D&C) 30
- disability
 - increase (older adults) 367
 - obesity, association 172
 - paradox 293–294
 - errors, impact 297
 - findings/studies 294
 - person-first perspective 275–276
- disabled elderly, care 42
- discrepancy, development (MI principle) 41
- discrimination, promotion 418
- disease
 - causes, biopsychosocial (BPS) perspective 323–325
 - proximate/ultimate explanations 324
 - stigma 57
 - components 57

- distress tolerance 87
- disulfiram, usage 440
- diversity, depressive comorbidity (relationship) 103–104
- donor sperm, usage 28–29
- Drinker's Check-up* 265
- drinking
 - motives 68
 - refusal self-efficacy 68
- Drive for Muscularity Scale 447
- Drug Abuse Screening Test (DAST-20) 238
- Dunbar, Flanders 357
- duodenal switch, usage 178
- dynamic psychological processes, data 165–166
- dysphoria
 - change, synergistic effect 103
 - gender dysphoria 351–352
- dysregulated stress reactions 155–156
- early childhood, attachment security (importance) 332
- early-onset Alzheimer's disease 95
- early-stage breast cancer, group therapy (impact) 101–102
- eating
 - behaviors, risk/disorder (approach) 396
 - eating-related behaviors, body image (relationship) 4
 - health psychology 395
 - problems, student assessment/support (Tier 3) 401–402
- Eating Disorder Examination 445
- eating disorders (EDs) 117, 118
 - acculturation levels/acculturative stress, impact 120–121
 - anxiety disorder rates, elevation 122
 - behavior, risk (reduction) 400
 - biological factors 119
 - comorbidity 121–122
 - complications 121
 - cultural risk factors 119–120
 - defining, method 118
 - diversity considerations 120–121
 - gender differences 120
 - genetic risk factors 119
 - medical management 125
 - monoamine oxidase inhibitors (MAOIs), usage 125
 - mortality rate, elevation 121
 - nutritional therapy 125
 - partial hospitalization 126
 - perfectionistic tendencies 119–120
 - prevention intervention, design/study 401
 - prognosis 126
 - psychological factors 119–120
 - psychotherapy, usage 122–125
 - residential treatment 126
 - risk factors 119–121
 - selective serotonin reuptake inhibitors (SSRIs), usage 125
 - self-monitoring 124
 - sexual orientation, correlation 121
 - social factors 119–120
 - substance use disorders, commonness 122
 - subthreshold levels 118–119
 - symptoms, athleticism/exercise (impact) 412
 - treatment 122–126
 - PHP option 126
 - tricyclic antidepressants (TCAs) 125
 - types 117–119
- e-cigarettes, components 224
- ecological framework, children (negative health status) 328
- ecological model (Bronfenbrenner) 323
- ecological momentary assessment (EMA) 165–166
- eczema 451, 452
- Edinburgh Postnatal Depression Scale (EPDS) 198
- education, prevention model framework 398
- effort
 - construct, complexity 165
 - deficits 164–165
 - effort-based performance deficits, pattern 165
- ego depletion 157
- elderly (depression management), pharmacotherapy/psychotherapy (efficacy) 369
- electroencephalography (EEG), usage 46–47
- electromagnetic therapy 247
- electronic health record (EHR)
 - impact 348
 - systems, usage 284, 287
- electronic nicotine delivery systems (ENDS), marketing 223
- emotion
 - abnormalities 163–164
 - paradox 164
 - regulation 399
 - deficits, impact 164
- emotional intelligence, score (elevation) 295
- emotional management 295
- emotional pain, distance 157
- emotional responses, strategic/intentional withholding 157
- emotional states, duration/intensity (overestimation) 294–295
- emotional stress, prevention 42
- emotional well-being, role 297–298
- empathy, expression (MI principle) 41
- Energy Expenditures for Rewards Task (EEfRT) 165

- energy therapies 247
- engaging (MI process) 268
- enhanced care 13
- Enhancing Recovery in Coronary Heart Disease (ENRICH) randomized controlled trials 313
- environmental skill building program 13
- epidemiological transition 323–324
- epilepsy, alcohol consumption (impact) 437–438
- episodic memory, delay 14
- episodic tension-type headache (ETTH) 131
 - acupuncture, efficacy 134
- erection, obtaining (difficulty) 388
- error-related negativity (ERN) response, examination 46–47
- Ethical Code (APA) 288–289
- “Ethical Principles of Psychologists and Code of Conduct” (APA) 289
- Ethics Code (APA) 428, 431
 - Standard 5.05 432
- ethnic minorities, healthcare (relationship) 329–331
- European Organization for Research and Treatment of Cancer (EORTC), quality of life questionnaire (QLC-C30) 241
- evoking (MI process) 268
- exercise
 - acute exercise, impact 409–410
 - aerobic exercises, participation (impact) 409
 - behavior, self-affirmation/implementation intention combination (impact) 49
 - cause 407–410
 - concurrent factors 411–413
 - effect 410–411
 - health psychology context 407
 - high-intensity exercise, impact 408–409
 - impact 412
 - injury 410
 - positive health outcomes 412
- exhibitionistic disorder 390
- exosystem 328
- Expanded Disability Status Scale (EDSS) 143
- Expectancy that Actions Produce Immediate Outcomes (EAPIO) 304
- falling, fear (older adults) 368–369
- fallopian tubes, blockage 28
- falsifiable hypotheses, experimental tests (findings replication) 322
- families
 - adjustment, pediatric cancer diagnosis 337
 - care partners, health (promotion) 98–99
 - children, health (impact) 328
 - ethnic minorities, healthcare (relationship) 329–331
 - family-based structural multisystem in-home intervention 13
 - health 327
 - social support, exerciser perceptions 411
 - family-based treatment (FBT), adolescent-focused therapy (comparison) 123–124
 - Family Caregiver Alliance, national registry (usage) 12
 - fatigue
 - experience 193
 - management (older adults) 367
 - fats, human craving 324
 - feeding disorders 118
 - female caregivers, financial burden 10
 - fetal alcohol syndrome (FAS) 438
 - fetishistic disorder 390
 - fibrocystic disease (FBD), risk 47
 - fight or flight response 357–358
 - first trimester miscarriages, genetics (impact) 30
 - focalism 295, 296
 - focusing (MI process) 268
 - follicle-stimulating hormone (FSH) levels, alteration 28
 - formal caring, need (older adults) 368
 - frailty, increase (older adults) 367
 - Framingham Risk Equation, risk calculator 85
 - frontotemporal lobar degeneration 95
 - frotteuristic disorder 390
 - frozen embryo transfer (FET) 28
 - frozen/thawed embryo transfer cycle 28
 - Functional Assessment Measure (FAM) 238
 - functional assessment, measurement ability 238
 - functional capacity 187
 - Functional Independence Measure (FIM) 238
 - functional magnetic resonance imaging (fMRI), usage 47
 - functional restoration (FR) programs 187–188
 - fungal skin diseases 451
 - future expectations, inaccuracy (increase) 294
 - future ischemic heart disease, indicators 88
 - gait disturbances 95
 - gamma-aminobutyric acid (GABA)
 - antagonists 464
 - inhibitory effects, enhancement 440
 - gastric banding procedure 177
 - gastric bypass 346
 - gastric sleeve procedure 177–178
 - gender dysphoria, psychologist treatment 351–352
 - gender identity, assessment 385
 - gender minorities
 - individuals, sexual risk behaviors (association) 422
 - persons
 - obesity, concern 422
 - substance use, elevation 421–422
 - term, usage 417

- general clinic supports 285
- generalized affective forecasts,
 - importance 298–299
- generalized anxiety disorder (GAD) 452, 453
- general practice, organization 284–285
- genetic test results, obtaining (intentions) 48
- genital herpes 385
- genital stimulation, sexual interest 390
- glasses, usage (problems) 97
- gonorrhea 385
- grief
 - complicated grief, occurrence 31
 - expressions 30
- Guidelines for Assessment of and Intervention with Persons with Disabilities* (APA) 275, 278
- Guidelines for Prevention in Psychology* 36–37
- “Guidelines for Psychological Practices in Health Care Delivery Systems” (APA) 345
- guilt, feelings 193
- Hamilton Rating Scale for Depression (HAM-D) 237, 239
- Handbook of Psychology and Health* 183
- hangovers, experience 70
- headaches 131
 - acetaminophen (paracetamol), usage 134
 - chiropractic, usage 248
 - cluster headaches 138
 - medication overuse headaches 136
 - migraine headaches 134–137
 - nonsteroidal anti-inflammatory drugs (NSAIDs), usage 134
 - tension headaches 131–134
- health
 - anxiety 452, 453–454
 - biopsychosocial (BPS) model 147
 - complications, binge eating disorder (association) 121
 - disease stigma, impact 57
 - disparities, minority stressors (relationship) 421–423
 - health-compromising behavior, termination 24
 - health-enhancing behavior, initiation 24
 - health-enhancing changes, making 37
 - health-promoting school, WHO definition 396
 - health-related decisions, conscious/unconscious processes (impact) 53
 - health-related examples 296
 - health-related quality of life 336
 - information
 - attitudes (changes), self-affirmation (impact) 50–51
 - resistance, overcoming 45
 - responsiveness (increase), self-affirmation (impact) 53
 - literacy, patient level 218–219
 - maintenance 358
 - messages, self-affirmation/responses 45
 - outcomes, structural stigma (influence) 60
 - prevention, relationship 41–42
 - problem, emergence 327
 - problems/diseases, chronic psychopathology (comorbidity) 316
 - providers, anti-stigma approaches 61–62
 - psychologist, role 90–91
 - relationships, impact 19
 - research 46
 - risk behaviors 155
 - service psychology, societal/healthcare trends (impact) 356–357
 - sexual minority populations, impact 417
 - subjective perceptions, PTEs (association) 154
 - suicide, impact 207
- Health at Every Size (HAES) program 400
- health behavior
 - change, self-affirmation process (understanding) 50–52
 - motivation, emotional well-being (impact) 297–298
 - self-affirmation, impact 46
- health belief model (HBM) 40–41
- healthcare
 - access 329
 - child access, disparities 330–331
 - contexts, challenging 350–351
 - delivery, psychologist competence/credentialing 347
 - ethnic minorities, relationship 329–331
 - factors 328–329
 - visits, occurrence 283
- health contexts
 - comorbidity 101
 - depression 101, 107
 - depressive relapse, examples 109–110
 - relapse 107
- Health Insurance Portability and Accountability Act (HIPAA) 288, 346
- confidentiality control 348
- Health Management Resources weight-loss program 174
- health promotion
 - CAM modalities, usage 246
 - health damage, contrast 51
 - protective factors 37–38
 - tier 1 (three-tier model) 398–400
- health psychology
 - affective forecasting 293
 - biopsychosocial (BPS) approach, evolution 321–323
 - biopsychosocial (BPS) practice 321

- health psychology (*cont'd*)
- clinical health psychology
 - history 355
 - trends 311
 - complementary, alternative, and integrative
 - interventions 245
 - defining 357–361, 358
 - eating 395
 - exercise 407
 - hypnosis
 - applications 259–260
 - relationship 257
 - implications 83, 143
 - knowledge, translation 315
 - neurocognitive disorders, relationship 95
 - pain, relationship 1832
 - relationship 356–357
 - research advance 45
 - schools 395
 - sport 407
 - suicide 203
 - prevention 207–208
 - theory of suicide 204–205
 - three-tier model, history 397–402
 - healthy eating behaviors (facilitation), tier 2
 - (three-tier model) 400–401
 - Healthy People 2020* (publication/mission) 42, 356
 - healthy physical activity 399
 - healthy school, notion 395
 - healthy student 397
 - approach, components 399
 - self-care 397
 - heart, comorbid depression/smoking
 - (impact) 87–88
 - hemorrhagic stroke, chronic drinking (impact) 437
 - herbal supplements, usage 247
 - heterosexism, experiences 418
 - hierarchical attitudes, inclusion 277
 - high blood pressure, chronic drinking (impact) 437
 - high-intensity exercise, impact 408–409
 - home exercise program (HEP) 302–305
 - homeopathy, usage 246
 - homophobia, behaviors 418
 - hopelessness, feelings 208
 - HORIZONS (STI/HIV prevention
 - intervention) 386
 - hospice programs, Medicare/Medicaid funding 42
 - Hospital Anxiety and Depression Scale
 - (HADS) 208, 241
 - human bodies, evolution 324
 - human immunodeficiency virus (HIV) 383
 - antiretroviral (ARV) medications 387
 - care, test and treat 387
 - contraceptive methods 387–388
 - medical male circumcision 387
 - post-exposure prophylaxis (PEP) drugs 387
 - pre-exposure prophylaxis (PrEP) 387
 - prevalence 385–386
 - prevention intervention approaches 386–387
 - prevention methods 384
 - prevention toolkit, expansion 387
 - risk 384
 - increase 386
 - stigma, sensitivity 62
 - test 420
 - testing/linkage/retention 387
 - transmission risk, reduction 387
 - Truvada (emtricitabine/tenofovir disoproxil fumarate), FDA approval 387
 - vaccination 387
 - human papillomavirus (HPV) 385
 - Huntington disease, genetic test results
 - (obtaining) 48
 - Huntington's disease, depression (association) 236
 - hyperhidrosis 451, 452
 - hypersexual disorder 389
 - hypersexuality 389–390
 - clinical guidelines/diagnostic criteria,
 - absence 389
 - treatment, multi-method 390
 - hyperthyroidism, depression (association) 236
 - hypnosis
 - applications 259–260
 - clinical hypnosis 257–258
 - experience, factors 258
 - health psychology, relationship 257
 - meta-analyses 259
 - portability, increase 258
 - positive outcomes, perspective 258
 - qualitative reviews 259
 - sleep-like state, contrast 257
 - usage 246, 253
 - hypnotherapy, usage 229–230
 - hypnotic suggestibility 258–259
 - hypotension, bulimia nervosa (association) 121
 - hypothalamic-anterior pituitary-adrenal (HPA) axis,
 - allostatic load (manifestation) 156
 - hypothalamic-pituitary-adrenal (HPA) axis,
 - trigger 75
 - hypothyroidism, depression (association) 236
 - if-then plans 49
 - illness
 - biopsychosocial (BPS) model 147
 - definition 185
 - prevention 358
 - stigma, influence 59–60
 - immune functioning, massage therapy (impact) 249
 - immune neglect 296
 - impact bias, research 295–296

- implementation intentions
 - self-affirmation implementation intention 49
 - self-affirmation, independent/synergistic effects 49
- impulsivity 204
- individuals (characteristics), self-affirmation effects (moderators) 47–48
- indoor tanning, quitting (intentions) 49–50
- infectious disease, death rate (US) 323
- infertility 27–29
 - causes 28
 - emotional consequences 28
 - infertility-specific mental health services 29
 - types 27–28
- informal caring, need (older adults) 368
- Information-Motivation-Strategy Model (IMS Model) 218
 - information element, impact 219
 - motivation, emphasis 220
 - strategies/resources, development 220
 - usage 219–220
- information processing, reduction 60
- informed consent 348–349, 429–430
- inhibitory emotion regulatory styles 156–157
- insomnia 459
 - actigraphy, usage 462
 - aromatherapy, usage 249
 - assessment 461–462
 - clinical interview 461
 - cognitive behavioral therapy for insomnia (CBT-I) 462–463
 - pharmacotherapy, contrast 464–465
 - cognitive behavior therapy (CBT), adherence (improvement) 315
 - comorbidities 460–461
 - conceptualization, changes 460
 - daily sleep diaries, usage 461–462
 - diagnosis/epidemiology 459–461
 - impact 459–460
 - improvement 187–188
 - melatonin receptor agonists (MRAs) 464
 - orexin receptor antagonists (ORAs) 464
 - over-the-counter (OTC) medications 464
 - pharmacotherapy 463–464
 - polysomnography, usage 462
 - prescription medications, approval/nonapproval 464
 - prevalence 459–460
 - psychological interventions 462–463
 - questionnaires, usage 462
 - risk factors 460–461
 - treatment 462–465
 - tricyclic antidepressants (TCAs) 464
 - types 460
- Insomnia Severity Index (ISI) 462, 463
- Institute of Medicine (IOM)
 - Living and Chronic Illness* report 183
 - “Relieving Pain in America” report 184
- integrated care
 - definition 208–209
 - possibility, EHRs (impact) 348
- integrative health techniques, ethics issues 427
- intentional patient nonadherence 217
- intentions (engendering), self-affirmation (impact) 51
- interdisciplinary care, multidisciplinary care (contrast) 185–186
- interdisciplinary pain management 185–187
 - models, effectiveness 186
- interdisciplinary pain programs
 - availability, improvement 186
 - heterogeneity 187
- interdisciplinary team
 - functioning, practice 276–277
 - science, research 278
- internal experiences, inhibition/avoidance/escape 156–157
- internalized stigma, reduction (efforts) 62
- International Classification of Headache Disorders (ICHD), International Headache Society creation 131
- Internet, obesity effects 173
- interpersonal psychotherapy (IPT) 108, 112
 - combinations, usage 108–109
 - interventions 109
 - usage 123
- interpersonal therapy (IPT) 198
- intervention strategies, self-affirmation (combination) 48–50
- Interview for Diagnosis of Eating Disorders 445
- intimate partners, health problem prevention 20
- intrauterine insemination (IUI) 28
- in vitro fertilization (IVF) 28
- irritable bowel syndrome (IBS)
 - hypnosis, application 260
 - invitation avoidance 60
- ischemic heart disease 83
 - depression
 - negative effects 84–86
 - relationship, smoking (role) 87–90
 - depressive symptomatology 85
 - health psychologist, role 90–91
 - incidence/mortality, depression prediction 84–85
 - indicators 88
 - measurement 84
 - prediction, smoking (impact) 87
 - prevalence, estimate 83
 - primary/secondary prevention, comorbid risk factors (understanding/treatment) 83

- ischemic heart disease (*cont'd*)
 - promotion 85
 - psychological conditions/behavioral risk factors, relationship 83–84
 - risk, increase
 - depression mechanisms, impact (hypothesis) 85
 - depressive symptoms, chronic exposure/accumulation (impact) 86
 - risk (reduction), smoking cessation (impact) 90
 - societal implications 90–91
- ischemic stroke, chronic drinking (impact) 437
- Iyengar yoga 250
- Jenny Craig weight-loss program 174
- job loss, impact 388
- Joint Principles of the Patient-Centered Medical Home (PCMH) 208–209
- Kinect application 306–307
- knowledge acquisition, concentrations 279–280
- Korsakoff's syndrome, chronic drinking (impact) 437
- Kundalini yoga 250
- label avoidance, impact 60
- language
 - children, disparities 331
 - patient language, forms 266
- lesbian, gay, and bisexual (LGB), term (usage) 417
- lesbian, gay, bisexual, or transgender (LGBT)
 - experience 385
- life events, affective forecasts (impact) 298
- lifespan, prevention/health 35
- lifestyle treatment effects, Diabetes Prevention Program (DPP) demonstration 175
- Likert scale 240, 446
- Liraglutide (Saxenda), usage 176–177
- liver diseases 438
 - alcoholic liver disease, treatment 441
- Living Well and Chronic Illness* (IOM report) 183
- long-term aspirations, actions (connections) 52
- Look AHEAD weight loss study 175
- lorcaserin (Belviq), usage 176
- lung disease risks (recognition), smoking (continuation) 297
- luteinizing hormone (LH), impact 28
- Maastricht Questionnaire 239
- macrosystem 328
- major depressive disorder, concept (combination) 108
- manipulative body practices 247
- massage
 - therapy 247, 249–250, 429
 - usage 133
- Matarazzo, Joseph 358
- Maudsley model of AN treatment for adults (MANTRA) 123
- Means-End Problem Solving (MEPS)
 - Procedure 238
- Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) 163
- medical culture, psychologists (interactions) 345–346
- medical disorders, anxiety disorders (comorbidity) 237
- medically assisted reproduction interventions, stress 29
- medically assisted reproduction technology, stress 29
- medically unexplained physical symptoms (MUPS) 207
- medical male circumcision 387
- medical nonadherence, addressing 346
- medical providers, relationships (management) 288–290
- medical psychology, intercultural skills 345–346
- medical regimens, adherence 217
- medical residents, stigma-related concerns 59
- medical settings
 - psychological assessment, practice implications 235
 - psychologists, ethics issues 345
- medication
 - effects, monitoring 97–98
 - medication overuse headaches 136
 - regimens, adolescent nonadherence 376
- medication adherence
 - barriers 338
 - children 337–339, 375–376
 - barriers 375–376
 - older adults 366–367
 - promotion, interventions (impact) 338–339
- Medifast weight-loss program 174
- meditation 400
 - usage 246–248
 - utility, demonstration 357–358
- melatonin receptor agonists (MRAs) 464
- memory
 - encoding 157
 - episodic memory, delay 14
 - semantic memory networks, activation (abnormality) 164
 - spatial working memory, decline 14
- men, cancer mortality 73
- mental health
 - deterioration 77
 - diagnoses, medical resident stigma-related concerns 59

- problems, risk 38–39
- professionals
 - adaptability/skill, importance 346
 - professional integrity 346–347
 - services/interventions, caregiver usage (barriers) 14
- Mental Health Parity Act, mental illness insurance coverage (expansion) 609
- mental illness
 - biogenetic explanations, blame/dangerousness (associations) 61
 - development, absence 154
 - insurance coverage expansion, Affordable Care Act/Mental Health Parity Act (impact) 60
 - suicide risk, association 207–208
- men who have sex with men (MSM), HIV (impact) 385
- meridians 251
- mesosystem 327–328
- metatheoretical frameworks, psychology reliance (reasons) 322
- mHealth programs, impact 173
- microbicides, usage 387
- microsystem 327–328
- middle childhood, developmental milestones 332
- migraine headaches 134–136
 - acupuncture 137
 - aura phase 135
 - biofeedback, usage 137, 252
 - cognitive behavioral treatment (CBT) focus 137
 - electrical activity, spread 135
 - etiology 136
 - headache phase 135
 - mindfulness-based methods 137
 - pathophysiology 135–136
 - pharmacological treatment 136
 - phases 135
 - process 135
 - psychotherapy treatments 137
 - symptomology, reasons (debate) 135
 - transcranial magnetic stimulation (TMS) 136
 - treatments 136–137
 - triggers 135
 - warning phase 135
- military active duty psychologists, impact 350
- Millennium Development Goals (MDGs), creation 209–210
- mind-body connection (suicide) 207
- mind-body medicine 246
- mindful awareness, usage 400
- mindfulness 399
 - mindfulness-based therapy, potential 109
- mindfulness-based cognitive therapy (MBCT) 198, 248
- mindfulness-based stress reduction (MBSR) 248
- minority identity 420–421
 - stress processes, association 420
- minority statuses
 - environmental circumstances, impact 420
 - intersectional interactions 419
- minority stress
 - experiences 421
 - framework, usefulness 419
 - model 418, 421
- minority stressors, health disparities (relationship) 421–423
- minors, gender dysphoria 351–352
- miscarriage 27, 29–32
 - complicated grief, occurrence 31
 - disappointment/despair 30
 - first trimester miscarriages, genetics (impact) 30
 - incidence 30
 - risk (increase), anorexia nervosa (association) 121
 - risk factors 30
- monoamine oxidase inhibitors (MAOIs), usage 125
- mood
 - inconsistent information, processing 157
 - Profile of Mood States (POMS) 408
 - rehabilitation domain 187
- morbidity
 - multi-morbidity 365–366
 - patterns, change (US) 323–324
- mortality patterns, change (US) 323–324
- mothers, pregnancy (depression) 194
- motivational enhancement therapy (MET) 269
- motivational interviewing (MI) 265
 - applications/adaptations 269–270
 - autonomy supportive characteristic 267
 - collaboration, spirit 267
 - evidence base 268–269
 - evocative characteristic 267
 - microskills 267
 - patient language perspective 266
 - principles 41
 - processes 268
 - skills/strategies 267–268
 - style/spirit 266–267
 - training 269
 - usage 439
- motivation, construct (complexity) 165
- motor skills, decrease 95
- multidisciplinary care, interdisciplinary care (contrast) 185–186
- Multi-Ethnic Study of Atherosclerosis (MESA)
 - calcium calculator 84
- multi-morbidity 365–366
 - consequences 366–370
- multiple relationships 430–431

- multiple sclerosis (MS) 207
 - behavioral approaches 148
 - caregiver study 11–12
 - depression/depressive symptoms, psychological manifestations 144
 - depressive symptoms, impact 145
 - functional impairment, physical disease (distinction) 148
 - relapsing-remitting multiple sclerosis (RRMS), study 146–147
 - Theory of Unpleasant Symptoms (TOUS) 145–146
 - walking/depression, health psychology implications 143
 - Multiple Sclerosis Walking Scale-12 (MSWS-12) 143
 - Muscle Appearance Satisfaction Scale 447
 - muscle tightness/strain (reduction), massage therapy (usage) 249
 - musculoskeletal pain (reduction), aerobic/strengthening exercises (impact) 410
 - music therapy, usage 246, 253
 - naltrexone/bupropion (Contrave), usage 176
 - National Alliance for Caregiving (NAC), family caregiver report 9
 - National Association of School Psychologists (NASP), school multitier model 398
 - National Center for Complementary and Integrative Health, Clinical Digest 229
 - National Epidemiological Survey on Alcohol and Related Conditions III data 68
 - National Health and Nutrition Examination Survey (NHANES), obesity data 171–172
 - National Health Interview Survey 246
 - National Institute of Mental Health (NIMH), schizophrenia initiatives 163
 - National Institutes of Health (NIH)
 - “National Pain Strategy” 184
 - Patient-Reported Outcomes Measurement Information System* (PROMIS) 186–187
 - “National Pain Strategy” (NIH) 184
 - National Society of Crippled Children and Adults 273
 - National Weight Control Registry (NWCR), obesity cohort 173–174
 - naturopathy, usage 246
 - neck pain (treatment), chiropractic (usage) 248
 - negative affect
 - negative emotionality 119
 - persistence 194
 - negative attitudes, reduction 61–62
 - neonatal loss (stillbirth) 27, 29–32
 - causes 30
 - complicated grief, occurrence 31
 - disappointment/despair 30
 - neural activity
 - excess disability, prevention 96–98
 - message processing, impact 52–53
 - self-affirmation, impact 46–47
 - neural networks, organization 155–156
 - Neurobehavioral Rating Scale (NRS) 239
 - neurocognitive disorders (NCDs)
 - comorbidities, detection/management 97
 - dementia/delirium, recognition/management 98
 - dependencies 96
 - depression/anxiety, prevention/addressing 98
 - etiology 95
 - family care partners, health (promotion) 98–99
 - health psychology
 - relationship 95
 - role 96
 - medication effects, monitoring 97–98
 - patterns 905
 - prevention/management (importance), American Academy of Neurology Ethics and Humanities Subcommittee emphasis 96–97
 - problems, coping 96
 - progression 99
 - quality of life, basic functioning (optimization) 97
 - substance use, detection/reduction 98
- neurodegeneration, changes 95
 - neurodegenerative disease, progressive effects (acceleration) 98
 - neurons, organization 155
 - neuropsychological evaluation, usage 274
 - nicotine
 - dependence 227
 - electronic nicotine delivery systems (ENDS), marketing 223
 - gum, development/usage 227–228
 - term, usage 223
 - use disorders 225
 - nicotine replacement therapy (NRT), modalities 228
 - Nicot, Jean 224
 - nociception, pain (contrast) 185
 - nonclinical depression, reduction 408–409
 - noncommunicable diseases, mortality reduction (United Nations Sustainable Development Goals) 90–91
 - non-orthopedic pain conditions 187
 - nonsteroidal anti-inflammatory drugs (NSAIDs), usage 134
 - no-show appointments, allowance 346
 - obesity 171
 - adjustable gastric band, usage 177
 - bariatric surgery 177–178

- behavioral lifestyle interventions 174–175
- biliopancreatic diversion, duodenal switch (usage) 178
- BMI definition 172
- clinical guidelines 178
- commercial weight-loss programs 174
- consequences 172
- daily caloric intake, goal setting 174
- environmental contributors 172–173
- etiology 171–173
- extended care maintenance programs, helpfulness 174
- genetic contributors 172–173
- hypnosis, application 260
- influences 172–173
- Internet, effects 173
- lifestyle treatment effects, Diabetes Prevention Program (DPP) demonstration 175
- liraglutide (Saxenda), usage 176–177
- lorcaserin (Belviq), usage 176
- medical costs 172
- mHealth interventions 173
- naltrexone/bupropion (Contrave), usage 176
- National Health and Nutrition Examination Survey (NHANES) data 171–172
- National Weight Control Registry (NWCR) cohort 173–174
- obesity-related comorbidity 175
- orlistat (Xenical), usage 175–176
- pharmacotherapy 175–177
- phentermine/topiramate (Qsymia), usage 176
- prevalence, increase 171
- rates, increase 327
- risk (increase), pediatric cancer (impact) 339–340
- roux-en-Y gastric bypass 177
- scope/seriousness 171–173
- self-help approaches 173–174
- treatment options 173–178
- vertical sleeve gastrectomy, usage 177–178
- weight loss, Look AHEAD study 175
- obesogenic environment 173
- obsessive-compulsive disorder (OCD) 452, 454–455
- offset responsibility 60–61
- offspring, postpartum depression 194–195
- older adults
 - behavioral medicine researchers/clinicians, perspectives 365
 - chronic physical health problems, depression management 369–370
 - cognitive behavior therapy, usage 370
 - disability, increase 367
 - falling, fear 368–369
 - fatigue, management 367
 - formal/informal caring, need 368
 - frailty, increase 367
 - health psychology researchers/clinicians, perspectives 365
 - medication adherence 366–367
 - pain, management 367
 - physical activity, reduction 369
 - problem-solving therapy, usage 370
 - shoulder impairment, epidemiology 301–302
- olfactory/taste perception, alteration 97
- Olmstead v. L.C. 60
- oncology
 - primary care setting 240–241
 - survivorship, periods 240
- onset responsibility 60–61
- OPTIFAST weight-loss program 174
- oral contraceptives, effectiveness (ASHA data) 387
- orexin receptor antagonists (ORAs) 464
- organ transplantation 346
 - assessment 241
 - primary care setting 240
 - psychologists, services 350–351
- orlistat (Xenical), usage 175–176
- osteopathic 247
- osteopenia, anorexia nervosa (association) 121
- osteoporosis 369
 - anorexia nervosa, association 121
- oxytocin augmentation therapy (OAT), meta-analysis 166
- pain
 - aromatherapy, usage 249
 - biopsychosocial (BPS) model 184–185
 - coping 187
 - functional restoration (FR) programs 187–188
 - health psychology, relationship 183
 - interdisciplinary pain management 185–187
 - effectiveness 186
 - measurement 303
 - nociception, contrast 185
 - relief, hypnosis (applications) 259
 - VA Polytrauma Network Site, veteran assessment 188
- pain management
 - care, multicomponent models 185
 - older adults 367
 - services, integration (importance) 186
- panic, agoraphobia (relationship) 455
- paracetamol (acetaminophen), usage 134
- paraphilic disorders 390–391
 - assessment, clinician investigation 390
- parasympathetic nervous system activity, decrease 85
- Parkinson's disease
 - depression, association 236
 - patients, caregiver study 10

- partial hospitalization programs (PHPs), ED treatment option 126
- partners
 - influence, characteristics 22
 - negative tactics, usage 24
- pathological worry, impact 453
- patient adherence (compliance) 217
 - factors 218–219
 - improvement, IMS Model (usage) 219–220
 - Information-Motivation-Strategy Model (IMS Model) 218
- Patient-Centered Outcomes Research Institute (PCORI), clinical trials requirement 298
- Patient Health Questionnaire 9 (PHQ-9) 198
- patient nonadherence
 - economic burden 217–218
 - types 217
- Patient Protection and Affordable Care Act (US ACA) 36, 38, 359, 361
 - insurance coverage expansion 60
 - prevention, relationship 38–39
- Patient-Reported Outcomes Measurement Information System* (PROMIS) (NIH) 186–187
- patients
 - informed consent 429–430
 - patient language, forms 266
 - self-prescribing 428–429
- pediatric cancer
 - depression/anxiety, relationship 336
 - diagnosis, family adjustment 337
 - health-related quality of life 336
 - impact 339–340
 - medication adherence 337–339
 - negative psychosocial outcomes 336–337
 - psychosocial functioning 335–337
 - resiliency 336–337
 - survivorship, health challenges 339–340
 - treatment, neurological functioning 339
- pediatric illnesses (treatment), telemedicine (usage) 377–378
- pediatric psychologists, roles 376–377
- pediatric psychology
 - cultural/ethical disparities 378
 - culturally competent care 378
 - definition 373
 - diagnosis, adjustment 374–375
 - interventions, impact 374–375
 - treatment delivery 376–377
- pedophilic disorder 390
- peers, transformational leadership qualities 411
- pelvic inflammatory disease (PID) 385–386
- perfectionistic tendencies 119–120
- performance-enhancing substances, impact 412
- pericranial muscles, tenderness (impact) 132
- perinatal depression 193
 - biological factors 195–196
 - clinical history/comorbidity 197
 - cognitive behavioral therapy (CBT) 198
 - complementary and alternative medicine (CAM) 199
 - conceptualization/classification 193–194
 - demographic factors 195
 - detection 197–199
 - development timing, impact 196
 - engagement 197–198
 - health factors 195–196
 - interpersonal therapy (IPT) 198
 - management 197–199
 - mindfulness-based cognitive therapy (MBCT) 198
 - prevention 198–199
 - prophylactic treatment 198
 - protective factors 195–197
 - psychological factors 196–197
 - psychotherapy, usage 198–199
 - risk 195–197
 - health-related behaviors, impact 196
 - screening 197–198
 - social support, importance 196–197
 - socioeconomic status (SES), factor 195
 - SSRI, usage 198
 - treatment 198–199
- perinatal loss
 - aftermath 31–32
 - factors 30
 - grief reaction 30
 - interventions, consensus (absence) 31–32
 - mental health disorders 31
 - psychotherapy/counseling 31
- permission, implication 349
- personality
 - characteristics, assessment 37
 - complexity 409
- personal wellness, improvement 36
- person-centered theory (PCT) 39
- phentermine/topiramate (Qsymia), usage 176
- phobias (treatment), chiropractic (usage) 248
- physical activity
 - anxiety-reducing effects 408
 - cause 407–410
 - concurrent factors 411–413
 - effect 410–411
 - effectiveness 408
 - feelings 408
 - healthy physical activity 399
 - participation, barriers 413
 - psychobiological factors, relationship 409–410
 - reduction, older adults 369
- physical health

- potentially traumatic events (PTEs), exposure
 - association 154–155
 - impact 153
 - problems, risk 38–39
- physical therapy (PT) 276, 302–305
- physicians
 - board certification 347
 - patient trust level, importance 219–220
- Pittsburgh Sleep Quality Index (PSQI) 462, 463
- placental abruption 30
- planned behavior (PB) 40
- planning (MI process) 268
- pleasure, experience capacity (reduction) 163–164
- polycystic ovary syndrome (PCOS) 28
- polysomnography, usage 462
- positive/negative social control, contrast 21
- positive psychology
 - research 278
 - usage 38
- post-caregiving 14–15
- post-exposure prophylaxis (PEP) drugs 387
- postpartum depression
 - correlates/consequences 194–195
 - deficiencies, association 196
- postpartum depressive symptoms, unhealthful
 - behaviors (association) 196
- posttraumatic growth (PTG) 11
 - caregiver, patient-related factors (relationship) 11
 - experiences 421
 - research 278
- posttraumatic stress disorder (PTSD) 188, 452, 454
 - lifetime prevalence 154
 - PTE exposure, poor health (association) 155
- posttraumatic stress, levels (increase) 335
- potentially traumatic events (PTEs)
 - biology, negative alterations (association) 153
 - emotional pain, distance 157
 - exposure
 - health problems, association 155
 - impact 153, 156
 - physical health, association 154–155
 - health, subjective perceptions (association) 154
 - inhibitory emotion regulatory styles 156–157
 - posttraumatic stress disorder (PTSD),
 - relationship 153–154
 - psychopathological syndromes, development 154
 - responses 154
- poverty, long-term impact 329
- Practice Guidelines (APA) 124
- prayer, usage 246, 247, 251
- preeclampsia 30
- pre-exposure prophylaxis (HIV) 387
- pre-exposure prophylaxis (PrEP) 387
- preference-sensitive decisions
 - affective forecasting, impact 2907
 - example 296
 - influences 296–297
- preference-sensitive prevention/treatment
 - decisions, affective forecasting (role) 296–297
- pregnancy
 - achievement, medical procedures 28–29
 - alcohol consumption, detrimental outcomes 438
 - depression, correlates/consequences 194–195
 - prevention methods, usage 384
- premature death, obesity (association) 172
- premature ejaculation 388
- premature ischemic heart disease morbidity/
 - mortality risk (increase), cigarette smoking (impact) 87
- Preparing People for Change* 265
- Preparing People to Change Addictive Behaviors* 265
- preterm delivery, risk (increase) 194–195
- prevention
 - activities 36–37
 - Affordable Care Act, relationship 38–39
 - definitional terms (Gordon), US Institute of Medicine's Committee on Prevention of Mental Disorders adoption 36
 - definitions 35–36
 - guidelines 36–37
 - health belief model (HBM) 40–41
 - health, relationship 41–42
 - intervention, conceptualization 36
 - motivational interviewing (MI) 41
 - psychological-based theoretical
 - interventions 39–41
 - research/practices 37
 - science, evidence 36
 - specialists, impact 37
 - theory of reasoned action and planned behavior (TTA/PB) 40
 - three-tier model, history 397–402
 - training 37
 - transtheoretical model of behavior change (TTM)
 - 39–40
 - types 35–36
- primary care health settings, anxiety disorders
 - (screening) 237
- primary care medicine, suicide (context) 205–209
- primary care physician (PCP)
 - healthcare visits, occurrence 283
 - referral management 286–287
 - referral placement, promotion 287
- primary care psychology, administrative issues 283
- primary care settings 236–238
 - behavioral health providers,
 - involvement 283–284
 - drug/alcohol abuse, concerns 237–238

- primary care settings (*cont'd*)
 - rehabilitation settings 238–239
 - specialty settings 239–241
- primary infertility, occurrence 27–28
- primary insomnia 460
- primary medical care (PC)
 - BHP work 284
 - definition 283
- primary prevention 35
- primary psychiatric disorders 451
- problem behaviors, reduction 37
- problems, aversion 36
- problem-solving skills training (PSST) 375
- problem-solving therapy 370
- Profile of Mood States (POMS) 408
- progressive muscle relaxation (PMR) 247, 250–251
- Project MATCH 269
- prostate-specific antigen (PSA) testing, prostate
 - cancer screening (USPSTF recommendations) 75
- protective factors
 - considerations 38
 - types 37
- psoriasis 451
- psychiatric comorbidity 227, 388
- psychiatric disorders 187
- psychobiological factors, physical activity
 - (relationship) 409–410
- psychodermatological (psychocutaneous) disorders,
 - classification 451
- psychodermatology, impact 451
- psychological conditions, cataloguing 451–452
- psychological disorders
 - development, risk (increase) 366
 - protective/risk factors, association 331
- psychological immune system 294
- psychological practice, CAM integration 433–434
- psychological services, assessment/provision
 - (importance) 275
- psychological stress, coping 238
- psychological treatment, complementary/
 - alternative/integrative health techniques (ethics issues) 427
- psychologists
 - advertising 431–432
 - boundaries 349–350
 - boundaries, rules/structures 430–431
 - CAM modality knowledge, necessity 429
 - clinical competence 428–429
 - competence/credentialing (healthcare delivery) 347
 - confidentiality 348
 - ethics issues (medical settings) 345
 - gender dysphoria treatment 351–352
 - growth 358–359
 - healthcare contexts, challenging 350–351
 - informed consent 348–349
 - loyalties, conflicts 351
 - military active duty psychologists, impact 350
 - multiple relationships 430–431
 - primary care integration 283
 - professional colleagues, consultation/
 - cooperation 431
 - professional integrity 346–347
 - public statements 431–432
 - services (organ transplantation) 350–351
 - transgender youth treatment 351–352
- psychology
 - health, relationship 356–357
 - medical psychology, intercultural skills 345–346
 - pediatric psychology 373
 - positive psychology, usage 38
 - prevention, guidelines 36–37
 - professional organizations, formation 357–361
 - role 204
- psychomotor agitation/retardation 193
- psychoneuroimmunology (PNI) 357
- psycho-oncology, cancer (relationship) 73
- psychophysiologic disorders 451
- psychosexual functioning, evaluation 274
- Psychosocial Assessment of Candidates for
 - Transplantation (PACT) 240
- psychosocial stressors (reactivity reduction),
 - exercise/physical activity (impact) 410
- psychosocial triggers, impact 75
- psychotherapy
 - migraine headache treatments 137
 - usage 198–199
- public health, three-tier model (history) 397–402
- public statements (psychologists) 431–432
- qigong 247
- quality of life
 - basic functioning, optimization 97
 - health-related quality of life 336
 - reduction
 - obesity, association 172
 - walking impairment, association 145
- quality of life questionnaire (QLC-C30) 241
- Quality of Life Scale 239, 241
- randomized controlled trials (RCTs) 230, 257,
 - 313, 369
- range of motion (ROM)
 - change 305
 - dynamic record 306
 - feasibility testing, proof-of-concept trial 306
 - measurement 303
 - normal ROM 303
 - recovery, optimization 302

- shoulder ROM, recovery (patient expectations) 306
- rapid eye movement (REM) sleep
 - acute exercise, impact 409–410
 - onset delay, alcohol (impact) 69–70
- Raynaud's disease, biofeedback (usage) 252
- Readiness for Transition Questionnaire (RTQ) 240
- readiness rulers 267–268
- real-world situations, limitations 315
- recreational sport, team size (negative impact) 411
- rehabilitation
 - black box perspective 277
 - interventions, theory-driven framework (development) 276
 - psychologists
 - advocate roles 278–279
 - roles 273–277
 - settings 238–239
- rehabilitation psychology 273
 - interdisciplinary team functioning, practice 276–277
 - intervention, practice 275–276
 - petition 279–280
 - practice, assessment 274–275
 - research 277–279
 - training 279–280
- reiki 247, 252
- relapsing-remitting multiple sclerosis (RRMS), study 146–147
- relationships
 - change, cognitions/readiness 22
 - close relationships 19
 - gender, impact 23
 - impact 19
 - multiple relationships 430–431
 - satisfaction 23
 - social relationships (breakdown), stigma (impact) 60
- relaxation
 - systematic relaxation 400
 - training, usage 133, 462–463
 - utility, demonstration 357–358
- “Relieving Pain in America,” IOM report 184
- religion, usage 247, 251
- reproductive consequences, anorexia nervosa (association) 121
- reproductive health 383
- reproductive trauma 31
- resilience (concept), utility (question) 421
- resistance, handling (MI principle) 41
- Resources for Enhancing Alzheimer's Caregiver Health (REACH) initiative/study 13
- risk
 - aversion 277
 - factor, definition 331
 - reduction 38
 - health-enhancing changes, making 37
- ritualistic behaviors 454
- Rogers, Carl 265
- rosacea 451, 452
- rotator cuff injury 303
- roux-en-Y gastric bypass, usage 177
- schizophrenia 161
 - anhedonia, understanding 163–164
 - assessment, implications 165–166
 - avolition, impact 164–165
 - cognitive deficits 162–163
 - cognitive impairments, severity 162
 - dynamic psychological processes, data 165–166
 - ecological momentary assessment (EMA) 165–166
 - effort-based performance deficits, pattern 165
 - effort deficits 164–165
 - emotion abnormalities 163–164
 - motivation 164–165
 - oxytocin
 - augmentation
 - therapy (OAT), meta-analysis 166
 - prodromal (prepsychotic) features 164–165
 - psychiatric/functional impairment 166
 - treatment, acupuncture (usage) 252
 - treatment as usual (TAU) 167
 - treatment, implications 166–167
- Schofield, William 358
- schools
 - eating disorder prevention intervention, design/study 401
 - education, prevention model framework 398
 - Health at Every Size (HAES) program 400
 - health-promoting school, WHO definition 396
 - health promotion
 - issues 396
 - system/community/school-level factors 396
 - health psychology 395
 - healthy school, notion 395
 - lunches, provision 399
 - setting, impact 395–396
 - social emotional learning (SEL) programs 399
 - well-being/health, promotion 399
- Screening, Brief Intervention, and Referral to Treatment (SBIRT) models 268, 270
- secondary infertility, occurrence 27–28
- secondary insomnia 460
- secondary prevention 35–36
- secondary psychiatric disorders 451
- seizure
 - disorders, depression (association) 236
 - management, benzodiazepines (effectiveness) 440

- selective serotonin reuptake inhibitors (SSRIs),
usage 125, 134, 198
- self-affirmation 45
 - effectiveness, considerations 47–48
 - effects, moderators 47–48
 - experimental induction 48
 - implementation intention
 - combination 50
 - independent/synergistic effects 49
 - intervention, development 49
 - influence 53
 - studies 46–47
 - intervention strategies, combination 48–50
 - message
 - content 47–48
 - framing, impact 49
 - process, understanding 50–52
 - research 46
 - single potential mediating variables, role 51
 - studies, research methods (diversity/application)
46–47
 - techniques, clinical samples 50
 - trajectory 52–54
- Self as Anchor, factor 304
- self-care 399
 - tier 2 (three-tier model) 400–401
- self-efficacy, support (MI principle) 41
- self-esteem
 - physical activity, impact 409
 - premorbid sources 275
- self-focus, increase 194
- self-management
 - resource access (restriction), label avoidance
(impact) 60
- self-management, stigma (influence) 59–60
- self-monitoring, improvement 14
 - meditational model 21
- self-protective responses, emergence 98
- self-prototypes 305–306
 - deviations 304
- self-related processing, increase 52–53
- self-report-based measures, usage 165–166
- self-reported anhedonia, emotion regulation deficits
(impact) 164
- semantic memory networks, activation
(abnormality) 164
- sensory loss, correction (barrier removal) 97
- serotonin-norepinephrine reuptake inhibitors
(SNRIs), usage 134
- service utilization 238
- severe clinical depression, depressive symptoms
(clarifications/distinctions) 111
- severe stress, experience 155
- sexual arousal, alcohol intake (impact) 69–70
- sexual behaviors 384
 - engagement 391
- sexual desire, absence 389
- sexual dysfunction 388–389
 - assessment, factors 388–389
 - gender specificity 388
- sexual functioning, restoration 389
- sexual/gender minority health, focus 110
- sexuality
 - attitudes 388
 - hypersexuality 389–390
- sexually transmitted diseases (STDs), medical
resident stigma-related concerns 59
- sexually transmitted infections (STIs) 383
 - contraceptive methods 387–388
 - infection methods 384
 - prevalence 385–386
 - prevention intervention approaches 386–387
 - risk, increase 386
- sexual masochism disorder 390
- sexual minority
 - health disparities, context 418–420
 - populations, health (relationship) 417
- sexual orientation 384–385
 - disordered eating, correlation 121
 - minority persons, research 419
 - minority, term (usage) 417
 - persons
 - obesity, concern 422
 - substance use, elevation 421–422
 - sexual risk behaviors, association 422
- sexual pleasure, experience (restoration) 389
- sexual risk behaviors 385–386
- sexual sadism disorder 390
- sexual trauma, negative health consequences 154
- shoulder impairment
 - baseline 306
 - epidemiology (older adults) 301–302
 - feasibility testing, proof-of-concept trial 306
 - home exercise program (HEP), usage
302–303
 - Kinect application 306–307
 - management
 - approach 302
 - efficacy 302–303
 - examination, CSM framework 303–304
 - outcomes
 - physical therapy, impact 306
 - self-prototypes/expectations/
timelines 305–306
 - timelines/expectations, technology
(usage) 306–307
 - physical therapy (PT), usage 302–303
 - range of motion, dynamic record 306

- self-prototypes 304–305
 - semi-structured interviews 305
- stick avatar, usage 306
- shoulder ROM, recovery (patient expectations) 306
- single-occasion interview-based measures,
 - usage 165–166
- single potential mediating variables, role 51
- Situational Inventory of Body Image Dysphoria 446
- skin cancer 451
 - risk, reduction 51
- skin disease, anxiety (relationship) 451
- skin-mind connection, psychodermatology
 - (impact) 451
- skin symptoms, responses/management 454–455
- sleep
 - daily sleep diaries, usage 461–462
 - disorder (insomnia) 459
 - disruption 193
 - alcohol consumption, impact 69–70
 - hygiene 463
 - problems 327
 - maintenance difficulties 459
 - restriction 462–463
 - sleep-wake cycle, maintenance 97
 - sleep-wake rhythms, changes 95
 - total sleep time (TST) 461, 463
 - improvement 464
 - wake time after sleep onset (WASO) 461
- sleep efficiency (SE) 461–462, 463
- sleep latency (SL) 461
- smoke break, avoidance 228
- smoking
 - cigarette smoking, impact 87
 - coronary artery calcification, relationship 88–90
 - depression, comorbidity 87
 - depression-smoking comorbidity, recognition/
 - treatment 90
 - dose-dependent predictor 87
 - emotion, association (traits) 87
 - health risk behavior 155
 - impact 87–88
 - smoking-related illnesses, mortality risk
 - (increase) 226–227
 - status, breath samples (carbon monoxide
 - analysis) 314–315
 - tobacco smoking 83–84
- smoking cessation
 - assessment, clinical areas 227
 - hypnosis, application 260
 - impact 90
 - programs, depressed smokers (treatment) 103
- social anxiety disorder (SAD) (social phobia)
 - 452–453, 455
- social cognition, definition 162
- social control 20–21
 - attempts
 - history 24
 - neuroticism 24
 - contextual model 22–24
 - delineation, attempts 21
 - dual-effects hypothesis 20–21
 - ethnic differences 23
 - gender, impact 23
 - positive/negative social control, contrast 21
 - usage, predictors/consequences 22
- social emotional learning (SEL) programs 399
- social justice 316–317
- Social Problem Solving Inventory-Revised
 - (SPSI-R) 238
- social relationships (breakdown), stigma
 - (impact) 60
- social support 20
 - contextual model 22–24
 - ethnic differences 23
 - exerciser perceptions 411
 - social control, relationship 22
- Society for Prevention Research 36
- Society of Counseling Psychology (APA) 36
- socioeconomic status (SES)
 - impact 329
 - perinatal depression factor 195
- Solomon, George F. 357
- spatial working memory, decline 14
- specialist supportive clinical management
 - (SSCM) 123
- specialty settings 239–241
- spinal manipulation 247
- spirituality, usage 246, 247, 251
- sport
 - activities, direct parental involvement 411
 - cause 407–410
 - concurrent factors 411–413
 - effect 410–411
 - health psychology context 407
 - injury rehabilitation, adherence 410
 - participation, impact 409
 - recreational sport, team size (negative
 - impact) 411
 - transformational leadership qualities 411
- Stanford Integrated Psychosocial Assessment for
 - Transplantation (SIPAT) 240
- stigma
 - combatting 61–62
 - components 57
 - co-occurring disease-related stigmas, concern 62
 - definitions/examples 58
 - harm 60
 - impact 59–60

- stigma (*cont'd*)
 - internalized stigma, reduction (efforts) 62
 - moderators 60–61
 - reduction 61
 - structural stigma, influence 60
 - types 58, 58–59
- stillbirth 27
- stimulus control 462–463
- stress
 - acculturative stress, impact 120–121
 - adjustment (improvement), interventions (usage) 374–375
 - aromatherapy, usage 249
 - chronic stress, body system (burden) 156
 - coping, assistance 20
 - dysregulated stress reactions 155–156
 - emotional stress, prevention 42
 - mindfulness-based stress reduction (MBSR) 248
 - processes, minority identity (association) 420
 - reactions, mitigation 20
 - severe stress, experience 155
- stressors
 - chronic stressors 418–419
 - coping 20
 - minority stressors, health disparities (relationship) 421–423
 - psychosocial stressors (reactivity reduction), exercise/physical activity (impact) 410
 - traumatic classification 153–154
- structural stigma, influence 60
- Structured Interview for Anorexia and Bulimic Disorders 445
- students. *See* healthy student
 - eating problems, assessment/support (Tier 3) 401–402
 - healthy student 397
 - SCOFF screening instrument 401
- Study of Women's Health Across the Nation Heart Study 86
- Substance Abuse and Mental Health Services Administration (SAMHSA), binge drinking definition 69
- substance use 204
 - detection/reduction 98
- disorders
 - classification, DSM change 67
 - eating disorders, relationship 122
 - elevation (sexual orientation/gender minority persons) 421–422
 - genetic risk factors 312, 314
 - health risk behavior 155
- subthreshold depressive symptoms, concepts (combinations) 108
- subthreshold ischemic heart disease, indicators 88
- suicidal ideation
 - levels, elevation 419
 - risk, elevation 335–336
- suicide
 - biopsychosocial (BPS) perspective 204–205
 - cognitions, reduction 208
 - cognitive behavioral therapy (CBT) 208
 - contagion 207
 - dialectical behavior therapy (DBT) 208
 - evidence-based psychological treatment approaches 208
 - health psychology context 203
 - theory 204–205
 - impact 207
 - integrated care 208–209
 - integrative care 212
 - The Joint Commission, recommendations 211
 - lifespan, perspective 205
 - mind-body connection 207
 - prevention
 - changes 209–210
 - health psychology, impact 207–208
 - primary care medicine context 205–209
 - presentation 206
 - training 206
- psychologists, role 209
- risk
 - identification 208
 - mental illnesses, association 207–208
- suicidal behavior, occurrence 212
- thoughts 193
- World Health Organization (WHO)
 - global goals 209–210
 - recommendations 210–211
- survivorship
 - pediatric cancer 339–340
 - periods 240
- sustainable development goals (SDGs), UN presentation 209–210
- sustain talk 266, 267
- sweets, human craving 324
- sympathetic-adrenal-medullary (SAM) system,
 - allostatic load (manifestation) 156
- sympathetic nervous system activity, increase 85
- syphilis 386
- System 1 (cognitive processing type) 324–325
- System 2 (cognitive processing type) 324–325
- systematic relaxation 400
- task, persistence 60
- telemedicine, usage 377–378
- telephone-linked computer intervention 13
- temporomandibular joint dysfunction (TMJD), TTH occurrence (likelihood) 133

- temporomandibular (jaw) joint, impact 133
- tension headaches 131–134
 - epidemiology 131–132
 - etiology 132–133
 - treatments 133–134
 - biofeedback, usage 252
- tension-type headache (TTH) 131
 - biofeedback training, treatment 133
 - cognitive behavioral therapy, efficacy 133
 - diagnostic criteria 132
 - etiology 132–133
 - manual therapy, usage 133
 - pericranial muscles, tenderness (impact) 132–133
 - relaxation training, usage 133
 - temporomandibular (jaw) joint, impact 133
 - treatment 133–134
- tertiary prevention 35–36
- The Joint Commission, suicide recommendations 211
- theory of reasoned action and planned behavior (TTA/PB) 40
- Theory of Unpleasant Symptoms (TOUS) 145–146
- therapeutic touch 247
- thinness, pursuit 396
- threat
 - bodily/physiological response 46
 - processing, openness 47
- threatening information, aversion (reduction) 51
- three-tier model, history 397–402
- Timed 25-Foot Walk (T25FW) 143
- time in bed (TiB) 461–462
 - restriction 463
- tobacco
 - abuse, term (usage) 225
 - companies, youth targeting 224–225
 - dependence, treatment 269
 - term, usage 223
- tobacco smoking 83
 - depression, relationship 83–84
 - role 87–90
- tobacco use
 - absence, health improvements 226–227
 - cessation
 - benefits 226–227
 - symptoms 226
 - cleaner smoke, design 224
 - e-cigarettes, components 224
 - history 224–225
 - initiation/maintenance 226
 - reduction, symptoms 226
 - studies 224–225
- tobacco use disorder
 - acupuncture 230
 - assessment 227
 - behavioral therapy 228–229
 - relapse prevention 229
 - classification 225–226
 - complementary and alternative treatments 229–231
 - confectionary chewing gum 230–231
 - DSM-5 definition 225
 - evidence-based treatment 227–229
 - hypnotherapy 229–230
 - randomized controlled trial (RCT) 230
 - pharmacotherapy 227–228
 - symptoms 225
 - tolerance/withdrawal, constructs (association) 225–226
 - treatment 223
- topiramate, usage 176
- total sleep time (TST) 461, 463
 - improvement 464
- Traditional Chinese medicine (TCM) 245, 246, 251
- transcranial magnetic stimulation (TMS), effectiveness 136
- transformational leadership qualities 411
- transgender youth, gender dysphoria (psychologist treatment) 351–352
- transtheoretical model (TTM) 39–40
- transvestic disorder 390
- trauma
 - definition 31
 - reproductive trauma 31
 - sexual trauma, negative health consequences 154
 - trauma-related emotions/thoughts/memories, coping 156
- traumatic events
 - cognitive process 11
 - potentially traumatic events (PTE), exposure (impact) 153
- treatment
 - adherence, stigma (influence) 59–60
 - quality, stigma (impact) 59
- treatment as usual (TAU) 167
- trichomoniasis 386
- tricycle antidepressants (TCAs) 464
- tricyclic antidepressants (TCAs) 125
- Truvada (emtricitabine/tenofovir disoproxil fumarate), FDA approval 387
- tuberculosis 438
- type 2 diabetes, patient adherence 218
- Uniform Code of Military Justice (UCMJ), offenses 350
- unintentional patient nonadherence 217
- United Nations Sustainable Development Goals, mortality reduction 90–91
- urinary tract infections (UTIs), prevention 97

- urticaria (hives) 451
- US National Health Interview Survey, smoking/
smoking cessation information 226–227
- US Preventive Services Task Force (USPSTF)
 - adult depression screening,
recommendation 197–198
 - cancer screening recommendations 75
- vaginal bleeding (miscarriage symptom) 30
- VA Polytrauma Network Site, veteran
assessment 188
- ventromedial prefrontal cortex (VMPFC)
 - activation, increase 47
 - activity 52–53
- vertical sleeve gastrectomy, usage 177–178
- violence, threats 421
- viral hepatitis 385
- viral warts 451
- vocational retraining program 276
- voyeuristic disorder 390
- wake time after sleep onset (WASO) 461, 463
- walking
 - depressive symptoms (bidirectional, cyclical
association) 145–146
 - Theory of Unpleasant Symptoms
(TOUS) 145–146
- walking/depression
 - bidirectional association 144–145
 - relationship 143
- walking impairment 143–144
 - behavioral approaches 148
 - changes, impact 148
 - depressive symptomology, cyclical
associations 144
 - depressive symptoms
 - association, possibility 145
 - association, studies 146–147
 - impact, reasons 145
 - longitudinal associations, tested model 147
 - quality of life, association 145
- Walter Reed National Military Medical Center 350
- weight loss
 - behavior, organization 396
 - hypnosis, application 260
 - Look AHEAD study 175
 - medications, FDA approval 175
- weight-related behaviors, body image
(relationship) 4
- Weight Watchers weight-loss program 174
- Weiss, Steven 358
- well-being
 - enhancement, prevention (impact) 36
 - promotion, protective factors 37–38
 - tier 1 (three-tier model) 398–400
- wellness (promotion), reiki (usage) 252
- Wernicke's encephalopathy, chronic drinking
(impact) 437
- Western societies, body image concerns
(prevalence) 1
- whole medical systems 246
- women
 - anxiety, physician screening (ACOG
recommendations) 197–198
 - cancer mortality 73
 - depression, physician screening (ACOG
recommendations) 197–198
 - pregnancy, medically assisted reproduction
technology (stress) 29
- World Health Organization (WHO)
 - global goals (suicide) 209–210
 - recommendations (suicide) 210–211
- World Report on Disability* 278
- worthlessness, feelings 193
- yoga 246, 247, 400
 - styles 250
- youth
 - self-perception, discussion 40
 - tobacco company targeting 224–225
 - transgender youth, gender dysphoria
(psychologist treatment) 351–352
- Zyban (bupropion hydrochloride) 228

The Wiley Encyclopedia
of Health Psychology

The Wiley Encyclopedia of Health Psychology

Volume 4
Special Issues in Health Psychology

Edited by

Suzy Bird Gulliver

Editor-in-Chief

Lee M. Cohen

WILEY Blackwell

This edition first published 2021
© 2021 John Wiley & Sons Ltd

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by law. Advice on how to obtain permission to reuse material from this title is available at <http://www.wiley.com/go/permissions>.

The right of Lee M. Cohen to be identified as the author of the editorial material in this work has been asserted in accordance with law.

Registered Offices

John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Office

111 River Street, Hoboken, NJ 07030, USA

For details of our global editorial offices, customer services, and more information about Wiley products visit us at www.wiley.com.

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Limit of Liability/Disclaimer of Warranty

While the publisher and authors have used their best efforts in preparing this work, they make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives, written sales materials or promotional statements for this work. The fact that an organization, website, or product is referred to in this work as a citation and/or potential source of further information does not mean that the publisher and authors endorse the information or services the organization, website, or product may provide or recommendations it may make. This work is sold with the understanding that the publisher is not engaged in rendering professional services. The advice and strategies contained herein may not be suitable for your situation. You should consult with a specialist where appropriate. Further, readers should be aware that websites listed in this work may have changed or disappeared between when this work was written and when it is read. Neither the publisher nor authors shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

Library of Congress Cataloging-in-Publication Data

Names: Paul, Robert D., editor.

Title: The Wiley encyclopedia of health psychology / edited by Robert Paul,
St. Louis MO, USA ; editor-in-chief, Lee Cohen, The University of
Mississippi, MS, USA.

Description: First edition. | Hoboken : Wiley-Blackwell, 2021. | Includes
index. | Contents: volume. 1. Biological bases of health behavior.

Identifiers: LCCN 2020005459 (print) | LCCN 2020005460 (ebook) | ISBN
9781119057833 (v. 1 ; cloth) | ISBN 9781119057833 (v. 2 ; cloth) | ISBN
9781119057833 (v. 3 ; cloth) | ISBN 9781119057833 (v. 4 ; cloth) | ISBN
9781119675785 (adobe pdf) | ISBN 9781119675761 (epub)

Subjects: LCSH: Clinical health psychology—Encyclopedias.

Classification: LCC R726.7 .W554 2021 (print) | LCC R726.7 (ebook) | DDC
616.001/903—dc23

LC record available at <https://lcn.loc.gov/2020005459>

LC ebook record available at <https://lcn.loc.gov/2020005460>

Cover image and design: Wiley

Set in 10/12.5pts ITC Galliard by SPi Global, Pondicherry, India

10 9 8 7 6 5 4 3 2 1

Contents

List of Contributors	ix
Foreword	xxvii
Preface	xxix
Preface Volume 4: Special Issues in Health Psychology	xxxix
Editor-in-Chief Acknowledgments	xxxiii
The Biopsychosocial Model <i>Robert J. Gatchel, Christopher T. Ray, Nancy Kishino, and Athena Brindle</i>	1
Death and Dying <i>David E. Balk</i>	9
The Role of Regulation in Health Behavior (Listed in System as “Federal/State Regulation (FDA)”) <i>David B. Portnoy</i>	21
Funding for Health Psychology Research <i>Robert T. Croyle</i>	29
Genetics and Psychology (Health) <i>Nathan A. Kimbrel and Christopher Fink</i>	35
The Role of Cultural Mistrust in Health Disparities <i>Brandon L. Carlisle and Carolyn B. Murray</i>	43
Health Disparities <i>Melissa Ward-Peterson and Eric F. Wagner</i>	51

Injury, Accident, and Injury Prevention <i>Kristi Alexander and Riley Cropper</i>	61
Integrated Primary Care <i>Sandra B. Morissette and Brian Konecky</i>	67
Neuroimaging <i>Steven M. Nelson</i>	73
Palliative Care <i>Mamta Bhatnagar and Robert C. Arnold</i>	79
Patient Navigation/Community Health Workers <i>Kristen J. Wells and Janna R. Gordon</i>	91
The Patient Protection and Affordable Care Act and Health Psychology <i>Ronald H. Rozensky</i>	101
Pediatric Psychology <i>Christina M. Amaro, Michael C. Roberts, Andrea M. Garcia, and Jennifer B. Blossom</i>	115
Placebo Effect <i>Diletta Barbiani and Fabrizio Benedetti</i>	127
Prescriptive Authority for Psychologists <i>Wendy P. Linda and Robert E. McGrath</i>	139
Health-Related Quality of Life <i>Amy E. Ustjanauskas and Vanessa L. Malcarne</i>	149
The Roles of Health Psychologists in Surgery <i>Lauren Decapora-Ryan and John D. Robinson</i>	155
The Bidirectional Relationship Between Sleep and Health <i>Elizabeth M. Stewart, Shane Landry, Bradley A. Edwards, and Sean P. A. Drummond</i>	165
Telepsychology <i>Shawna Wright and Eve-Lynn Nelson</i>	189
Traditional Healing Practices and Healers <i>Robert M. Huff</i>	199
Training in Health Psychology <i>Kevin T. Larkin</i>	205

Training Non-psychologists in the Healthcare Settings <i>Cesar A. Gonzalez, Kristine M. Diaz, and Richard J. Seime</i>	213
Transgender Health <i>DeAnna L. Mori, Colleen A. Sloan, Heather Walton Flynn, and Claire Burgess</i>	219
Veterans' Health Issues <i>Cynthia J. Willmon</i>	229
Team Science in Health <i>Sasha Cukier, Ekaterina Klyachko, and Bonnie Spring</i>	243
Family and Health <i>Brittany N. Sherrill, Janet Deatrck, and Keith Sanford</i>	253
Index	263

List of Contributors

- Amanda M. Acevedo**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Zeba Ahmad**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- Leona S. Aiken**, Department of Psychology, Arizona State University, Tempe, AZ, USA
- Olusola Ajilore**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Damla E. Aksen**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Kristi Alexander**, Department of Psychology, University of Central Florida, Orlando, FL, USA
- Andrea G. Alioto**, Palo Alto University, Palo Alto, CA, USA
Department of Neurology, University of California, Davis, CA, USA
- Michael L. Alosco**, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA
- Christina M. Amaro**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Judith Andersen**, Department of Psychology, University of Toronto, Mississauga, ON, Canada
- Aviva H. Ariel**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Danielle Arigo**, Psychology Department, The University of Scranton, Scranton, PA, USA
- Jamie Arndt**, Psychology Department, University of Missouri System, Columbia, MO, USA
- Robert C. Arnold**, Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
- Melissa V. Auerbach**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Lisa Auster-Gussman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Aya Avishai**, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Hoda Badr**, Icahn School of Medicine at Mount Sinai, New York, NY, USA
- David E. Balk**, Department of Health and Nutrition Sciences, Brooklyn College of the City University of New York, Brooklyn, NY, USA

- Kirk Ballew**, Department of Communication, Fontbonne University, St. Louis, MO, USA
 Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
 Department of Communication, University of Illinois at Urbana-Champaign College of
 Liberal Arts and Sciences, Urbana, IL, USA
- Diletta Barbiani**, Department of Neuroscience, University of Turin Medical School, Turin,
 Italy
 Hypoxia Medicine, Plateau Rosà, Switzerland
- Jeffrey E. Barnett**, College of Arts and Sciences, Loyola University Maryland, Baltimore,
 MD, USA
- Petronilla Battista**, Istituti Clinici Scientifici Maugeri IRCCS, Pavia, Institute of Bari, Italy
- Brittney Becker**, Psychology Department, Texas A&M University System, College Station,
 TX, USA
- Alexandra A. Belzer**, Colby College, Waterville, ME, USA
- Fabrizio Benedetti**, Department of Neuroscience, University of Turin Medical School,
 Turin, Italy
 Hypoxia Medicine, Plateau Rosà, Switzerland
- Danielle S. Berke**, Massachusetts Veterans Epidemiology Research and Information Center
 (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Mamta Bhatnagar**, Department of Medicine, University of Pittsburgh Medical Center,
 Pittsburgh, PA, USA
- Kelly Biegler**, Department of Psychological Science, University of California, Irvine, CA,
 USA
- Susan J. Blalock**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill,
 Chapel Hill, NC, USA
- Brittany E. Blanchard**, Department of Psychological Sciences, Texas Tech University,
 Lubbock, TX, USA
- Jennifer B. Blossom**, Clinical Child Psychology Program, University of Kansas, Lawrence,
 KS, USA
- Ryan Bogdan**, BRAIN Lab, Psychological and Brain Sciences, Washington University in
 St. Louis, St. Louis, MO, USA
- Jacob Bolzenius**, Missouri Institute of Mental Health, University of Missouri-St. Louis,
 St. Louis, MO, USA
- Bruce Bongar**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Dianna Boone**, Clinical Psychology Program, Department of Psychological Sciences, Texas
 Tech University, Lubbock, TX, USA
- Jerica X. Bornstein**, Department of Human Development and Family Sciences, University of
 Texas at Austin, Austin, TX, USA
- Joaquín Borrego Jr.**, Department of Psychological Sciences, Texas Tech University, Lubbock,
 TX, USA
- Kyle J. Bourassa**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Lara A. Boyd**, Department of Physical Therapy, University of British Columbia, Vancouver,
 BC, Canada
 Djavad Mowafaghian Centre for Brain Health, University of British Columbia, Vancouver,
 BC, Canada
- Patrick Boyd**, Department of Psychology, University of South Florida, Tampa, FL, USA
- Adam M. Brickman**, Columbia University Medical Center, New York, NY, USA
- Athena Brindle**, Department of Psychology, College of Science, The University of Texas at
 Arlington, Arlington, TX, USA

- Jennifer L. Brown**, Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati College of Medicine, Cincinnati, OH, USA
- Kathleen S. Brown**, Private Practice, Fort Myers, FL, USA
- Wilson J. Brown**, Pennsylvania State University, The Behrend College, Erie, PA, USA
- Steven E. Bruce**, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Angela D. Bryan**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Katherine R. Buchholz**, VA Boston Healthcare System, Boston, MA, USA
National Center for Post Traumatic Stress Disorder, White River Junction, VT, USA
- Claire Burgess**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Edith Burns**, Medical College of Wisconsin, Milwaukee, WI, USA
- Rachel J. Burns**, Douglas Mental Health University Institute, McGill University, Montreal, QC, Canada
- Johannes B.J. Bussmann**, Erasmus MC University Medical Center Rotterdam, CA Rotterdam, South Holland, The Netherlands
- Fawn C. Caplandies**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Angela L. Carey**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Brandon L. Carlisle**, Department of Psychiatry, University of California, San Diego, CA, USA
- Delesha M. Carpenter**, Eshelman School of Pharmacy, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- Allison J. Carroll**, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL, USA
- Charles S. Carver**, Department of Psychology, University of Miami, Coral Gables, FL, USA
Center for Advanced Study in the Behavioral Sciences, Stanford University, Stanford, CA, USA
- Isabella Castiglioni**, Dipartimento di Fisica “G. Occhialini”, Università degli Studi di Milano-Bicocca – Piazza della Scienza, Milan, Italy
- Gretchen B. Chapman**, Department of Psychology, Rutgers University–New Brunswick, New Brunswick, NJ, USA
- Alyssa C.D. Cheadle**, Psychology Department, Hope College, Holland, MI, USA
- Rebecca E. Chesher**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- William J. Chopik**, Psychology Department, Michigan State University, East Lansing, MI, USA
- Nicholas J.S. Christenfeld**, Department of Psychology, University of California San Diego, La Jolla, CA, USA
- L.E. Chun**, University of Colorado, Boulder, CO, USA
- Briana Cobos**, Department of Psychology, Texas State University, San Marcos, TX, USA
- Alex S. Cohen**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Jenna Herold Cohen**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Lee M. Cohen**, Department of Psychology, College of Liberal Arts, The University of Mississippi, Oxford, MS, USA
- Catherine Cook-Cottone**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA

- Sarah A. Cooley**, Department of Neurology, School of Medicine, Washington University in Saint Louis, St. Louis, MO, USA
- Stephen Correia**, Department of Psychiatry and Human Behavior, Alpert Medical School, Brown University, Providence, RI, USA
- Patrick Corrigan**, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
- Juliann Cortese**, School of Information, Florida State University, Tallahassee, FL, USA
- Riley Cropper**, Stanford University Counseling and Psychological Services, Stanford Hospital and Clinics, Stanford, CA, USA
- Robert T. Croyle**, National Cancer Institute, Bethesda, MD, USA
- Tegan Cruwys**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Sasha Cukier**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Gregory Dahl**, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Beth D. Darnall**, Department of Anesthesiology, Perioperative, and Pain Medicine, Stanford University School of Medicine, Stanford, CA, USA
- Savannah Davidson**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Elizabeth L. Davis**, Department of Psychology, University of California, Riverside, CA, USA
- Rachael L. Deardorff**, Center for Biomedical Imaging, Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Janet Deatrick**, Department of Family & Community Health, University of Pennsylvania School of Nursing, Philadelphia, PA, USA
- Lauren Decaportale-Ryan**, Departments of Psychiatry, Medicine, & Surgery, University of Rochester Medical Center, Rochester, NY, USA
- Jordan Degelia**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Anita Delongis**, Department of Psychology, University of British Columbia, Vancouver, BC, Canada
- Naiara Demnitz**, Department of Psychiatry, University of Oxford, Oxford, UK
- Eliot Dennard**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Kristine M. Diaz**, Walter Reed National Military Medical Center, Bethesda, MD, USA
- Sally S. Dickerson**, Department of Psychology, Pace University, New York, NY, USA
- M. Robin DiMatteo**, Department of Psychology, University of California Riverside, Riverside, CA, USA
- Sona Dimidjian**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Genevieve A. Dingle**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Laura J. Dixon**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Michelle R. Dixon**, Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Tiffany S. Doherty**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Michael D. Dooley**, Department of Psychology, University of California, Riverside, Riverside, CA, USA

- Tessa L. Dover**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Colleen Doyle**, Institute of Child Development, College of Education and Human Development, University of Minnesota, Minneapolis, MN, USA
- Ira Driscoll**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Claudia Drossel**, Department of Psychology, Eastern Michigan University College of Arts and Sciences, Ypsilanti, MI, USA
- Sean P.A. Drummond**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Christine Dunkel Schetter**, Department of Psychology, University of California, Los Angeles, CA, USA
- William L. Dunlop**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Jacqueline C. Duperrouzel**, Florida International University, Miami, FL, USA
- Allison Earl**, Department of Psychology, University of Michigan, Ann Arbor, MI, USA
- Amanda C. Eaton**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Ulrich W. Ebner-Priemer**, Karlsruhe Institute of Technology, Karlsruhe, Germany
- Robin S. Edelstein**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Jamie Edgin**, Department of Psychology, The University of Arizona, Tucson, AZ, USA
- Bradley A. Edwards**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Naomi I. Eisenberger**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Amber S. Emanuel**, Psychology Department, University of Florida, Gainesville, FL, USA
- Patrick England**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Ipek Ensari**, Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA
- Emily Evans**, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
- James Evans**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Anne M. Fairlie**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Angelica Falkenstein**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Zachary Fetterman**, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
- Christopher Fink**, Durham Veterans Affairs Medical Center, Durham, NC, USA
- North Carolina State University, Raleigh, NC, USA
- Alexandra N. Fisher**, Psychology Department, University of Victoria, Victoria, BC, Canada
- Tabitha Fleming**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Tasheia Floyd**, Washington University School of Medicine, St. Louis, MO, USA

- Lauren A. Fowler**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- Elinor Flynn**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Heather Walton Flynn**, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Harvard Medical School, Boston, MA, USA
- Elizabeth S. Focella**, Department of Psychology, University of Wisconsin Oshkosh, Oshkosh, WI, USA
- Amy Frers**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA
- Howard S. Friedman**, Psychology Department, University of California Riverside, Riverside, CA, USA
- Paul T. Fuglestad**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Kentaro Fujita**, Department of Psychology, The Ohio State University, Columbus, OH, USA
- Kristel M. Gallagher**, Thiel College, Greenville, PA, USA
- Andrea M. Garcia**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- Paola García-Egan**, Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, MO, USA
- Robert García**, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA
- Casey K. Gardiner**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Robert J. Gatchel**, Endowment Professor of Clinical Health Psychology Research, Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA
- Justine Megan Gatt**, Neuroscience Research Australia, Sydney, NSW, Australia
School of Psychology, University of New South Wales, Sydney, NSW, Australia
- Nicole K. Gause**, Department of Psychology, University of Cincinnati, Cincinnati, OH, USA
- Ashwin Gautam**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Fiona Ge**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Andrew L. Geers**, Department of Psychology, University of Toledo, Toledo, OH, USA
- Margaux C. Genoff**, Department of Psychiatry and Behavioral Sciences, Memorial Sloan Kettering Cancer Center, New York, NY, USA
- James I. Gerhart**, Department of Psychosocial Oncology, Cancer Integrative Medicine Program, Rush University Medical Center, Chicago, IL, USA
- Meg Gerrard**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Frederick X. Gibbons**, Department of Psychological Sciences, University of Connecticut, Storrs, CT, USA
- Arielle S. Gillman**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Marci E.J. Gleason**, Department of Human Development and Family Sciences, University of Texas at Austin, Austin, TX, USA
- Teresa Gobble**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

- Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA
Katharina Sophia Goerlich, Department of Psychiatry, Psychotherapy and Psychosomatics,
RWTH Aachen University, Aachen, Germany
Jamie L. Goldenberg, Department of Psychology, University of South Florida, Tampa,
FL, USA
Cesar A. Gonzalez, Mayo Clinic Minnesota, Rochester, MN, USA
Raul Gonzalez, Florida International University, Miami, FL, USA
Brittany F. Goodman, Department of Psychological Sciences, Center for Trauma Recovery,
University of Missouri-St. Louis, St. Louis, MO, USA
Janna R. Gordon, San Diego State University and SDSU/UC San Diego Joint Doctoral
Program in Clinical Psychology, San Diego, CA, USA
Edward C. Green, Department of Anthropology, The George Washington University,
Washington, DC, USA
Joseph P. Green, Department of Psychology, Ohio State University at Lima, Lima, OH,
USA
Michael G. Griffin, Department of Psychological Sciences and Center for Trauma Recovery,
University of Missouri–St. Louis, St. Louis, MO, USA
Steven Grindel, Medical College of Wisconsin, Milwaukee, WI, USA
Kristina Gryboski, Project Hope, Millwood, VA, USA
Suzy Bird Gulliver, Baylor and Scott and White Hospitals and Clinics, Waco, TX, USA
School of Public Health, Texas A&M University, College Station, TX, USA
Megan R. Gunnar, Institute of Child Development, College of Education and Human
Development, University of Minnesota, Minneapolis, MN, USA
Jenna Haddock, Department of Psychological Sciences, University of Missouri-St. Louis,
St. Louis, MO, USA
Martin S. Hagger, Health Psychology and Behavioral Medicine Research Group, School of
Psychology and Speech Pathology, Faculty of Health Sciences, Curtin University, Perth,
WA, Australia
Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland
School of Applied Psychology and Menzies Health Institute Queensland, Behavioural Bases
for Health, Griffith University, Brisbane, QLD, Australia
Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central
Queensland University, Rockhampton, QLD, Australia
Jada G. Hamilton, Department of Psychiatry and Behavioral Sciences, Memorial Sloan
Kettering Cancer Center, New York, NY, USA
Paul K.J. Han, Center for Outcomes Research and Evaluation, Maine Medical Center
Research Institute, Portland, ME, USA
Grace E. Hanley, Psychology Department, University of California, Riverside, Riverside,
CA, USA
Madeline B. Harms, Department of Psychology, University of Wisconsin-Madison, Madison,
WI, USA
Adam Harris, Department of Psychology and University Health Sciences Center, University
of Colorado Denver, Denver, CO, USA
Lauren N. Harris, VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA
Peter R. Harris, School of Psychology, University of Sussex, Brighton & Hove, UK
Philine S. Harris, School of Psychology, University of Sussex, Brighton & Hove, UK
William Hart, Psychology Department, University of Alabama, Tuscaloosa, AL, USA
M.J. Hartsock, University of Colorado, Boulder, CO, USA

- Kelly B. Haskard-Zolnierrek**, Department of Psychology, Texas State University, San Marcos, TX, USA
- S. Alexandar Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Catherine Haslam**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Karen Hasselmo**, Department of Psychology, University of Arizona, Tucson, AZ, USA
- Kathryn S. Hayward**, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada
- Stroke Division, Florey Institute of Neuroscience and Mental Health, University of Melbourne, Melbourne, VIC, Australia
- NHMRC Centre for Research Excellence in Stroke Rehabilitation and Recovery, Parkville, VIC, Australia
- Jodi M. Heaps-Woodruff**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Dietlinde Heilmayr**, Psychology Department, Moravian College, Bethlehem, PA, USA
- Vicki S. Helgeson**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Joseph A. Helpern**, Center for Biomedical Imaging, Department of Neuroscience, Department of Neurology, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Garrett Hisler**, Department of Psychology, Iowa State University, Ames, IA, USA
- Molly B. Hodgkins**, Department of Psychological and Brain Sciences, University of Maine, Orono, ME, USA
- Michael Hoerger**, Department of Psychology, Tulane University, New Orleans, LA, USA
- Jeanne S. Hoffman**, US Department of the Army, Washington, DC, USA
- Stephanie A. Hooker**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Vera Hoorens**, Center for Social and Cultural Psychology, Katholieke Universiteit Leuven, Leuven, Belgium
- Arpine Hovasapian**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jennifer L. Howell**, Psychology Department, Ohio University, Athens, OH, USA
- Robert M. Huff**, Department of Health Sciences, California State University, Northridge, Morgan Hill, CA, USA
- Jacob Huffman**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Department of Psychiatry, Washington University in St. Louis School of Medicine, St. Louis, MO, USA
- Lauren J. Human**, Department of Psychology, McGill University, Montreal, QC, Canada
- Jeffrey M. Hunger**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Tanya Hunt**, Department of Clinical Psychology, Palo Alto University, Palo Alto, California, USA
- Steven Hyde**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- Christina Di Iorio**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA

- Jamie M. Jacobs**, Department of Psychiatry, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA
- Lauren James**, Department of Psychology, University of North Florida, Jacksonville, FL, USA
- Rebekah Jazdzewski**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Brooke N. Jenkins**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Jens H. Jensen**, Center for Biomedical Imaging, Department of Neuroscience, and Department of Radiology and Radiological Science, Medical University of South Carolina, Charleston, SC, USA
- Donna C. Jessop**, School of Psychology, University of Sussex, Brighton & Hove, UK
- Jolanda Jetten**, Faculty of Health and Behavioural Sciences, School of Psychology, University of Queensland, St. Lucia, QLD, Australia
- Kimberly Johnson**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- Doerte U. Junghaenel**, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA
- Vanessa Juth**, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA
- Samantha Kahn**, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
- David A. Kalkstein**, Department of Psychology, New York University, New York, NY, USA
- Alexander Karan**, Department of Psychology, University of California, Riverside, CA, USA
- Natalia M. Kecala**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri–St. Louis, St. Louis, MO, USA
- Robert G. Kent de Grey**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- Margaret L. Kern**, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia
- Nathan A. Kimbrel**, Department of Veterans Affairs' VISN 6 Mid-Atlantic MIRECC, Durham, NC, USA
Durham Veterans Affairs Medical Center, Durham, NC, USA
Psychiatry and Behavioral Sciences, Duke University School of Medicine, Durham, NC, USA
- Tricia Z. King**, The Neuroscience Institute, Georgia State University, Atlanta, GA, USA
- Nancy Kishino**, West Coast Spine Restoration, Riverside, CA, USA
- Ekaterina Klyachko**, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA
- Brian Konecky**, Boise VAMC Virtual Integrated Multisite Patient Aligned Care Team Hub, Boise, ID, USA
- Gerald P. Koocher**, Department of Psychology, DePaul University, Chicago, IL, USA
- Laura E. Korthauer**, Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, USA
- Abigail O. Kramer**, Palo Alto University, Palo Alto, CA, USA
Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Joel H. Kramer**, Weill Institute for Neurosciences, University of California, San Francisco Memory and Aging Center, San Francisco, CA, USA
- Zlatan Krizan**, Department of Psychology, Iowa State University, Ames, IA, USA

- Monica F. Kurylo**, Departments of Medicine and Public Health, University of Kansas Medical Center, Kansas City, KS, USA
- Onawa P. LaBelle**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa H. Laitner**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Mark J. Landau**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Shane Landry**, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia
- Sleep and Circadian Medicine Laboratory, Department of Physiology, School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia
- Kevin T. Larkin**, Department of Psychology, West Virginia University, Morgantown, WV, USA
- Jason M. Lavender**, Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA
- Kristin Layous**, Department of Psychology, California State University East Bay, Hayward, CA, USA
- Thanh Le**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Thad R. Leffingwell**, Department of Psychology, Oklahoma State University, Stillwater, OK, USA
- Angela M. Legg**, Department of Psychology, Pace University, New York, NY, USA
- Jana Lembke**, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA
- Elizabeth Lemon**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Richie L. Lenne**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Alex Leow**, Department of Psychiatry, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Department of Bioengineering, College of Medicine, University of Illinois at Chicago, Chicago, IL, USA
- Howard Leventhal**, Department of Psychology, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA
- Linda J. Levine**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Melissa A. Lewis**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA
- Meng Li**, Department of Health and Behavior Sciences, University of Colorado Denver, Denver, CO, USA
- Huijun Liao**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Alexander P. Lin**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Wendy P. Linda**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Nikolette P. Lipsey**, Psychology Department, University of Florida, Gainesville, FL, USA
- Dana M. Litt**, Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

- Andrew K. Littlefield**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Brett Litz**, Department of Psychology, Boston University & VA Boston Healthcare System, Boston, MA, USA
- Marci Lobel**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Todd Lucas**, C. S. Mott Endowed Professor of Public Health Associate Professor of Epidemiology and Biostatistics Division of Public Health College of Human Medicine Michigan State University, Wayne State University, Detroit, MI, USA
- Mia Liza A. Lustria**, School of Information, Florida State University, Tallahassee, FL, USA
- Steven Jay Lynn**, Department of Psychology, Binghamton University, Binghamton, NY, USA
- Salvatore R. Maddi**, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
- Renee E. Magnan**, Washington State University Vancouver, Vancouver, WA, USA
- Brenda Major**, Department of Psychological and Brain Sciences, University of California Santa Barbara, Santa Barbara, CA, USA
- Vanessa L. Malcarne**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
UC San Diego Moores Cancer Center, San Diego, CA, USA
Department of Psychology, San Diego State University, San Diego, CA, USA
- Kelsey A. Maloney**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Andrew W. Manigault**, Psychology Department, Ohio University, Athens, OH, USA
- Traci Mann**, Psychology Department, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Soe Mar**, Neurology and Pediatrics, Division of Pediatric Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Shannon M. Martin**, Department of Psychology, Central Michigan University, Mount Pleasant, MI, USA
- Allison Marziliano**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Katilyn Mascatelli**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Tyler B. Mason**, Department of Psychology, University of Southern California, Los Angeles, CA, USA
Neuropsychiatric Research Institute, Fargo, ND, USA
- Kevin S. Masters**, Psychology Department, University of Colorado at Denver – Anschutz Medical Campus, Aurora, CO, USA
- Kevin D. McCaul**, North Dakota State University, Fargo, ND, USA
- Tara P. McCoy**, Psychology Department, University of California, Riverside, Riverside, CA, USA
- Michael McCrea**, Brain Injury Research Program, Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA
- Scout N. McCully**, Department of Psychological Sciences, Kent State University, Kent OH, USA
- Don McGeary**, Department of Psychiatry, Department of Family and Community Medicine, The University of Texas Health Science Center San Antonio, San Antonio, TX, USA

- Jessica McGovern**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Robert E. McGrath**, School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA
- Michael P. Mead**, North Dakota State University, Fargo, ND, USA
- Alan Meca**, Department of Psychology, Old Dominion University, Norfolk, VA, USA
- Matthias R. Mehl**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Timothy P. Melchert**, Department of Psychology, Marquette University, Milwaukee, WI, USA
- Paola Mendoza-Rivera**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Maria G. Mens**, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
- Sai Merugumala**, Department of Radiology, Center for Clinical Spectroscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
- Rebecca A. Meza**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Dara Mickschl**, Medical College of Wisconsin, Milwaukee, WI, USA
- Tricia A. Miller**, Santa Barbara Cottage Hospital, Santa Barbara, CA, USA
- Department of Psychology, University of California Riverside, Riverside, CA, USA
- Kyle R. Mitchell**, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
- Mona Moieni**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Phillip J. Moore**, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA
- DeAnna L. Mori**, VA Boston Healthcare System, West Roxbury, MA, USA
- Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA
- Sandra B. Morissette**, Department of Psychology, The University of Texas at San Antonio College of Sciences, San Antonio, TX, USA
- Amber Morrow**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Ariel J. Mosley**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Robert W. Motl**, Department of Physical Therapy, University of Alabama at Birmingham, Birmingham, AL, USA
- Laurent Mottron**, Department of Psychiatry, Faculty of Medicine, Universite de Montreal, Montreal, QC, Canada
- Anne Moyer**, Department of Psychology, Stony Brook University, Stony Brook, NY, USA
- Elaine M. Murphy**, Population Reference Bureau, Washington, DC, USA
- Carolyn B. Murray**, Psychology, University of California, Riverside, CA, USA
- Christopher S. Nave**, Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA
- Department of Psychology, Rutgers University Camden, Camden, NJ, USA
- Eve-Lynn Nelson**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Steven M. Nelson**, VISN 17 Center of Excellence for Research on Returning War Veterans, Waco, TX, USA
- Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA

Center for Vital Longevity, School of Behavioral and Brain Sciences, University of Texas at Dallas, Dallas, TX, USA

Elizabeth Neumann, Medical College of Wisconsin, Milwaukee, WI, USA

Jason L. Neva, Department of Physical Therapy, University of British Columbia, Vancouver, BC, Canada

Jody S. Nicholson, Department of Psychology, University of North Florida, Jacksonville, FL, USA

Christina Nisson, Department of Psychology, University of Michigan, Ann Arbor, MI, USA

Mary-Frances O'Connor, Psychology Department, University of Arizona, Tucson, AZ, USA

Laura O'Hara, Department of Communication, Fontbonne University, St. Louis, MO, USA

Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA

Gbolahan O. Olanubi, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA

Nils Olsen, Department of Psychological and Brain Sciences, The George Washington University, Washington, DC, USA

Yimi Omofuma, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA

Elizabeth Ortiz-Gonzalez, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Ileana Pacheco-Colón, Florida International University, Miami, FL, USA

Aliza A. Panjwani, Graduate School and University Center, City University of New York, New York, NY, USA

Mike C. Parent, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Department of Educational Psychology, University of Texas at Austin, Austin, TX, USA

Thomas J. Parkman, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Robert H. Paul, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

Missouri Institute of Mental Health, St. Louis, MO, USA

Stephanie A. Peak, Battelle Memorial Institute, Columbus, OH, USA

Michael G. Perri, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

Laura Perry, Department of Psychology, Tulane University, New Orleans, LA, USA

Alyssa Phelps, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Carissa L. Philippi, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA

L. Alison Phillips, Iowa State University, Ames, IA, USA

Paula R. Pietromonaco, Department of Psychological and Brain Sciences, University of Massachusetts Amherst, Amherst, MA, USA

Craig P. Pollizi, Department of Psychology, Binghamton University, Binghamton, NY, USA

Seth D. Pollak, Department of Psychology, University of Wisconsin–Madison, Madison, WI, USA

David B. Portnoy, US Food and Drug Administration, Silver Spring, MD, USA

Rachel Postupack, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

- Matthew R. Powell**, Division of Neurocognitive Disorders, Department of Psychiatry & Psychology, Mayo Clinic Minnesota, Rochester, MN, USA
- Candice Presseau**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Sarah D. Pressman**, Department of Psychological Science, University of California Irvine, Irvine, CA, USA
- Rebecca N. Preston-Campbell**, Missouri Institute of Mental Health, St. Louis, MO, USA
- Isabel F. Ramos**, Department of Psychology, University of California, Los Angeles, CA, USA
- Christopher T. Ray**, College of Health Sciences, Texas Woman's University, Denton, TX, USA
- Amanda L. Rebar**, Centre for Physical Activity Studies, School of Human, Health and Social Sciences, Central Queensland University, Rockhampton, QLD, Australia
- Allecia E. Reid**, Colby College, Waterville, ME, USA
- May Reinert**, Department of Psychology, Pace University, New York, NY, USA
- Kelly E. Rentscher**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Tracey A. Revenson**, Department of Psychology, Hunter College, New York, NY, USA
- C. Steven Richards**, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Laura River**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Megan L. Robbins**, Department of Psychology, University of California, Riverside, CA, USA
- Michael C. Roberts**, Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA
- John D. Robinson**, Department of Surgery and Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine/Hospital, Washington, DC, USA
- Theodore F. Robles**, Psychology Department, University of California Los Angeles, CA, USA
- Catherine Rochefort**, Department of Psychology, Southern Methodist University, Dallas, TX, USA
- Daniel G. Rogers**, Department of Psychology, University of Mississippi, MS, USA
- John L. Romano**, Department of Educational Psychology, University of Minnesota Twin Cities, Minneapolis, MN, USA
- Karen S. Rook**, Department of Psychological Science, University of California, Irvine, CA, USA
- Ann Rosenthal**, Medical College of Wisconsin, Milwaukee, WI, USA
- J. Megan Ross**, Florida International University, Miami, FL, USA
- Tania L. Roth**, Department of Psychological and Brain Sciences, University of Delaware, Newark, DE, USA
- Alexander J. Rothman**, Department of Psychology, University of Minnesota, Minneapolis, MN, USA
- Ronald H. Rozensky**, Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
- Kristen L. Rudd**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Kirstie K. Russell**, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada
- Arseny A. Ryazanov**, Department of Psychology, University of California San Diego, La Jolla, CA, USA

- Napapon Sailasuta**, Centre for Addiction and Mental Health, Toronto, ON, Canada
Stella Sakhon, Department of Psychology, The University of Arizona, Tucson, AZ, USA
Lauren E. Salminen, Imaging Genetics Center, University of Southern California, Marina del Rey, CA, USA
Christian Salvatore, Scuola Universitaria Superiore IUSS Pavia, Piazza della Vittoria, Pavia, Italy
DeepTrace Technologies S.R.L., Via Conservatorio, Milan, Italy
Peter M. Sandman, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
Keith Sanford, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
David A. Sbarra, Department of Psychology, University of Arizona, Tucson, AZ, USA
Michael F. Scheier, Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
Adam T. Schmidt, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Elana Schwartz, Department of Psychology, Louisiana State University College of Science, Baton Rouge, LA, USA
Esther N. Schwartz, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
Seth J. Schwartz, Department of Public Health Sciences, University of Miami Miller School of Medicine, Miami, FL, USA
Ralf Schwarzer, Health Psychology, Freie Universität Berlin, Berlin, Germany
Health Psychology, Australian Catholic University, Melbourne, VIC, Australia
Richard J. Seime, Mayo Clinic Minnesota, Rochester, MN, USA
Claire E. Sexton, Department of Psychiatry, University of Oxford, Oxford, UK
Louise Sharpe, School of Psychology, Faculty of Science, The University of Sydney, Sydney, NSW, Australia
Lindsay Sheehan, Department of Psychology, Illinois Institute of Technology College of Science, Chicago, IL, USA
Paschal Sheeran, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
Daniel Sheinbein, BRAIN Lab, Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA
James A. Shepperd, Psychology Department, University of Florida, Gainesville, FL, USA
Brittany N. Sherrill, Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
Molin Shi, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
Emily W. Shih, Department of Psychology, University of California, Riverside, CA, USA
Ben W. Shulman, Psychology Department, University of California, Los Angeles, CA, USA
Elizabeth Silberman, Neurology Resident, Washington University in St. Louis, St. Louis, MO, USA
Department of Neurology, Oregon Health and Science University, Portland, OR, USA
Roxane Cohen Silver, Department of Psychological Science, University of California, Irvine, Irvine, CA, USA
Colleen A. Sloan, VA Boston Healthcare System, West Roxbury, MA, USA
Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA

Rachel Smallman, Psychology Department, Texas A&M University System, College Station, TX, USA

Kathryn E. Smith, Neuropsychiatric Research Institute, Fargo, ND, USA
Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA

Patrick J. Smith, Duke University Medical Center, Durham, NC, USA

Todd A. Smitherman, Department of Psychology, University of Mississippi, University, MS, USA

Joshua M. Smyth, The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA

Dara H. Sorkin, Department of Psychological Science, University of California, Irvine, CA, USA

R. L. Spencer, University of Colorado, Boulder, CO, USA

Melissa Spina, Psychology Department, University of Missouri System, Columbia, MO, USA

Bonnie Spring, Department of Preventive Medicine, Northwestern University, Evanston, IL, USA

Annette L. Stanton, Department of Psychology, University of California, Los Angeles, Los Angeles, CA, USA

Ellen Stephenson, Department of Psychology, University of British Columbia, Vancouver, BC, Canada

Robert A. Stern, Boston University Alzheimer's Disease Center and Boston University CTE Center, Department of Neurology, Boston University School of Medicine, Boston, MA, USA

Department of Neurosurgery and Anatomy & Neurobiology, Boston University School of Medicine, Boston, MA, USA

Angela K. Stevens, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA

Courtney J. Stevens, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA

Elizabeth M. Stewart, Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences, Monash University, Melbourne, VIC, Australia

Danu Anthony Stinson, Psychology Department, University of Victoria, Victoria, BC, Canada

Michelle L. Stock, Department of Psychological and Brain Sciences, George Washington University, Washington, DC, USA

Arthur A. Stone, USC Dornsife Center for Self-Report Science, University of Southern California, Los Angeles, CA, USA

Department of Psychology, Dana and David Dornsife College of Letters Arts and Sciences, University of Southern California, Los Angeles, CA, USA

Jeff Stone, Psychology Department, University of Arizona, Tucson, AZ, USA

John A. Sturgeon, Center for Pain Relief, University of Washington, Seattle, WA, USA

Kieran T. Sullivan, Department of Psychology, Santa Clara University, Santa Clara, CA, USA

Amy Summerville, Psychology Department, Miami University, Oxford, OH, USA

Jessie Sun, Center for Positive Psychology, Graduate School of Education, University of Melbourne, Parkville, VIC, Australia

Department of Psychology, University of California, Davis, CA, USA

Steve Sussman, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

- Trevor James Swanson**, Department of Psychology, University of Kansas, Lawrence, KS, USA
- Allison M. Sweeney**, Department of Psychology, University of South Carolina, Columbia, SC, USA
- Kate Sweeny**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Sergey Tarima**, Medical College of Wisconsin, Milwaukee, WI, USA
- Carly A. Taylor**, Boston Medical Center, Boston, MA, USA
- George T. Taylor**, Behavioral Neuroscience Program, Department of Psychological Sciences, University of Missouri–St. Louis, St. Louis, MO, USA
- Interfakultäre Biomedizinische Forschungseinrichtung (IBF) der Universität Heidelberg, Heidelberg, Germany
- A. Janet Tomiyama**, Psychology Department, University of California Los Angeles, Los Angeles, CA, USA
- Victoria A. Torres**, Department of Psychology, College of Liberal Arts, University of Mississippi, Oxford, MS, USA
- David R.M. Trotter**, Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA
- Bert N. Uchino**, Department of Psychology, University of Utah, Salt Lake City, UT, USA
- John A. Updegraff**, Department of Psychological Sciences, Kent State University, Kent, OH, USA
- Jay Urbain**, Milwaukee School of Engineering, Milwaukee, WI, USA
- Amy E. Ustjanauskas**, San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- UC San Diego Moores Cancer Center, San Diego, CA, USA
- Jason Van Allen**, Clinical Psychology Program, Department of Psychological Sciences, Texas Tech University, Lubbock, TX, USA
- Erika D. Van Dyke**, Department of Sport Sciences, College of Physical Activity and Sport Sciences, West Virginia University, Morgantown, WV, USA
- Department of Psychology, Springfield College, Springfield, MA, USA
- Judy L. Van Raalte**, Department of Psychology, Springfield College, Springfield, MA, USA
- Maryke Van Zyl-Harrison**, Department of Clinical Psychology, Palo Alto University, Palo Alto, CA, USA
- Rachel Vanderkruik**, Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO, USA
- Elizabeth J. Vella**, Department of Psychology, University of Southern Maine, Portland, ME, USA
- Birte von Haaren-Mack**, German Sport University Cologne, Köln, Nordrhein-Westfalen, Germany
- David Von Nordheim**, University of Missouri–St. Louis, St. Louis, MO, USA
- Missouri Institute of Mental Health, St. Louis, MO, USA
- Mikhail Votinov**, Department of Psychiatry, Psychotherapy and Psychosomatics, RWTH Aachen University, Aachen, Germany
- Institute of Neuroscience and Medicine (INM-10), Jülich, Germany
- Rebecca K. Vujnovic**, Department of Psychology, University at Buffalo, The State University of New York, Buffalo, NY, USA
- Amy Wachholtz**, Department of Psychology and University Health Sciences Center, University of Colorado Denver, Denver, CO, USA

- Eric F. Wagner**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Ryan J. Walker**, Psychology Department, Miami University, Oxford, Ohio, USA
- Rachel A. Wamser-Nanney**, Department of Psychological Sciences, Center for Trauma Recovery, University of Missouri-St. Louis, St. Louis, MO, USA
- Britney M. Wardecker**, Psychology Department, University of Michigan, Ann Arbor, MI, USA
- Melissa Ward-Peterson**, Department of Epidemiology, Florida International University, Miami, FL, USA
- Lisa Marie Warner**, Health Psychology, Freie Universitat Berlin, Berlin, Germany
- Neil D. Weinstein**, Department of Human Ecology, Rutgers University New Brunswick, New Brunswick, NJ, USA
- Suzanne E. Welcome**, Department of Communication, Fontbonne University, St. Louis, MO, USA
- Department of Communication, University of Missouri at Saint Louis, St. Louis, MO, USA
- Kristen J. Wells**, San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
- Amy Wenzel**, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, PA, USA
- Private Practice, Rosemont, PA, USA
- Summer L. Williams**, Department of Psychology, Westfield State University, Westfield, MA, USA
- Cynthia J. Willmon**, Reno VA Sierra Nevada Health Care System, Reno, NV, USA
- Sara M. Witcraft**, Department of Psychology, University of Mississippi, Oxford, MS, USA
- Stephen A. Wonderlich**, Neuropsychiatric Research Institute, Fargo, ND, USA
- Department of Psychiatry and Behavioral Science, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA
- E.R. Woodruff**, University of Colorado, Boulder, CO, USA
- Patrick W. Wright**, Neurology, Washington University in St. Louis, St. Louis, MO, USA
- Robert C. Wright**, Department of Psychology, University of California, Riverside, CA, USA
- Shawna Wright**, KU Center for Telemedicine & Telehealth, University of Kansas Medical Center, University of Kansas, Kansas City, KS, USA
- Tuppett M. Yates**, Department of Psychology, University of California, Riverside, Riverside, CA, USA
- Julie D. Yeterian**, Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), VA Boston Healthcare System, Boston, MA, USA
- Lok-Kin Yeung**, Columbia University Medical Center, New York, NY, USA
- Alex J. Zautra**, Psychology Department, Arizona State University, Tempe, AZ, USA
- Colin A. Zestcott**, Psychology Department, University of Arizona, Tucson, AZ, USA
- Peggy M. Zoccola**, Psychology Department, Ohio University, Athens, OH, USA

Foreword

Until the 1970s, there were no books, journals, or university courses on health psychology. Although the field's intellectual roots stretch back to the beginnings of psychology more than a century ago, its formal emergence depended on a convergence of influences (Friedman & Silver, 2007), including psychosomatic medicine, social-psychological and socio-anthropological perspectives on medicine, epidemiology, and medical and clinical psychology. Today, health psychology is a principal area of significant social science research and practice, with vital implications for the health and well-being of individuals and societies. Understanding the explosive trajectory of health psychology is useful to appreciating the strengths of the field and to approaching this new encyclopedia, the *Wiley Encyclopedia of Health Psychology*.

What is the nature of health? That is, what does it mean to be healthy? The way that this question is answered affects the behaviors, treatments, and resource allocations of individuals, families, health practitioners, governments, and societies. For example, if it is thought that you are healthy unless and until you contract a disease or suffer an injury, then attention and resources are primarily allocated toward “fixing” the problem through medications or surgical repairs. This is the traditional biomedical model of disease (sometimes called the “disease model”). Indeed, in the United States, the overwhelming allocation of attention and resources is to physicians (doing treatments) and to pharmaceuticals (prescription drugs and their development). In contrast, health psychology developed around a much broader and more interdisciplinary approach to health, one that is often termed the biopsychosocial model.

The biopsychosocial model (a term first formally proposed by George Engel in 1968) brings together core elements of staying healthy and recovering well from injury or disease (Stone et al., 1987). Each individual—due to a combination of biological influences and psychosocial experiences—is more or less likely to thrive. Some of this variation is due to genetics and early life development; some depends on the availability and appropriate applications of medical treatments; some involves nutrition and physical activity; some depends on preparations for, perceptions of, and reactions to life's challenges; and some involves exposure to or seeking out of healthier or unhealthier environments, both physically and socially. When presented in this way, it might seem obvious that health should certainly be viewed in this broader interdisciplinary way. However, by misdirecting its vast expenditures on health care, the United States gives its residents mediocre health at high cost (Kaplan, 2019). To approach these matters in a thorough manner, this encyclopedia includes four volumes, with Volume 1 focusing on the biological bases of health and health behavior, Volume 2 concentrating on the social bases, Volume 3 centering around the psychological and clinical aspects, and Volume 4 focused more broadly on crosscutting and applied matters.

Key parts of health depend on biological characteristics and how they interact with our experiences and environments. So, for example, in Volume 1, there are articles on injury to the brain, alcohol effects on the brain, nutrition, drug abuse, psychophysiology, and the tools and key findings of neuroscience. Note that even individuals with the same genes (identical twins) can and do have different health and recovery outcomes, and the articles delve into such complexities of health. Of course humans are also social creatures, and people's growth, development, and health behaviors take place in social contexts. So, in Volume 2, there are articles on such fundamental matters as social support, coping, spirituality, emotion, discrimination, communication, psychosocial stress, and bereavement.

Because our approaches to and conceptions of health are heavily influenced by society's institutions and structures revolving around medical care, much of health psychology derives from or intersects with clinical psychology and applied behavioral medicine. Volume 3 covers such clinical topics as psycho-oncology, depression, drug abuse, chronic disease, eating disorders, and the psychosocial aspects of coronary heart disease. Finally and importantly, there are a number of special and cross-cutting matters that are considered in Volume 4, ranging from relevant laws and regulations to telehealth and health disparities. Taken together, the articles triangulate on what it truly means to be healthy.

What are the most promising directions for the future that emerge from a broad and deep approach to health? That is, to where do these encyclopedia articles point? One key clue arises from a unique opportunity to enhance, extend, and analyze the classic Terman study of children who were followed and studied throughout their lives (Friedman & Martin, 2012). These studies revealed that there are lifelong trajectories to health, thriving, and longevity. Although anyone can encounter bad luck, a number of basic patterns emerged that are more likely to lead to good health. That is, for some individuals, certain earlier life characteristics and circumstances help propel them on *pathways* of healthier and healthier behaviors, reactions, relationships, and experiences, while others instead face a series of contingent stumbling blocks. There are multipart but nonrandom pathways across time linking personalities, health behaviors, social groups, education, work environments, and health and longevity. The present encyclopedia necessarily is a compendium of summaries of the relevant elements of health and thriving, but one that would and can profitably be used as a base to synthesize the long-term interdependent aspects of health.

In sum, this encyclopedia is distinctive in its explicit embrace of the biopsychosocial approach to health, not through lip service or hand-waving but rather through highly detailed and extensive consideration of the many dozens of topics crucial to this core interdisciplinary understanding.

References

- Friedman, H. S., & Martin, L. R. (2012). *The longevity project: Surprising discoveries for health and long life from the landmark eight-decade study*. New York: Penguin Plume Press.
- Friedman, H. S., & Silver, R. C. (Eds.) (2007). *Foundations of health psychology*. New York: Oxford University Press.
- Kaplan, R. M. (2019). *More than medicine: The broken promise of American health*. Cambridge, MA: Harvard University Press.
- Stone, G., Weiss, M., Matarazzo, J. D., Miller, N. E., Rodin, J., Belar, C. D., ... Singer, J. E. (Eds.) (1987). *Health psychology: A discipline and a profession*. University of Chicago Press.

Howard S. Friedman
University of California, Riverside
August 15, 2019

Preface

The field of health psychology is a specialty area that draws on how biology, psychology, behavior, and social factors influence health and illness. Despite the fact that the formal recognition of the field is only about a half of a century old, it has established itself as a major scientific and clinical discipline. The primary reason for this is that there have been a number of significant advances in psychological, medical, and physiological research that have changed the way we think about health, wellness, and illness. However, this is only the tip of the iceberg.

We have the field of health psychology to thank for much of the progress seen across our current healthcare system. Let me provide some examples that illustrate the diversity of these important contributions. Starting with a broad public health perspective, health psychologists have been involved in how our communities are planned and how urban development has a significant and direct impact on our health behaviors. That is, if we live close to where we work, play, and shop, we are more likely to walk or bike to our destination rather than drive or take public transportation. Focusing on a smaller, but no less important, source of data, health psychologists are involved in using information obtained from our genes to help counsel individuals to make good, well-informed health decisions that could have an impact on the individual immediately or in the future. Being well-informed can direct a person toward the best possible path toward wellness, whether it be measures aimed at prevention, further monitoring, or intervention.

Of course, there are many other contributions. For instance, data indicates that several of the leading causes of death in our society can be prevented or delayed (e.g., heart disease, respiratory disease, cerebrovascular disease, and diabetes) via active participation in psychological interventions. Knowing that we can improve health status by changing our behaviors seems like an easy “fix,” but we know that behavior change is tough. As such, it is not surprising that health psychologists have been involved in trying to improve upon treatment success by examining patient compliance. When we better understand what motivates and discourages people from engaging in treatment or pro-health behaviors, we can improve upon compliance and help individuals adopt more healthy lifestyles. Health psychologists have also played a role in shaping healthcare policy via identifying evidence-based treatments. This work has direct effects on how individuals receive healthcare as well as what treatments are available/reimbursable by insurance companies. Finally, there are also factors beyond medical care to consider (e.g., economic, educational) that can lead to differential health outcomes. Thus, health psychologists likewise examine ways to reduce health disparities, ensuring that the public and government officials are made aware of the impact of the social determinants of health.

I could go on and on, however, examples like this illustrate the significant impact this broad and exciting field has had (and will continue to have) on our understanding of health and wellness. Given the constantly changing nature of the field, it is not possible to be all inclusive; however, the aim of these four volumes is to provide readers an up-to-date overview of the field. Each entry is written to stand alone for those who wish to learn about a specific topic, and if the reader is left wanting more, suggested readings are provided to expand one's knowledge. Volume I, *Biological Bases of Health Behavior*, includes entries that cover topics in the broad areas of neuroscience and biopsychology relevant to health behavior. General topics include degenerative and developmental conditions, emerging methodologies available in clinical research, functional anatomy and imaging, and gene×environment interactions. Volume II, *Social Bases of Health Behavior*, addresses topics related to theories and concepts derived from social psychology. Specifically, topics related to the self, social cognition, social perception, attitudes and attitude change, perception, framing, and pro-health behaviors are included. Volume III, *Clinical Health Psychology and Behavioral Medicine*, covers the applied aspects of the field of health psychology including practical topics that clinical health psychologists face in the workplace, behavioral aspects of medical conditions, the impact of unhealthy behaviors, and issues related to the comorbidity of psychiatric disorders and chronic health concerns. Finally, Volume IV, *Special Issues in Health Psychology*, contains a wide array of topics that are worthy of special consideration in the field. Philosophical and conceptual issues are discussed, along with new approaches in delivering treatment and matters to consider when working with diverse and protected populations.

It is my sincere hope that the *Wiley Encyclopedia of Health Psychology* will serve as a comprehensive resource for academic and applied psychologists, other health care professionals interested in the relationship of psychological and physical well-being, and students across the health professions. I would very much like to thank and acknowledge all those who have made this work possible including Michelle McFadden, the Wiley editorial and production teams, the volume editors, and of course all of the authors who contributed their outstanding work.

Lee M. Cohen, PhD

Preface Volume 4: Special Issues in Health Psychology

Special issues, by definition, are an evolving target, especially in a topic area as fluid and diverse as health psychology. When we began this volume some three summers ago (June 2016), a cursory overview of peer-reviewed journal articles in health psychology journals reveals multiple topics: assessment techniques, education of providers, populations and subpopulations, new and evolving diseases, new treatments, environmental changes, systemic changes, and of course the repackaging of older, more established interventions to any of the preceding “special issues.” Where health psychology seeks to understand the role of behavior and perhaps cognition and behavior in the health of individuals, groups, nations, and humanity writ large, a volume of special issues could range from the molecular to the near universal.

In the present volume, we assembled 26 entries, each drafted by content experts in the special issue addressed. Every effort was made to ensure that each entry is evidence-based yet accessible to the most junior student of health psychology. The goal of this volume is to address at least a bit of each category foreseeably concerning health psychologists in the first two decades of the twenty-first century. To that end, we have three entries on assessment, two entries regarding education of providers, three entries on subpopulations, three entries on new or evolving diseases, six entries on treatments, and eight entries on environmental and systemic/cultural change. No doubt, as we go to press, newer and potentially more expansive knowledge will come to the fore, and each of our authors will wish that they had more time or more space to tell us about the latest information relevant to the health of humanity. To the wishful rewriters, I imagine that another opportunity will present itself.

In the meantime, I am indebted to the many authors that took time from busy schedules to draft the entries contained herein. Even for the fortunate few that write as easily as they breathe, these chapters are a labor of love. To synthesize one’s lifework and academic passion to a few cogent paragraphs of use to the novice and the expert alike is no small feat. I am also indebted to Ms. Alexia Maness, whose Herculean effort involved mastering multiple software changes while wrangling time zones and mixed messages across continents. Without her consistent good cheer, we would not have a volume for which to write a preface!

A resounding thank you to all.

Suzy Bird Gulliver

Editor-in-Chief Acknowledgments

I would like to publicly recognize and thank my family, Michelle, Ross, Rachel, and Becca for being supportive of my work life while also providing me with a family life beyond my wildest dreams. I love you all very much. I would also like to note my great appreciation and thanks to the faculty and staff of the Doctoral Training Program in Clinical Psychology at Oklahoma State University for providing me with an opportunity to expand my education and for providing extraordinary training. In particular, I would like to thank my late mentor Dr. Frank L. Collins Jr., Dr. Larry Mullins, Dr. John Chaney, and Patricia Diaz Alexander. Further, I am grateful to the University of Mississippi and Texas Tech University (and the colleagues and students I have had the privilege to work alongside) for affording me excellent working environments and the support to do the work I am honored to be a part of. Finally, I would like to recognize and thank the volume editors for their vision and perseverance to this project as well as to each of the contributors for their excellent entries and their dedication to this very important field.

Lee M. Cohen, Ph.D.
Editor-in-Chief

The Biopsychosocial Model

Robert J. Gatchel¹, Christopher T. Ray²,
Nancy Kishino³, and Athena Brindle¹

¹*Department of Psychology, College of Science, The University of Texas at Arlington,
Arlington, TX, USA*

²*College of Health Sciences, Texas Woman's University, Denton, TX, USA*

³*West Coast Spine Restoration, Riverside, CA, USA*

Overview

At the outset, it should be noted that the term *biopsychosocial model* was originally introduced by George Engel (1977, 1980) because he felt that the traditionally embraced biomedical reductionist model in medicine was outdated in light of the newer advances in medicine. He emphasized that modern medicine needed to encompass a more dynamic system approach, which included different levels of organization that included not only factors related to the patient and physician but also other important sociocultural and lifestyle variables in a “systems theory” manner initially introduced in biology by scientists such as Weiss (1969, 1977) and von Bertalanffy (1968, 1969). In terms of the biomedical model, Engel (1980) succinctly noted that: “...The crippling flaw of the model is that it does not include the patient and his attributes as a person, a human being. Yet in everyday works of the physician the prime object of study is a person, and many of the data necessary for hypothesis development and testing are gathered within the framework of an ongoing human relationship and appear in behavioral and psychological forms, namely, how the patient behaves and what he reports about himself and his life. The biomedical model can make provision neither for the person as a whole nor for data of a psychological or social nature, for the reductionism and mind-body dualism in which the model is predicated requires that these must first be reduced to physico-chemical terms before they have any meaning...” (p. 536).

It should also be noted that Minotti, Licciardone, Kearns, and Gatchel (2010) have indicated how osteopathic medicine actually preceded traditional allopathic medicine in embracing the biopsychosocial model approach to patient care. Indeed, one important tenet of osteopathic medicine is that a patient is composed of body, mind, and spirit, and one needs to

treat all three of components of the “whole person” in a holistic manner (Seffinger et al., 2011). Such a holistic approach involves the integration of physical structure, function, emotion, and social factors in order to achieve healing and health. “Knowing the whole person” is of paramount importance in the osteopathic approach, as it is in the biopsychosocial model. Thus, as will be reviewed in the next section, physical disorders (such as pain) are the result of the dynamic interaction among physiological (*bio*), psychological (*psycho*), and social (*social*) factors (i.e., *biopsychosocial*) that often perpetuate and may worsen the clinical symptom presentation of patients, as well as accounting for the individual differences across patients.

Unfortunately, this new *biopsychosocial model* conceptualization of illness was not immediately embraced by the general allopathic medical community. This may have been due to the fact that Engel was a psychiatrist, with psychiatry being at the “bottom of the ladder” in terms of medical specialties at that point in time. Fortunately, however, a synergy of events was occurring in the fields of pain medicine and behavioral medicine/health psychology that made the *biopsychosocial model* very attractive for many clinical researchers interested in the assessment of treatment of chronic pain. Below is a brief listing of how this synergy developed:

- Beecher (1959) highlighted the important role that conditioning played in patients’ experiences of pain and helped to further advances the idea that the biomedical model of pain was too limited.
- Melzack and Wall (1965) introduced the *gate control theory of pain*, which emphasized the important effects of emotion and cognitive factors in the experience of pain. Subsequently, Melzack and Casey (1968) extended the model further by introducing the *neuromatrix theory of pain*, which again emphasized the need to incorporate cognitive and motivational-affective components in order to better understand pain.
- Loeser (1982) proposed a general model of pain that included four dimensions: physical nociception, the subjective perception of pain due to the nociception, suffering or the emotional responses that are triggered by nociception or some other negative event associated with it (such as fear and anxiety), and pain behaviors, which are things that pain sufferers do, such as avoiding activities of daily living or work because of fear of reinjury or the further exacerbation of pain.
- The pioneering work by Fordyce and colleagues (Fordyce, Fowler, Lehmann, & DeLateur, 1968; Fordyce & Steger, 1979) successfully applied behavioral psychological principles (such as operant conditioning) to the treatment of chronic pain.

Ever since the above contributions to the study of pain, the *biopsychosocial model* has become the most heuristic approach to the assessment, treatment, and prevention of chronic pain (e.g., Gatchel, Peng, Peters, Fuchs & Turk, 2007; Turk & Monarch, 2002), as will be reviewed next.

The Biopsychosocial Model of Chronic Pain

Indeed, the acceptance of the *biopsychosocial model* is increasingly important in order to recognize the comorbidity of chronic pain and other persistent diseases. Fortunately, this model has consistently led to a better understanding of the considerably complex interactive process among components. In the context of pain, the *biopsychosocial model* approaches it as a consequence of the dynamic interaction among biological, psychosocial, and social components. This interaction perpetuates and may even worsen the clinical

presentation of pain. Many other clinical researchers have also emphasized the importance of this model (e.g., Feinberg & Brigham, 2013; Gatchel, McGeary, McGeary, & Lippe, 2014; Turk & Monarch, 2002). In addition, Gatchel (2004) examined the amplified measures of emotional suffering and psychopathology that disrupted effective treatment of patients with chronic pain. Furthermore, the degree of pain experienced by a patient depends on genetic factors, prior knowledge of pain, overall psychosocial well-being, and sociocultural variables (Gatchel et al., 2007). This resulting well-balanced *biopsychosocial model* has shown exceptional utility in chronic illness diagnosis and management. Unlike the framework of the biomedical model, the *biopsychosocial model* emphasizes the importance of the human experience of illness, in addition to the laboratory documentation that represents disease potential by providing a framework of a hierarchy that illustrates the interrelationships of a patient's psychosocial environment to their biological systems, as originally suggested by Engel (1980).

As noted above, the traditional biomedical reductionist model has been insufficient in capturing all of the aspects that are needed in order to appropriately diagnose and treat a patient living with chronic pain. Since the designation of 2001–2010 as being the “Decade of Pain Control and Research” by Congress, there have been other initiatives launched to address the growing problem of chronic pain. For example, chronic pain was highlighted as an important area for future research in the Institute of Medicine's report titled “Living Well and Chronic Illness: A Call for Action” (Institute of Medicine, 2012).

This was because chronic pain is becoming the most prevalent illness among Americans today. Nahin (2015) reported that 25.3 million Americans suffer from consistent daily pain, and 23.4 million Americans report “a lot of pain.” The Center for Disease Control and the National Center for Health Statistics (2015) also reported that 15.6% of Americans over the age of 18 reported experiencing a consistent severe headache or migraine, 29% reported consistent low back pain, and 14.9% reported consistent neck pain. In addition, pain accounts for approximately 80% of all doctors' visits, with medical treatment and lost productivity due to pain costing approximately \$625 billion annually (Gatchel, 2004; Institute of Medicine of the National Academy of Science, 2011).

Although, up until this point, we have been discussing the great utility of the *biopsychosocial model* for effectively evaluating and managing chronic pain, it is also a heuristic approach for dealing with chronic illnesses in general. This is because there is a close comorbidity of mental and physical disorders (Gatchel, 2004). Most chronic illnesses are accompanied by psychosocial factors (such as anxiety, depression, and anger), as well as cognitive distortions (such as fear avoidance, dysfunctional attitudes and beliefs, and distorted expectations). Fortunately, there are now psychometrically sound tests to measure these psychosocial factors (e.g., Gatchel, 2005). In addition, medical technology has advanced over the years to better evaluate possible biological or pathophysiological underpinnings of these illnesses (e.g., with the use of functional magnetic resonance imaging). However, despite the many improvements in technology and the better understanding of biopsychosocial processes, there are still future advances that will need to be made (e.g., Suls, Krantz, & Williams, 2013).

Beyond the Biopsychosocial Model

With growing research in neuroscience and genetics, the significance of a *diathesis–stress component* to the biopsychosocial model must be acknowledged. This new *diathesis–stress biopsychosocial model* places an additional emphasis that predispositional factors interact with

stressors such as health problems/illnesses. Once an illness is established, it becomes a stressor in its own right (Gatchel, 2005). Gatchel (2005) also pointed out that Melzack's (1999) *neuromatrix theory* of pain can be viewed as incorporating a *diathesis-stress model* component to it. Such a *diathesis-stress* component, originally promoted by Levi (1974), assumed that individuals are predisposed toward a specific disorder and manifest that disorder when negatively affected by stress. In other words, the degree/complexity of the interaction among biological, psychological, and social factors often depends on certain *diatheses* that people have when exposed to stressors such as an illness (especially if it is chronic in nature). For example, as illustrated in Figure 1, individuals “bring with them” certain diatheses or predispositions, such as certain candidate genes. In the case of chronic pain, examples of such candidate genes are neurotransmitter transporter, serotonin, solute carrier family (5-HTT), catechol-O-methyltransferase (COMT), interleukin 6 (IL-6), and μ -opioid receptor (OPRM). Also, for such candidate genes, the growing field of *epigenetics* has started to elucidate how these genes can be ultimately expressed as behavioral phenotypes (e.g., Belfer & Diatchenko, 2014; Biemont, 2010). Environmental factors, such as stress, can also alter their phenotypic expression and can have long-lasting effects on health and behavior (Hunter, 2012). One mechanism involved in the epigenetics process is the shortening of *telomeres* that help protect chromosomes. If they become too short, or are totally decimated by environmental stress factors, the chromosome becomes more vulnerable to possible mutation (Sibille, Witek-Janusek, Mathews, & Fillingim, 2012).

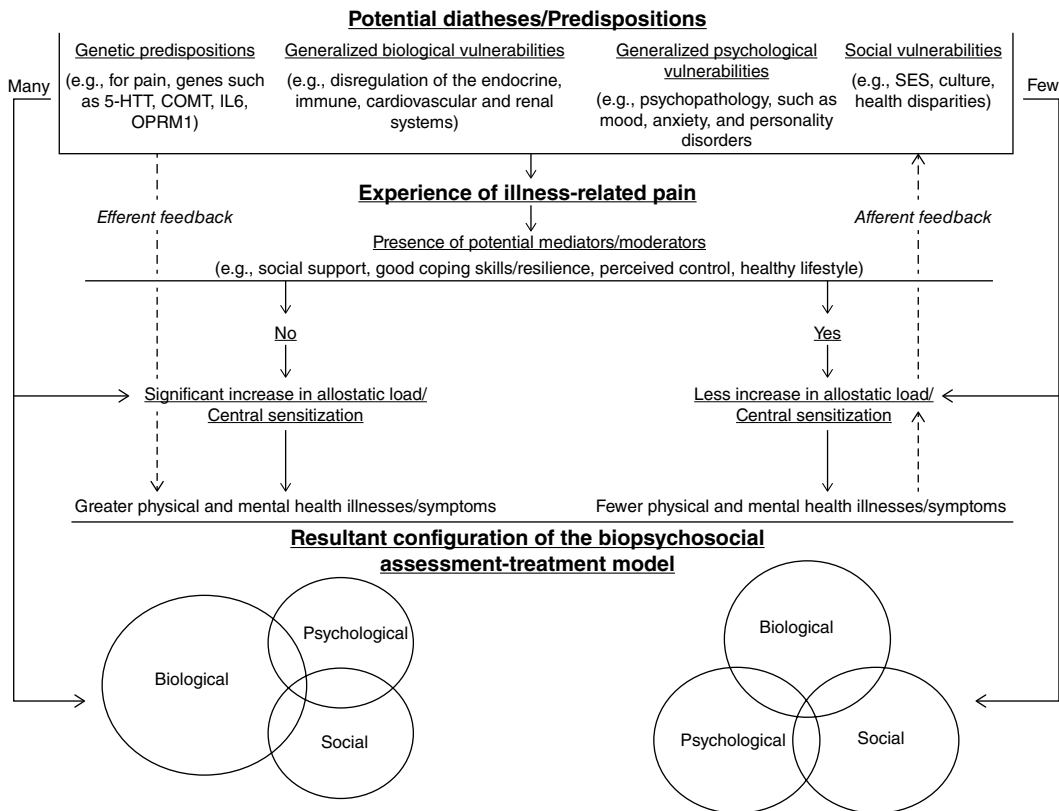


Figure 1 The diathesis-stress biopsychosocial model of chronic illness.

There are additional potential biological factors (e.g., dysregulation of the endocrine, immune, and cardiovascular systems), psychological factors (e.g., psychopathology, such as mood, anxiety, and personality disorders), and social vulnerabilities (e.g., SES, culture and health disparities) that may affect a person's ultimate experience of an illness. In turn, the probability of this illness to become chronic may be lessened by the presence of potential mediators/moderators (such as social support, coping skills, etc.). If mediators or moderators are absent, then they cannot "buffer" the individual from the full impact of the illness. In the event that pain is associated with the illness, *central sensitization* (CS) may result (a term introduced by Yunus, 2007 to refer to an abnormal and intensified experience of pain by mechanisms such as hyperexcitability in the central nervous system). This CS is postulated to underpin central sensitivity syndromes (CSSs) such as fibromyalgia, TMJ disorders, irritable bowel syndrome, and other persistent pain disorders (Adams & Turk, 2015; Mayer et al., 2012; Woolf, 2011; Yunus, 2000). In addition, the lack of effective mediators/moderators may lead to significant increase in the *allostatic load* of the patient (a term originally introduced by McEwen (2000)). Allostatic load refers to the significant decrease in body homeostasis due to the chronic exposure to the stress or trauma of an illness. Neuroendocrine-immune mechanisms, for example, can be negatively affected and result in greater "wear and tear of the body" (thus accelerating the development of diseases such as cardiovascular disorders, as well as psychoneuroendocrinology disorders).

The above will have implications for the mental and physical health status of the patient and may affect the resultant configuration of the biopsychosocial response pattern displayed. This type of configuration, in turn, will determine how complex the assessment–treatment process will need to be for each individual patient. The higher confluence of genetic and biological predispositions may necessitate greater attention to those components of the biopsychosocial interactions (see bottom left configuration in Figure 1). Without doubt, though, a comprehensive interdisciplinary assessment–treatment approach will need to be administered to patients.

Summary and Conclusions

The present chapter provided a comprehensive review of the heuristic value of the *biopsychosocial model* in better understanding health and illness. George Engel (1977, 1980) was the first to introduce the model because of his dissatisfaction of the traditionally embraced biomedical reductionist approach to illnesses that attempted to reduce all illnesses to only physiological/biochemical underpinnings, with the complete exclusion of potentially important psychosocial factors. Unfortunately, this new *biopsychosocial model* was not accepted in the general medical community. However, as we discussed, a synergy of events was occurring in the fields of pain medicine and behavioral medicine/health psychology that made this new model extremely attractive to clinical researchers involved in the assessment and management of chronic pain.

A more detailed discussion of the *biopsychosocial model* of chronic pain was next presented. In doing so, the epidemiology and high costs of this prevalent disorder was presented, as well as the various biopsychosocial components associated with it. It was also noted that the *biopsychosocial model* is generalizable to chronic illnesses in general. Additional advancements in the field, though, are still needed, especially with the development of new technologies that can more reliably evaluate the *bio* component of the model.

Finally, a *diathesis–stress biopsychosocial model* was introduced for an even better understanding of health and illness. The *diathesis–stress* component was originally proposed by Levi (1974). Levi suggested that individuals are predisposed toward specific disorders and will

manifest those disorders when negatively affected by stress. In the conceptual model we propose (see Figure 1), it is assumed that patients “bring with them” specific diatheses or predispositions, such as genetic factors, as well as potential biological and psychosocial vulnerabilities that may affect a patient’s ultimate expression of an illness. Obviously, the construct validity of this new model will need to be determined by future research efforts.

Author Biographies

Robert J. Gatchel—Robert J. Gatchel, PhD, ABPP, is a professor in the Department of Psychology, College of Science, at the University of Texas at Arlington and a clinical research program consultant at the Eugene McDermott Center for Pain Management at the University of Texas Southwestern Medical Center at Dallas. He is the director of the Center of Excellence for the Study of Health and Chronic Illness at the University of Texas at Arlington, as well as director of Biopsychosocial Research in Osteopathic Research Center at the University of North Texas Health Science Center.

Christopher T. Ray—Ray holds a doctorate degree in movement studies from the University of Georgia and earned both a master’s (in human performance and sport studies) and bachelor’s degree (in exercise science) from the University of Tennessee, Knoxville. His postdoctoral fellowship work was at the Veterans Administration Medical Center in Atlanta, which included a summer institute in epidemiology and biostatistics at the University of Washington. His research involves kinesiology and geriatric health.

Nancy Kishino—Nancy Kishino, OTR/L, CVE, is the director and founder of West Coast Spine Restoration Center. She is a licensed and registered occupational therapist.

Athena Brindle—Athena attended the University of Texas at Arlington and is now a candidate for a Doctor of Pharmacy at Texas Tech University Health Sciences Center.

References

- Adams, L. M., & Turk, D. C. (2015). Psychosocial factors and central sensitivity syndromes. *Current Rheumatology Reviews*, 11(2), 96–108.
- Beecher, H. K. (1959). *Measurement of subjective responses: Quantitative effects of drugs*. New York, NY: Oxford University Press.
- Belfer, I., & Diatchenko, L. (2014). *Pain genetics: Basic to translational science*. New York, NY: Wiley.
- Biemont, C. (2010). From genotype to phenotype. What do epigenetics and epigenomics tell us[quest]. *Heredity*, 105(1), 1–3.
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196(4286), 129–136.
- Engel, G. L. (1980). The clinical application of the biopsychosocial model. *The American Journal of Psychiatry*, 137(5), 535–544.
- Feinberg, S. D., & Brigham, C. R. (2013). Assessing disability in the pain patient. In T. R. Deer, M. S. Leong, A. Buvanendran, V. Gordin, P. S. Kim, S. J. Panchal, & A. L. Ray (Eds.), *Comprehensive treatment of chronic pain by medical, interventional, and integrative approaches: The American academy of pain medicine textbook on patient management*. New York, NY: Springer.
- Fordyce, W. E., Fowler, R. S., Lehmann, J. F., & DeLateur, B. J. (1968). Some implications of learning in problems of chronic pain. *Journal of Chronic Diseases*, 21, 179–190.
- Fordyce, W. E., & Steger, J. C. (1979). Chronic pain. In O. F. Pomerleau & J. P. Brady (Eds.), *Behavioral medicine: Theory and practice*. Baltimore, MD: Williams & Wilkins.

- Gatchel, R. J. (2004). Comorbidity of chronic mental and physical health disorders: The biopsychosocial perspective. *American Psychologist*, 59, 792–805.
- Gatchel, R. J. (2005). *Clinical essentials of pain management*. Washington, DC: American Psychological Association.
- Gatchel, R. J., & McGeary, D. (in press). Pain and health psychology. In C. S. Richards (Ed.), *Health psychology*. New York, NY: Wiley.
- Gatchel, R. J., McGeary, D. D., McGeary, C. A., & Lippe, B. (2014). Interdisciplinary chronic pain management: Past, present and the future. *American Psychologist, Special Issue on "Psychology and Chronic Pain"*, 69(2), 119–130.
- Gatchel, R. J., Peng, Y., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, 133, 581–624.
- Hunter, R. G. (2012). Epigenetic effects of stress and corticosteroids in the brain. *Frontiers in Cellular Neuroscience*, 6. doi:10.3389/fncel.2012.00018
- Institute of Medicine. (2012). *Living well with chronic illness: A call for public health action*. Washington, DC: The National Academies Press.
- Institute of Medicine of the National Academy of Science. (2011). *Relieving pain in America: A blueprint for transforming prevention, care, education, and research*. Washington, DC: Institute of Medicine.
- Levi, L. (1974). Psychosocial stress and disease: A conceptual model. In E. K. Gunderson & R. H. Rahe (Eds.), *Life stress and illness*. Springfield, IL: Charles C. Thomas.
- Loeser, J. D. (1982). Concepts of pain. In J. Stanton-Hicks & R. Boaz (Eds.), *Chronic low back pain*. New York, NY: Raven Press.
- Mayer, T. G., Neblett, R., Cohen, H., Howard, K. J., Choi, Y. H., Williams, M. J., ... Gatchel, R. J. (2012). The development and psychometric validation of the central sensitization inventory. *Pain Practice*, 12, 276–285.
- McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacology*, 22, 108–124.
- Melzack, R. (1999). Pain and stress: A new perspective. In R. J. Gatchel & D. C. Turk (Eds.), *Psychosocial factors in pain: Critical perspectives*. New York, NY: Guilford Publications, Inc.
- Melzack, R., & Casey, K. L. (1968). Sensory, motivational, and central control determinants of pain: A new conceptual model. In D. Kenshalo (Ed.), *The skin senses* (pp. 423–443). Springfield, IL: Thomas.
- Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science*, 50, 971–979.
- Minotti, D. E., Licciardone, J. C., Kearns, C., & Gatchel, R. J. (2010). The osteopathic medicine approach to pain management. *Practical Pain Management*, 10, 28–43.
- Nahin, R. L. (2015). Estimates of pain prevalence and severity in adults: United States, 2012. *The Journal of Pain*, 16(8), 769–780. doi:10.1016/j.jpain.2015.05.002
- National Center for Health Statistics. (2015). *Health, United States, 2014: With special feature on adults aged 55-64*. Hyattsville, MD: U.S. Government Printing Office.
- Seffinger, M. A., King, H. H., Ward, R. C., Jones, J. M., Rogers, F. J., & Patterson, M. M. (2011). Osteopathic philosophy. In A. G. Chila (Ed.), *Foundations of osteopathic medicine*. New York, NY: Lippincott Williams & Wilkins.
- Sibille, K. T., Witek-Janusek, L., Mathews, H. L., & Fillimgim, R. B. (2012). Telomeres and epigenetics: Potential relevance to chronic pain. *Pain*, 153(9), 1789–1793. doi:10.1016/j.pain.2012.06.003
- Suls, J., Krantz, D. S., & Williams, G. C. (2013). Three strategies for bridging different levels of analysis and embracing the biopsychosocial model. *Health Psychology*, 32(5), 597–601. doi:10.1037/a0031197
- Turk, D. C., & Monarch, E. S. (2002). Biopsychosocial perspective on chronic pain. In D. C. Turk & R. J. Gatchel (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (2nd ed.). New York, NY: Guilford.
- von Bertalanffy, L. (1969). Chance or law. In J. Smythies & A. Koestler (Eds.), *Beyond reductionism*. New York, NY: Macmillan Publishing Co.
- von Bertalanffy, L. V. (1968). *General systems theories: Foundations, development and applications*. New York, NY: G. Braziller.

- Weiss, P. (1969). The living system: Terminism stratified. In J. Smythies & A. Koestler (Eds.), *Beyond reductionism*. New York, NY: Macmillan Publishing Co.
- Weiss, P. (1977). The system of nature and the nature of systems: Empirical holism and practical reductionism harmonized. In K. E. Schaefer, H. Hensel, & R. Brody (Eds.), *Toward a man-centered medical science*. Mt. Kisco, NY: Futura Publishing Co.
- Woolf, C. J. (2011). Central sensitization: Implications for the diagnosis and treatment of pain. *Pain*, 152, 2–15.
- Yunus, M. B. (2000). Central sensitivity syndromes: A unified concept for fibromyalgia and other similar maladies. *Journal of Indian Rheumatism Association*, 8, 27–33.
- Yunus, M. B. (2007). Fibromyalgia and overlapping disorders: The unifying concept of central sensitivity syndromes. *Seminars in Arthritis and Rheumatism*, 36, 339–356.

Death and Dying

David E. Balk

*Department of Health and Nutrition Sciences, Brooklyn College of the City University of
New York, Brooklyn, NY, USA*

The dying trajectories and principal causes of death in the United States—and in other developed countries, for instance, Canada, Germany, and Japan—have changed dramatically over the past century. Whereas a typical dying trajectory in the early twentieth century was sudden and unexpected death due to a fatal, infectious disease or accident, the process of dying has become principally a lingering, drawn-out trajectory involving a series of chronic, debilitating conditions. The principal causes of death have become heart disease, malignant neoplasms, and circulatory problems.

A major factor that has produced these changes in dying trajectories and causes of death is the triumph of the biomedical model. Other influences have come from advances in the production and protection of food, success in insuring clean water, architectural and engineering triumphs (buildings with better heating and cooling, for instance), and public health campaigns educating communities about healthy and unhealthy behaviors.

The biomedical model became the framework organizing medical school curriculum and defining good medical practices. This model looks for biological and chemical reasons to explain disease. Disease is defined as physical deviations from established norms. The body is seen as a complicated, highly organized set of interrelated physical systems. The fundamental goal is to maintain life, and death is the enemy to resist at all costs.

The triumph of the biomedical model is no doubt due to its success in producing new knowledge and in overcoming previously fatal diseases. In consort with better hygiene and nutrition, the biomedical model helped increase longevity in the United States from an average of 47 years of age in 1910 to a lifespan exceeding 78 years today; whereas in the early twentieth century over 53% of all deaths in the United States occurred to persons 14 years of age and younger, the conquering of infectious diseases has made childhood mortality very uncommon.

As a conceptual framework the biomedical model became the paradigm for medical education and medical practice. The biomedical model has gained widespread cultural acceptance in developed countries. On the whole, most persons in developed countries in the

twenty-first century view life, health, disease, and death from within the assumptive world of the biomedical model.

A clear upshot of the success of the biomedical model is that American society now has large numbers of individuals living significantly beyond their 60s and developing the many infirmities endemic to aging bodies and minds. Concomitant with many progressive infirmities is the presence of functional limitation, such as difficulty walking, stooping, or swallowing. Of the nearly 328 million persons in the United States, 14% (nearly 46 million) have both chronic, debilitating conditions and functional limitations, and this 14% accrues over 56% of all health-care costs incurred in the United States. These figures will only increase as the aging population continues to grow. In many cases, these persons form a new societal phenomenon, the “frail elderly,” persons living into their 80s and 90s. In other eras these frail elderly would have died without the life-prolonging technologies used today. And what is of considerable concern is when life-prolonging technologies produce a continuation of life bereft of quality. Increasing apprehension has emerged over medical interventions to keep persons alive but helpless, incontinent, lacking dignity, connected to all sorts of instruments, and in pain.

Faced with the strong possibility that heroic efforts will be exerted to keep people alive despite their preference to die rather than remain helpless and very possibly in pain, various kinds of written directives have been devised to enable individuals to decline such efforts ahead of time. These written documents are termed advance directives.

An advance directive is a legal document that permits competent adults to state their preferences prior to a medical emergency or some other life-threatening situation. There are four forms of such directives: do not resuscitate orders (DNR), living wills, healthcare power of attorney (HCPOA), and physician orders for life-sustaining treatment (POLST).

DNRs are medical orders explicitly stating that the patient has rejected use of cardiopulmonary resuscitation should he (she) go into cardiac arrest. DNRs are written for medical staff to follow, are placed prominently in the patient’s chart, and denote the explicit preferences of the patient (or of the patient’s surrogate holding HCPOA).

Living wills provide the means for mentally competent adults to express preferences regarding the use of specified medical interventions should the person’s wishes be otherwise impossible to determine; an example would be if the person is in a coma. This type of advance directive is grounded in the right of mentally competent adults to refuse medical treatment. Living wills have gained legal status in each state of the United States and in the District of Columbia. However, specific provisions may differ from state to state regarding what a living will must contain to be legal.

Healthcare power of attorney, sometimes called durable power of attorney or healthcare proxy, offers an alternative to a living will. With HCPOA an individual empowers someone else to make healthcare decisions for the individual if the person is incapable to make such decisions.

POLST is an advance directive a person writes in collaboration with her (his) physician. The POLST makes individual preferences into actionable medical orders signed by the physician and the patient. Reviews of compliance indicate POLSTs are followed more consistently than living wills.

Attitudes toward use of advance directives vary considerably across different ethnic and racial groups in the United States, and these group differences underscore the communication challenges facing health psychologists working with persons from diverse backgrounds. When examining these group differences, it is prudent to recall that individual differences within a group can be as varied as differences between groups.

Advance directives are grounded in respect for the autonomy of the individual and in belief that promoting individual autonomy is a preferred value. Various ethnic groups place greater importance on familial support and guidance than on individual autonomy, and paternalistic decision making is the norm.

Advance care planning is observed more commonly in the United States among European Americans than among African, Hispanic, and Asian American groups. As examples, African Americans on the whole are much less likely than European Americans to complete advance directives. A history of discrimination from the majority White culture and documented cases of medical neglect as well as abuse (consider the infamous Tuskegee syphilis study) lead many African Americans to suspect that signing an advance directive gives the medical establishment a blank check to deny them treatment.

Researchers have studied Hispanic beliefs and intentions regarding end-of-life matters less than those of other ethnic minority groups. One common phenomenon that has emerged, however, is that the family holds a place of primacy among Hispanics and that the decisions of an elderly male possess prominence. While individual Hispanics have told researchers they would want care focused on relieving pain if seriously ill, they had not discussed these matters with family members. At the same time, despite never having voiced these views with their family, these respondents also indicated they wanted their families to be involved in end-of-life decision making. It is not difficult to see the challenges facing health psychologists to find ways to cut through the barriers presented by a hesitancy to discuss wishes and yet wanting family members to be involved in end-of-life decision making.

Asian Americans value saving face and regulating emotions, and this desire to avoid shame and to protect loved ones from shame may lead family members to insist that information about a terminal illness be kept from the patient. Research with groups of Asian patients suggests they are less likely than African American or Hispanic patients to want to learn the life expectancy facing them. The family, particularly a male figure, holds importance when critical decisions are to be made.

The realities that many persons die in a lingering, pain-filled trajectory filled with intrusive medical procedures—procedures that extend length of life but diminish quality of life—have inspired efforts to allow terminally ill persons to die a good death. The notion of a good death is a difficult concept with differing meanings contingent on the assumptive world(s) dominant in a society. In some societies, such as Japanese and Navajo, an underlying concern is whether the wrong kind of death will expose the living to vengeance from the deceased. In countries debating physician-assisted suicide, fundamentally opposed answers are given to whether consciously chosen self-termination to end unremitting pain offers a good death. In countries wedded to the biomedical model, ethicists and physicians are asking whether the healthcare system carries obligations to foster a good death when persons are nonresponsive to treatment and whether resources need to be rationed differently in end-of-life situations.

In twenty-first century Westernized secular societies a good death refers to the process of dying. The emphasis is on how a person's life ends, not on consequences in an afterlife. Emphasis is on several humanistic factors: to be free of distressing symptoms, to be free of pain, to be conscious, to be with loved ones, to be in familiar surroundings, and to be able to take care of oneself.

Consistent answers are given when persons are asked to identify their wishes regarding dying. They want to die free of pain, surrounded by loved ones, and preferably in familiar surroundings. An overwhelming majority of persons dying from a terminal illness, however, experience distressing symptoms such as nausea and vomiting; feel intense, unremitting pain,

helpless, and hopeless; are hooked up to machines; undergo invasive, painful procedures; and are surrounded by strangers in a medical or other institutional setting.

A counterforce to keeping terminally ill persons alive at all costs has been the hospice movement. Inspired by pioneering work in England, American hospice programs began in 1974 and now are present in every state. These programs take a holistic view of human existence. A holistic view is a thread connecting much contemporary thought about and work with persons facing the end of life.

Evaluation evidence has demonstrated noticeable family satisfaction with hospice vis-à-vis other forms of care for the dying. Further, evaluators have found significantly decreased costs for persons in hospice in contrast with matched controls; the decreased costs amounted to over \$2,300 a year. However, concerns have been raised over the projected costs to be incurred with the increasing numbers of frail elderly who will develop cancer or other debilitating terminal illnesses.

To receive federal funding, American hospice programs must admit persons only if they are diagnosed as having 6 months or less to live and if they forego curative treatment. Hospice efforts are exerted to prevent and manage all pain so that the individual and his (her) family may experience a good death. The practice and the policy are not to keep the terminally ill person alive at all costs.

The hospice movement has been instrumental in holding forth that a good death is possible. First and foremost, hospice understands that the dying person is primarily a living human being. Second, the patient in hospice care is the terminally ill person and the family. Further, hospice promotes quality of life by focusing on six fundamental dimensions of human existence: the physical, the behavioral, the emotional, the cognitive, the interpersonal, and the spiritual/existential. Uncontrollable, unrelenting, agonizing pain prevents persons from dying on their own terms, from dying with dignity, and from attending to the many aspects that make existence worthwhile.

Two useful frameworks on coping with a terminal illness have been proposed (Corr, 1992; Doka, 2013). One framework reviews various holistic tasks facing a dying person, the family, and caregivers; the other looks at changes that occur over time once a diagnosis of terminal illness has been learned.

The holistic tasks address physical, behavioral, cognitive, interpersonal, emotional, and spiritual dimensions of existence. Looking at a few of these tasks, consider that a terminally ill person may need help with such physical and behavioral tasks as washing, getting into and out of bed, using the toilet, swallowing, sleeping, and coping with the all-encompassing issue of excruciating pain that affects all dimensions of being alive. Chemotherapy may impair a person's cognitive capacities. Spiritual tasks center on the virtue of hope and on the viability of the person's assumptive world in the face of death from a terminal illness.

The second framework for coping entails responses of ill individuals, family members, and caregivers over time once a diagnosis is learned. Persons move from a pre-diagnostic phase through treatment and then the possibilities of remission, relapse, recovery, and dying. The holistic tasks mentioned above are carried out within these various phases once the diagnosis is learned.

Adopting a holistic perspective could be interpreted as an act of defiance in the face of the technique-driven biomedical model. Holism's roots in existential phenomenology place it in opposition to a positivistic approach to life and to the allure of techniques over mindfulness. Yet, such an interpretation would be incomplete: attending to the cares and the suffering of a terminally ill person and that person's family may well draw on methods and protocols inherent to best practices in biomedical science; a proper dosage of morphine may be exactly what the person needs. Awareness that humans have devised effective means to ameliorate physical

suffering can increase hope. Quality care, yet, also recognizes that the dying person is more than the sum of his (her) physical systems, death is not simply a medical phenomenon, and the person will not be reduced to biological or chemical systems that the biomedical model treats very well. The Institute of Medicine (IOM) took a forceful stand on the place of a good death in medical practice. The IOM wrote (1997, p. 5):

People should be able to expect and achieve a decent or good death – one that is free from avoidable distress and suffering for patients, families, and caregivers; (one that is) in general accord with patients' and families' wishes; and (one that is) reasonably consistent with clinical, cultural, and ethical standards.

Note the main topics in this IOM statement.

- 1 Achieving a good death is made a norm of medical practice.
- 2 There is explicit mention of preventing distress and suffering.
- 3 The stakeholders include more than the person who is dying. A good death will free patients, family members, and caregivers from distress and suffering.
- 4 The wishes of the patient and of the family will be followed.
- 5 Accepted clinical standards will be followed, as will cultural and ethical standards.

The IOM document was distributed shortly after publication of the SUPPORT study (1995), a very disappointing longitudinal intervention to improve end-of-life care at five teaching hospitals in the United States. The first year of the study focused on gathering data on staff understanding of patients' end-of-life preferences, medical decision making relevant to terminally ill persons, and medical interventions employed with terminally ill persons. The second year of the study provided each hospital a medical expert trained to consult with the medical teams on four objectives:

- 1 Staff would know terminally ill patients' end-of-life preferences.
- 2 Nurses and doctors would employ better pain control procedures.
- 3 Physician–patient communication would improve.
- 4 Outcomes would be patient oriented.

None of the objectives was achieved. An alarming finding from the SUPPORT study was that in nearly a third of cases physicians did not know their patients' DNR preferences and in at best 50% of the cases spouses were aware of these DNR preferences. It is not unreasonable to surmise that the 1997 IOM study, at least in part, was a direct response to the very disappointing outcomes regarding prospects for quality end-of-life care that the SUPPORT study had disclosed.

Looking back on the SUPPORT objectives, one can readily infer—based on the success of hospice—steps that may be taken within hospitals to accomplish what the SUPPORT intervention set out to do. Efforts to implement more circumscribed pain management systems have met with resistance in some US hospitals, so rosy-colored expectations of easy success to accomplish the SUPPORT objectives are naïve. However, given those qualifiers about resistance to change, there are some objectives to consider. As examples, in order for staff to know the end-of-life preferences of terminally ill persons, education about working with the dying needs to become holistic, not merely biomedical. Further, integrating hospital teams with

professionals alert to the social, emotional, and spiritual realities of dying persons would be a means to address the SUPPORT objective of attending to better pain procedures and of improving patient–staff communication.

The 1997 IOM report made several recommendations in order that a good death would become the norm of medical practice. Three of the recommendations focused on (a) expectations about excellent care at the end of life, (b) commitment from all healthcare professionals to gain and use the best practices and knowledge to care for the dying, and (c) education of health psychologists that would give importance to care for the dying. In 2014 the IOM reviewed their recommendations and concluded that gains had been made and gaps remain (Institute of Medicine, 2014):

- 1 Expectations about excellent care. Among gains have been noticeable increases in the percentage of terminally ill persons who received at least 3 days of hospice care; an ideal would be to increase involvement with hospice to at least 2 months given the evidence of the benefits to the terminally ill person and long-term benefits to the family following the death. In addition, numerous pilot programs implemented to demonstrate quality end-of-life care produced not only new care delivery models but also several pilot programs made hospice care accessible in rural areas previously underserved or not served at all.
Gaps identified include the lack of palliative care services in at least one-third of US hospitals. While palliative medicine has become a board-certified specialty, accreditation standards for hospitals and nursing homes in the United States do not require the provision of quality palliative care.
- 2 Commitment from all healthcare professionals to gain and use the best practices and knowledge for caring for the dying. The chief success reported is the significant increase in palliative care teams within hospital and medical centers. Between 2000 and 2012 the number of these teams increased dramatically from 600 to 1,600.
- 3 Gaps still needing attention include a continuing neglect in preventing and managing pain, particularly for patients nearing death. More attention needs to be paid to what healthcare professionals know about taking care of persons near the end of life.
Education of healthcare professionals must give importance to caring for the dying. Several training programs are now producing annually about 230 new physicians specializing in palliative medicine. Nursing programs are required to prepare students for end-of-life and palliative care situations. More than 2,000 individuals have been educated via the Education on Palliative and End-of-Life Care Project; graduates work to educate physicians and other healthcare professionals on providing quality palliative care.
Gaps still needing attention include the sparse attention given end-of-life care and virtual neglect of death and dying in medical school curricula. Further, of the 49 accredited schools of public health, only 3 have even one course on end-of-life policy. Analogous to the two IOM reports calling for radical changes in medical care at the end of life, a monograph recently appeared calling for a public health perspective on the end of life. The authors accept that quality care at the end of life is a public health imperative, and they focus on such issues as healthcare inequalities in this country and on the imperative to establish a public health agenda on policy insuring quality end-of-life care.

Seemingly in tandem with the IOM document on a good death, two nonprofit organizations in New York City sponsored a project to examine what is meant by a good death. These organizations were the Commonwealth Fund, a nonprofit interested in health policy, and the Nathan Cummings Foundation, which is devoted to social justice, democracy, and healthcare.

The authors of the Commonwealth–Cummings project (Emanuel & Emanuel, 1998) accept that multidimensionality marks a good death and that a holistic framework needs to be adopted to examine the multidimensional aspects of dying. Their holistic framework differs slightly with the framework used by hospice. The Commonwealth–Cummings framework focuses on six dimensions to human existence, namely, physical symptoms, psychological symptoms (hospice refers to emotional and cognitive), social relationships and support (hospice refers to the interpersonal dimension), economic demands and caregiving (not addressed in the hospice framework), spiritual/existential beliefs, and hopes and expectations (subsumed in the hospice framework under spiritual/existential).

Cicely Saunders, the founder of the modern hospice movement, coined the term “total pain,” an idea based on a holistic understanding of human experience and emphasizing distress in such existential dimensions as the physical, the cognitive, the interpersonal, and the spiritual and including refusal to accept one’s terminal condition. As seen directly above, the Commonwealth–Cummings project built on this seminal idea of holism and identified six dimensions that come into play when human pain is considered.

The Commonwealth–Cummings authors refer to “total pain” to depict the suffering that blocks a good death. Suffering is considered to consist of two situations: (a) poor conditions in all, or nearly all, of the six holistic dimensions or (b) an overwhelmingly bad experience in two of the dimensions.

The suffering in each dimension is examined:

- 1 Physical suffering has been studied extensively. By the end of the twentieth century, reliable, valid pain assessment tools were available. Physical suffering also includes such distressing symptoms as fatigue, insomnia, vomiting, and nausea. Pain management experts estimate that in 95% of cases, opioid or adjuvant drugs produce pain relief; however, these experts also estimate that pain remains poorly treated in 20–70% of dying patients. Further, around 5% of cases of physical pain do not respond to drug treatment, and other approaches are tried including acupuncture, hypnosis, and yoga.
- 2 Psychological symptoms include both cognitions and emotions. The symptoms may include depression, anxiety, and irritability. Some medical staff discount the importance of alleviating psychological symptoms because (a) the staff are not skilled in treating them and/or (b) the staff consider these psychological reactions a normal part of dying. One can see the importance of a multidisciplinary team, as in hospice, to ensure that at the end of life a person’s psychological suffering is attended to. Various evidence-based practices are available to assist in alleviating psychological symptoms.
- 3 Maintaining social relationships is a key adaptive task when coping with life crises. Maintaining links with others not only provides important outlets for emotions but also sources of information, solace, and acceptance. Much more work is needed to determine the kinds of social support dying patients rely on and whether support external to the family helps the person who is dying.
- 4 The reality of economic demands and caregiver needs at the end of life is a welcome addition to this holistic framework. There are sufficient research data to conclude that caring for a dying loved one imposes significant burdens on families. There can be staggering losses in income; the demands of the disease may induce physical and psychological fatigue. There is a link between severe physical and psychological pain in a patient at the end of life and increases in the caregiving needs that impose economic burdens that leave the family less capable of coping following the death. The concerns over caregiver burnout and complicated grief following the death are well documented. A note of hope has emerged

regarding caregiver needs when caring for a loved one at the end of life: empirical data indicate that early entrance to hospice—2 months or more prior to the death—proves beneficial for family members when later grieving the death.

- 5 Spiritual/existential beliefs form the category denoting how people find meaning, purpose, and value in their lives. Spirituality is integral to being human whether or not a person is religious. Hospice has recognized the central place of spirituality in the life of a dying person and in the lives of others left to grieve someone's death. Meaning making is at the core of prominent models to explain human responses to bereavement. Persons greatly at risk during times of crisis are those overwhelmed by the conclusion that nothing matters. It is difficult to overestimate the profound difficulty a person experiences when what gave her (his) life meaning has been disclosed to be without substance. One can see this scenario playing out when a death due to a terminal illness—for instance, a young child's—so challenges a parent's assumptive world that drifting into unrelenting, chronic grief becomes the only alternative.
- 6 Some scholars subsume hopes and expectations under the notion of spiritual/existential beliefs. Further, it is well understood that trauma from major life issues can so truncate a person's hopes and expectations that for all practical purposes the person has no future. For the Commonwealth–Cummings project authors, hope and expectations are a bit more prosaic than the points of view mentioned directly above. Persons at the end of life will remain alive until certain anticipated events have taken place; examples would be anniversaries, birthdays, graduations, and the return of a soldier from a war zone. A concern is that terminally ill patients tend to overestimate their life expectancy and therefore tend to put off advance care planning with family members and physicians.

Medical training should make achieving a good death the standard of care. Such training needs to start with making physicians and medical students comfortable with caring for persons who are dying and competent in caring for the terminally ill. This sort of curriculum objective would mean relaxing the intent of the biomedical model to keep terminally ill patients alive at all costs for as long as possible.

Medical training focused on thanatology could start by educating medical doctors how to talk to persons about dying, just as they are taught to talk with someone about an illness. Some aspects of talking to patients about dying would be how to deliver bad news, talking about spiritual concerns, and giving solace and consolation.

Around the same time as the Commonwealth–Cummings project, authors writing in *The Journal of the American Medical Association (JAMA)* gathered information from physicians, terminally ill patients, family members, and other stakeholders about achieving a good death. Karen Steinhauser et al. (2000) conducted a mixed-methods study using focus group interviews and a national survey to identify what people consider preferable at the end of life. The numerous focus groups were composed of physicians, nurses, hospice volunteers, chaplains, and terminally ill patients. The surveys were sent to a comparable list of individuals; out of 2,000 surveys mailed, 1,462 were returned, a phenomenal response rate of 73.1%.

Three main findings were reported:

- 1 The respondents overwhelmingly agreed on the general importance of being prepared for the end of life and of knowing one's family is prepared. Examples of such advance preparation included completing funeral arrangements and of having a sense when death was likely to occur. All respondents agreed that having time to prepare gives a greater chance for patients, family members, and medical caregivers to experience a good death.

- 2 Respondents wanted to know the treatment options for and the preferences of the dying person. Family members said it helped immensely to know what the dying person wanted to happen if he (she) were unconscious and unable to make wishes known. For instance, does the person want DNR orders carried out? There are obvious implications in this second finding for the functioning of healthcare proxies.
- 3 Dying persons wanted time to say goodbye to important persons in their lives. They wanted the chance to complete unfinished business. They wanted the opportunity to talk about the meaning of death with someone willing to listen attentively to their fears about dying. In this third set of findings, there were some stark differences between seriously ill persons and physicians. For instance, being mentally aware, at peace with God, and not a burden on one's family were endorsed by nearly all seriously ill persons, but by less than two-thirds of physicians. The differences in what patients and physicians consider attributes of a good death offer topics for medical education sensitizing physicians about end-of-life communication. Some of the differences may stem from patients' lack of experience with palliative care, such as the overwhelming majority wishing to be mentally aware up to the end; some end-stage cancer pain is so excruciating that only heavy dosages of morphine, which basically keep the person asleep, can alleviate the pain. It seems unlikely that physicians will consider it good use of their time to converse with the patient about being at peace with God or to spend time praying with patients. These topics are typically seen as the province of chaplains and thus the presence of ministers on hospice teams.

While there were some notable differences uncovered between patients and physicians over characteristics seen as important for a good death, another set of findings showed broad across-the-board agreement on the ranking of nine attributes linked to achieving a good death. In these findings, the three sets of respondents (patients, family members, and physicians) agreed on the importance of being free from pain, being at peace with God, and of the family being present at the end. Patients and family members placed more importance than physicians on being mentally aware. Physicians gave more importance than patients or family members to whether life was meaningful and whether conflicts had been resolved. Dying at home was ranked last by all the respondents.

Legalization of physician-assisted suicide has occurred in a handful of European countries (the Netherlands, Belgium, and Switzerland, in particular; it has been expressly rejected in Great Britain). Seen as serious issues not to be taken lightly, assisted suicide laws have been written with strict guidelines to govern against abuse; and these guidelines have set the template followed in other places. These guidelines specify age requirements and corroborated diagnoses by licensed physicians; requests must be given voluntarily over time and are not to be prompted by suggestions from the physician. The person must be judged to be acting free of any psychiatric or emotional duress. In order to ensure assisted suicides will not be forced by financial circumstances, Belgium makes palliative care available regardless of ability to pay.

Physician-assisted suicide was legalized in the state of Oregon in the late 1990s, and since then the states of Washington, California, Colorado, and Vermont have adopted protocols with guidelines quite similar to the ones specified in Oregon. In these states, a person must have a corroborated diagnosis of a terminal illness with less than 6 months to live, and the person must be able to ingest the lethal overdose on his (her) own. A New Mexico judge in 2014 ruled that assisted suicide was a matter between physicians and patients, but that ruling was overturned. The Montana Supreme Court ruled that nothing in that state's constitution prohibited assisted suicide. A voter referendum to adopt physician-assisted suicide was rejected in the state of Massachusetts.

The typical assumption is that a terminally ill person's primary motive for selecting assisted suicide is to avoid pain. While such a motive is present in many cases, other intents have been identified: fears about the future and fears of being a burden on others, loss of independence, lack of dignity, and decreasing ability to engage in activities making life meaningful. Typical life conditions people wish to end with assisted suicide include various forms of cancer, amyotrophic lateral sclerosis, early-onset Alzheimer's or some other dementia, and complete paralysis. Belgium and the Netherlands leave open the possibility for a person who is not terminally ill to make use of assisted suicide if in a permanently hopeless situation such as complete paralysis.

Cross-References

Biopsychosocial model; Caregivers and their role in health; Family and health; Older adult health; Palliative care; Quality of life.

Author Biography

David E. Balk was Director of Graduate Studies in Thanatology and Chair of the Department of Health and Nutrition Sciences at Brooklyn College, where he is now Professor Emeritus. Among his books is *Dealing with Dying, Death, and Grief during Adolescence* (2014, New York: Routledge).

References

- Corr, C. A. (1992). A task-based approach to coping with dying. *Omega*, 24, 81–94. <http://dx.doi.org/10.2190/CNNF-CX1P-BFXU-GGN4>
- Doka, K. J. (2013). Historical and contemporary perspectives on dying. In D. K. Meagher & D. E. Balk (Eds.), *Handbook of thanatology: The essential body of knowledge for the study of death, dying, and bereavement* (pp. 17–23). New York, NY: Routledge.
- Emanuel, E. J., & Emanuel, L. L. (1998). The promise of a good death. *The Lancet*, 351, 21–29. [https://doi.org/10.1016/S0140-6736\(98\)90329-4](https://doi.org/10.1016/S0140-6736(98)90329-4)
- Institute of Medicine. (1997). *Approaching death: Care at the end-of-life*. Washington, DC: National Academies Press.
- Institute of Medicine. (2014). *Dying in America: Improving quality and honoring individual preferences near the end-of-life*. Washington, DC: National Academies Press.
- Steinhauser, K. E., Christakis, N. A., Clipp, E. C., McNeilly, M., McIntyre, L., & Tulsky, J. A. (2000). Factors considered important at the end-of-life by patients, family, physicians, and other care providers. *Journal of the American Medical Association*, 284, 2476–2482. doi:10.1001/jama.284.19.2476
- SUPPORT Principal Investigators. (1995). A concentrated trial to improve care for seriously ill hospitalized patients. *Journal of the American Medical Association*, 274, 1591–1598.

Suggested Reading

- Balk, D. E. (2014). *Dealing with dying, death, and grief during adolescence*. New York, NY: Routledge.
- Benoliel, J. Q., & Degner, L. F. (1995). Institutional dying: A convergence of cultural values, technology, and social organization. In H. Wass & R. A. Neimeyer (Eds.), *Handbook of dying: Facing the facts* (2nd ed., pp. 117–141). Philadelphia, PA: Taylor & Francis.

- Callahan, D. (1995). *Setting limits: Medical goals in an aging society with "a response to my critics"*. Washington, DC: Georgetown University Press.
- Chapple, H. S. (2010). *No place for dying: Hospitals and the ideology of rescue*. Walnut Creek, CA: Left Coast Press.
- Clark, D. (2015). Hospice care of the dying. In J. M. Stillion & T. Attig (Eds.), *Death, dying and bereavement: Contemporary perspectives, institutions, and practices* (pp. 135–149). New York, NY: Springer Publishing.
- Cohen, J., & Deliens, L. (Eds.) (2012). *A public health perspective on end-of-life care*. New York, NY: Oxford University Press.
- Connor, S. R. (2009). *Hospice and palliative care: The essential guide* (2nd ed.). New York, NY: Routledge.
- Corr, C. A., & Corr, D. M. (2018). *Death and dying, life and living* (8th ed.). Belmont, CA: Wadsworth/Cengage Learning.
- Kastenbaum, R. J. (2011). *Death, society, and human experience* (11th ed.). Boston, MA: Pearson.
- Nuland, S. B. (1994). *How we die: Reflections on life's final chapter*. New York, NY: Knopf.
- Saunders, C., Baines, M., & Dunlop, R. (1995). *Living with dying: A guide for palliative care* (3rd ed.). New York, NY: Oxford University Press.

The Role of Regulation in Health Behavior (Listed in System as “Federal/State Regulation (FDA)”)

David B. Portnoy

US Food and Drug Administration, Silver Spring, MD, USA

Regulation Defined

Regulation includes those activities by a government that allows it to intervene in the private domain. Regulations, or implementation of laws, constitute a “binding legal norm...that intends to shape the conduct of individual and firms” (Orbach, 2013, p. 25). Thus, regulations can be defined as a series of rules established by a government to effect change among both individuals and firms in the private and public sectors. Whereas legislation establishes the authority for government actions and can be enforced by the courts, regulations refer to the much more specific rules developed by the governing body to implement those laws. Despite this distinction, many actions by governments, whether they occur through legislation or rule-making, are often considered part of regulations.

The commonality between these and other definitions of regulation is that they all identify the source of action as a government (or components thereof) and the recipient or intended target of those actions as individuals and organizations in the private sector. However, beyond that broad definition, the meaning of regulation is subject to much variability, the greatest source of which derives from the level of the action. Regulation can occur at federal, state/territory, county, and many other levels of government. Level-specific differences in purview, jurisdiction, and power result in different types of regulations; for example, the regulations issued by a city council may greatly differ from those issued by a federal government. However, this power disparity does not mean that regulations at different levels must be distinct in their focus or that federal regulations are always “stronger” than local ones. For example, federal agencies may not have the authority to regulate specifically local issues and vice versa, although federal regulations may sometimes preempt those at lower levels of government.

Regulation in Context

Ecological models of health behavior recognize that health behaviors are influenced not only by individual factors but also by the larger context in which those behaviors occur and in which the individual lives. These models are most effective when they are organized around a specific health behavior, have multiple levels, and recognize influences across these levels, that is, interventions at one level can affect other levels as well. However, certain frameworks seek to focus on multiple related risk factors concurrently that are intertwined at the community level. Although the specific levels may change from model to model, they generally start at the most proximal (individual) level, progress to the community level that can include workplaces or a place-based focus, and end with the outermost (i.e., national) level. That outermost level is sometimes referred to as public policy, policy environment, or community/policy. Regardless of the terminology, the most distal level of influence on health behavior is that of the law, public policy, and other large-scale factors that influence health through the other levels. Interventions at this level are intended to affect entire populations, as compared with interventions that focus solely on individuals or specific groups of individuals. Despite this, interventions at this level are by definition broader and thus must work in concert with local regulations, interventions at the individual level, and within the defined social and cultural context. One such review of tobacco control programs noted that comprehensive programs, such as those that include multiple levels of intervention, reduce tobacco use (Community Preventive Services Task Force, 2014). These types of models have received support and endorsement in guiding strategic plans for public health, such as the US Department of Health and Human Services' Healthy People 2020 and others.

Support for the Role of Regulation in Health

Regulation as a strategy to improve health, both at individual and population levels, has received wide support. The Institute of Medicine report on ensuring the health of the public in the twenty first century (2003) notes that federal, state, local, and tribal governments all play a role in identifying those in need of support and intervention and providing necessary services. The report notes that federal governments can intervene in six main areas, the first of which is policy making; decisions on policy optimally result from "...creation and use of an evidence base, informed by social values, so that public decision makers can shape legislation, regulations, and programs" (2003, p. 113). This emphasizes the need for strong regulatory science (as discussed below) to inform, support, and implement policy.

Dr. Thomas Frieden, director of the US Centers for Disease Prevention and Control (CDC) from 2009 to 2017, also notes the many ways in which regulation can contribute to improved public health outcomes. He identifies three main areas in which the government can protect public health and safety (Frieden, 2013). First, the government can require the provision of truthful information, such as nutrition information or calorie counts on menus, in order to empower individuals to make better health decisions; similarly, government-mandated warnings serve to inform the public about health risks and in certain cases to counter industry efforts to suppress or undermine science, such as the tobacco industry's history or disputing the science on the harms of smoking and of secondhand smoke (Samet & Burke, 2001). Second, the government can take action to prevent harm caused by others; examples include laws and regulations to reduce drunk driving, occupational hazards, and the inclusion of harmful ingredients in foods (e.g., recent local bans on trans fats and their removal from FDA's list

of foods that are “generally recognized as safe”). These specific examples represent regulation, some of which have been accompanied by industry self-regulation prior to enforcement as well as public education. The effectiveness of industry self-regulation varies widely from industry to industry, but self-regulation is often ineffective for public health benefit (Sharma, Teret, & Brownell, 2010). Such self-regulation is often an attempt forestall stronger and more enforceable local, state, and federal regulations. Finally, Dr. Frieden notes that the government can take actions to encourage healthy behaviors or create obstacles to harmful choices, such as zoning regulations that promote physical activity or that limit community density of retailers of harmful products such as alcohol or tobacco. He notes that a critical but often overlooked aspect of how people respond to such actions is “the degree to which individual actions are influenced by marketing, promotion, and other external factors,” a ripe area for research and intervention by health psychologists.

Perhaps the most sweeping regulation that supports changes in health behavior and subsequent population health is the Patient Protection and Affordable Care Act. This Act spawned a wide-ranging series of regulations, many of which focused on encouraging the use of preventive services (e.g., screening and immunizations), employer incentives for employee health promotion, nutrition labeling at chain restaurants, and other activities. In support of the Act, “the health of the individual is almost inseparable from the health of the larger community. And the health of each community and territory determines the overall health status of the Nation” (Koh, 2010, p. 1656).

Examples

A diverse and ever-growing list of health topics can be addressed through regulation. Many regulations focus on information, warnings, and public education about health risks and informed health decision making or promoting health promotion services. Two examples of this form of regulation are the provision of nutrition information on food products and (more recently) on restaurant menus and the provision of health warnings on tobacco products. As in most health-related regulation, the industry, rather than the target audience, is being regulated, but the focus of the impact is on the public.

Nutrition labeling began in the United States with a White House recommendation, which resulted in FDA issuing a final rule in 1973. These regulations were revised to allow health claims about foods and were again updated in the Nutrition Labeling and Education Act (NLEA) of 1990, which mandated nutritional labeling on most foods in the now-familiar nutrition facts panel (Institute of Medicine, 2010); an additional update occurred in 1993. At each step in this process, policy makers, scientists, and the public provided significant input regarding the benefits of such labeling as well as optimal labeling formats and content, which further illustrates that regulation is a constantly evolving process. More recent changes made include revising the format of the nutrition facts panel, changing the content of the nutrients, including “added sugars,” and other changes (US Food and Drug Administration, 2016, <https://www.federalregister.gov/documents/2016/12/30/2016-31597/food-labeling-nutrition-labeling-of-standard-menu-items-in-restaurants-and-similar-retail-food>). In recent years, many states and localities have begun to require calorie counts and other forms of nutrition information to be displayed on restaurant menus. In 2014, FDA issued a final rule requiring such information be listed on chain restaurant menus and menu boards, with the following stated purpose: “To help make nutrition information for these foods available to consumers in a direct, accessible, and consistent manner to enable consumers to make informed

and healthful dietary choices” (US Food and Drug Administration, 2014). Both menu and package labeling have been the subject of a wide variety of studies of the basic theory behind, potential impacts of, and best practices for labeling (e.g., Andrews, Burton, & Kees, 2011; Roberto & Kawachi, 2014).

The Family Smoking Prevention and Tobacco Control Act (FSPTCA) required that FDA publish a list of the harmful and potentially harmful constituents of tobacco and tobacco smoke “in a format that is understandable and not misleading to a lay person.” However, beyond requiring that FDA conduct periodic consumer research to ensure that the list is “understandable and not misleading,” Congress provided no further instructions as to the purpose of this list, unlike the rule on food menu labeling, which is explicit in its attempt to make nutrition information accessible for consumers. However, some intent regarding public health protection can be inferred given that the overall focus of the FSPTCA is to protect public health and that evidence that consumers were misled about the health impact of “light” and “low tar” cigarettes was cited in support of the law.

What Is Regulatory Science and How Can Health Psychology Contribute?

Regulations may be directly required or authorized by statute. In some cases, scientific support may be needed to help justify a particular regulation. This is especially true for regulations that seek to influence health through behavior. However, the science used to support regulation may differ from what is traditionally found in the published literature. For example, regulatory science focuses on “ensuring that scientifically valid techniques, tools, and models are available to evaluate products and, subsequently, to inform regulatory actions that promote optimal health outcomes” (Ashley, Backinger, van Bommel, & Neveleff, 2014, p. 1046). Thus, not all published literature may be specific enough to inform regulation. For example, if a new technique is published, it may move the science forward and may be innovative, but it is unlikely that a new and unvalidated tool would be used to support a regulation, assuming that other more accepted methods are available. Regulatory agencies generally rely upon scientific data to base their actions, as illustrated by the current public debate over the potential benefits or harms of electronic cigarettes.

Considerable efforts are being made to increase the regulatory science that is being conducted. The science used to support regulation may need to be more focused than typical scientific research and in general must focus on areas for which regulatory actions may be possible. For example, a study that examines the psychological mechanism underlying why youth post on social media about their use of tanning beds may be interesting, may advance scientific knowledge, and may inform the design of interventions. Although this type of study may inform the need for a regulation, it would be unlikely to directly inform the regulation itself. In other words, mechanistic or basic psychological research is critically important to advance the science, to allow for development of interventions, and for many other reasons; however it is unlikely to be useful in direct support of regulation unless it can be tied directly to a specific regulatory activity. It may, however, have relevance for advocacy and spur other research on the topic. For example, basic psychological research on judgment and decision biases, health literacy, and presentation format could be used to support regulations on restriction of potentially false or misleading claims made in an advertisement for a prescription drug (e.g., Aikin, O’Donoghue, Swasy, & Sullivan, 2011).

For health psychology research to be maximally useful in regulatory science, it should minimally fulfill the following criteria. First, the research should address a topic of interest to a regulatory agency; agencies typically announce topics of interest or solicit comments on agency websites, in notices in the *Federal Register*, or in similar official methods of public notice. Second, the research should be tailored to the appropriate study populations to ensure that study findings can be directly applied to the potential regulation. For example, a study conducted in women over age 65 may be less relevant if the regulation of interest focuses on women of reproductive age. Third, the stricter the sampling using, the more controlled the methodology and analytical controls, and ensuring a sufficient sample size will increase the likelihood that the research findings will be useful to help support regulatory actions and that conclusions will withstand challenges. In addition, reporting study details and results using one of the many reporting guidelines is helpful to ensure a full description of the study is provided. Complementing quantitative data, strong qualitative data can be useful to info to help explain “why” and “how” of certain issues. Fourth, interpretation of results should not extend beyond what the data portray; interpretations should not generalize beyond what is possible with the study design or suggest that cross-sectional associations are causal. All of these misinterpretations and statistical errors, which are common in published research in psychology and many other fields (e.g., Ioannidis, Munafo, Fusar-Poli, Nosek, & David, 2014), will weaken the potential usefulness of the study. Lastly, any conflicts of interest should be clearly disclosed. Research studies that adhere to these criteria are more likely to be used by an agency to inform regulation.

Researchers can provide study findings to regulators in a number of ways so that the agency may officially consider these findings in its rulemaking. Researchers may directly submit their research along with a lay explanation directly to local and state agencies, some of which may have processes for submission of public comments; other agencies may accept such findings without public notice. For federal regulation, public comment solicitations are generally announced in the *Federal Register*, and comments can be submitted online (www.regulations.gov). Public comments summarizing research findings can also be provided at in-person public meetings. Even without such outreach efforts by researchers, high-quality regulatory science research published in peer-reviewed journals is often referenced and used by agencies in designing and implementing regulations.

Summary

Regulations are actions taken by a government to implement laws. In the context of health, regulations can focus on individuals, industries, or larger organizations. Seen through the lens of ecological models of health behavior, regulations can influence health behavior changes through multiple pathways, interacting with interventions at the individual and community levels as well as state and local laws. Regulations related to health can be informed by strong and specific regulatory science.

Disclaimer

This publication represents the views of the author and does not represent FDA/CTP position or policy.

See Also

EHP0064	Psychosocial: Substance abuse: Tobacco
EHP0080	Health behavior change
EHP0133	Effects of health-related policies
EHP0167	Health behavior interventions
EHP0130	Cigarette smoking and cessation
EHP0156	Affordable Care Act ("Obama Care")

Author Biography

Dr. David B. Portnoy is a Social Science Branch Chief in the Office of Science at the US Food and Drug Administration Center for Tobacco Products. Dr. Portnoy received his MA and PhD in Social Psychology from the University of Connecticut, MPH from the Johns Hopkins Bloomberg School of Public Health, and postdoctoral training in cancer prevention and control at the National Cancer Institute. His research interests focus on risk perception and communication and the interplay between basic social psychological processes and health behaviors. At FDA, he conducts research on consumer perceptions of tobacco products and health warnings, reviews industry submissions, and works to develop the science base for regulations and health communication efforts. He has published on the topics of tobacco regulatory science, consumer perceptions of tobacco products, factors affecting tobacco use, and the effects of risk and worry on health behaviors.

References

- Aikin, K. J., O'Donoghue, A. C., Swasy, J. L., & Sullivan, H. W. (2011). Randomized trial of risk information formats in direct-to-consumer prescription drug advertisements. *Medical Decision Making*, 31(6), E23–E33.
- Andrews, J. C., Burton, S., & Kees, J. (2011). Is simpler always better? Consumer evaluations of front-of-package nutrition symbols. *Journal of Public Policy & Marketing*, 30(2), 175–190.
- Ashley, D. L., Backinger, C. L., van Bommel, D. M., & Neveleff, D. J. (2014). Tobacco regulatory science: Research to inform regulatory action at the Food and Drug Administration's Center for tobacco products. *Nicotine & Tobacco Research*, 16(8), 1045–1049.
- Community Preventive Services Task Force. (2014). Guide to Community Preventive Services. Reducing tobacco use and secondhand smoke exposure: comprehensive tobacco control programs. Retrieved from <https://www.thecommunityguide.org/findings/tobacco-use-and-secondhand-smoke-exposure-comprehensive-tobacco-control-programs>.
- Frieden, T. R. (2013). Government's role in protecting health and safety. *New England Journal of Medicine*, 368(20), 1857–1859.
- Institute of Medicine. (2003). *The future of the public's health in the 21st century*. Washington, DC: National Academy Press.
- Institute of Medicine. (2010). *Front-of-package nutrition rating systems and symbols: Phase I report*. Washington, DC: The National Academies Press.
- Ioannidis, J. P. A., Munafo, M. R., Fusar-Poli, P., Nosek, B. A., & David, S. P. (2014). Publication and other reporting biases in cognitive sciences: Detection, prevalence, and prevention. *Trends in Cognitive Sciences*, 18(5), 235–241.
- Koh, H. K. (2010). A 2020 vision for healthy people. *New England Journal of Medicine*, 362(18), 1653–1656.

- Orbach, B. (2013). *What is regulation?* In R. C. Clark (Ed.), *Why and How the state regulates, University Casebook Series*. New York, NY: Thompson Reuters/Foundation Press.
- Roberto, C. A., & Kawachi, I. (2014). Use of psychology and behavioral economics to promote healthy eating. *American Journal of Preventive Medicine*, 47(6), 832–837.
- Samet, J. M., & Burke, T. A. (2001). Turning science into junk: The tobacco industry and passive smoking. *American Journal of Public Health*, 91(11), 1742–1744.
- Sharma, L. L., Teret, S. P., & Brownell, K. D. (2010). The food industry and self-regulation: Standards to promote success and to avoid public health failures. *American Journal of Public Health*, 100(2), 240–246.
- US Food and Drug Administration. 2014. Food Labeling; Nutrition Labeling of Standard Menu Items in Restaurants and Similar Retail Food Establishments. edited by US Food and Drug Administration. 79 Federal Register.

Suggested Reading

- Frieden, Thomas R. (2013). Government's role in protecting health and safety. *New England Journal of Medicine* 368(20), 1857–1859.
- Orbach, Barak. (2013). *Regulation: How and why the state regulates*. Edited by Robert C. Clark, *University Casebook Series*. New York, NY: Thompson Reuters/Foundation Press.
- Sallis, James F, Neville Owen, and Edwin B Fisher. (2008). Ecological models of health behavior. In *Health behavior: Theory, research, and practice*, edited by Barbara K. Karen Glanz and K. Viswanath Rimer, 465–486. San Francisco, CA: John Wiley & Sons, Inc.
- Uchiyama, Mitsuru. 1995. Regulatory science. *PDA Journal of Pharmaceutical Science and Technology*, 49, 185–187.

Funding for Health Psychology Research

Robert T. Croyle

National Cancer Institute, Bethesda, MD, USA

Funders of Health Psychology Research

Both the public and private sectors provide funding for health psychology research. As the largest funder of health psychology research, the federal government plays an essential role in maintaining and enhancing the vitality of the field. Although several federal agencies provide funding, the largest source is the National Institutes of Health (NIH). Other agencies within the Department of Health and Human Services also play a significant role, including the Centers for Disease Control and Prevention and the Substance Abuse and Mental Health Services Administration. Because the latter two agencies are focused on support for program services, their research funding tends to focus on the evaluation of those services as well as public health surveillance to determine the scope and distribution of diseases and disease risk factors.

The NIH contains 27 institutes and centers. Because the governance structure delegates substantial autonomy and authority to the institutes, support for health psychology research can vary substantially. Institute directors are authorized to make final funding selections in most cases and therefore have a substantial impact on funding priorities and allocations. Health psychologists employed by the institutes can and have made a substantial contribution in ensuring that grant applicants in the field receive a fair review and appropriate consideration in funding decisions. Nevertheless, the selection of a new institute director can have a significant impact on that institute's support for health psychology.

In recent years, the NIH Office of the Director has played a larger role in funding and coordinating research across the agency. The NIH Common Fund, which is overseen by the NIH Director, supports trans-NIH initiatives, although individual grants are managed within the institutes.

In 1993, Congress established the Office of Behavioral and Social Sciences Research (OBSSR) at NIH. This action reflected the growing recognition of the role of behavior in health as well as the need for better coordination of related programs across the agency. Notably, four of the five directors of the office to date are health psychologists. From an operational perspective, this means that a health psychologist has been a member of the agency's leadership team ever since, and this has afforded the field as a whole with greater opportunities to shape the priorities of the agency.

Two other federal departments that have long histories of health psychology research funding are the Department of Defense and the Department of Veterans Affairs. Both departments also support clinician scientist training and have housed many of the most influential health psychologists in the United States.

The nonprofit sector has also played a key role in funding for health psychology research. The Robert Wood Johnson Foundation, the American Cancer Society, and the American Heart Association are among the many organizations that have provided support. Because these organizations operate outside of the constraints of the federal government bureaucracy, they can often rely on more nimble or rapid funding processes. Foundations have proven to be an essential source of support for applied and policy-related work, especially that focused on underserved and stigmatized populations. For example, in the areas of tobacco control and obesity, foundations supported much of the seminal work detailing the role of industry in product marketing and regulatory policy, including strategies for targeting ethnic minority populations. The Robert Wood Johnson Foundation has provided significant leadership in forging collaborations between scientists, health officials, and community organizations to facilitate the dissemination of evidence into clinical and public health practice.

The role of prominent individual philanthropists has been less significant in health psychology than in other fields. Large individual donations to universities and cancer centers, for example, have rarely targeted work focused on health behavior.

Clinical trials supported by industry have supported health psychologists in a wide variety of research domains but often in a secondary role. Not surprisingly, the scientific role of health psychologists in industry-supported drug trials has often focused on the issues of recruitment, adherence, and retention. As more trials incorporated measures of quality of life and other psychosocial constructs as secondary endpoints, opportunities for health psychologists have expanded. Industry-funded trials continue to provide important contributions to the research literature in a variety of behavioral medicine domains, such as smoking cessation, weight loss, diabetes and pain management, and cancer treatment symptom management. The contemporary interest in patient-reported outcomes and comorbidities among both industry and regulatory agencies such as the Food and Drug Administration are likely to further strengthen support for health psychology research within the context of clinical trials.

The Role of Special Initiatives

Special funding initiatives from both government and nonprofit sector organizations have played an important role in the development of health psychology. Although most have not been focused on health psychology per se, health psychology investigators have utilized a wide variety of special funding opportunity announcements to build the field, scale up research, and accelerate progress in research methods. The growth in funding for interdisciplinary research concerning

common health problems has enabled health psychologists to expand their participation on large-scale research consortia, clinical trials, centers of excellence, and cohort studies.

Several special initiatives supported by the NIH have been led and coordinated by the OBSSR. Initiatives especially relevant to health psychology include the Behavior Change Consortium (co-funded by the American Heart Association and the Robert Wood Johnson Foundation), launched in 1999, and a follow-on initiative, the Health Maintenance Consortium.

Individual or subsets of NIH institutes also have led important efforts. The National Cancer Institute (NCI), for example, funded a series of transdisciplinary team science research centers in which health psychologists played key roles as principal investigators, project leaders, and co-investigators. The first in the series, the Transdisciplinary Tobacco Use Research Centers, was co-funded by the National Institute on Drug Abuse, the Robert Wood Johnson Foundation, and the National Institute on Alcohol Abuse and Alcoholism. This initiative was followed by similar efforts that supported the Centers for Population Health and Health Disparities, the Centers of Excellence in Cancer Communication Research, and the Transdisciplinary Research on Energetics and Cancer Centers. More recently, the National Heart, Lung, and Blood Institute, in collaboration with NCI, the National Institute of Diabetes and Digestive and Kidney Diseases, the National Institute of Child Health and Human Development, and OBSSR, funded the Obesity-Related Behavioral Intervention Trials (ORBIT) initiative. ORBIT focused on the translation of findings from basic research on human behavior into more effective clinical, community, and population interventions to reduce obesity.

The NIH Common Fund has supported the Science of Behavior Change initiative, which focuses on the initiation, personalization, and maintenance of behavior change. The utilization of central NIH funds for this effort is a further reflection of the progress that health psychology has made within the NIH leadership community, given that Common Fund topic ideas compete with all areas of biomedical science. Interestingly, the growing interest in and recognition of the importance of behavioral risk factors has also influenced priority setting within mechanistically oriented biomedical research. The NIH recently launched a Common Fund initiative titled Molecular Transducers of Physical Activity in Humans.

At the NIH, high-priority initiatives are solicited via requests for applications (RFAs), published in the NIH Guide for Grants and Contracts, which are developed by program staff and reviewed by institute advisory boards prior to their release. Unlike Program Announcements, which also are used by institutes to communicate their research priorities to the scientific community, RFAs are associated with funds that are set aside specifically to support grants submitted in response to the announcement. Applications are reviewed by ad hoc panels that are established specifically for the initiative. Grants awarded using RFA-associated funds often are the basis for a research network or consortium. In many cases, the consortium is convened on a regular basis to enable work in progress to be shared among the funded investigators. This structure also allows the funder to monitor progress more closely and ensure that roadblocks are addressed in a timely fashion.

Two of the signature efforts led by the foundation sector are the Healthy Eating Research and Active Living Research initiatives, both supported by the Robert Wood Johnson Foundation. The foundation has also supported interdisciplinary population science research training efforts that have helped integrate health psychologists into other fields and settings. One such effort, the Health and Society Scholars Program, supported institutional postdoctoral fellowship grants, which were complemented with an annual national meeting of program participants. Given the individual- and group-level focus of traditional clinical health psychology

training programs, special initiatives such as this one have helped to strengthen the ability of health psychologists to participate fully in population-level research and programs.

Challenges and Opportunities in Health Psychology Research Funding

Support for health psychology research now seems firmly established among major research funders. The challenge to sustained support will lie in the productivity and impact of research supported to date and in the future. The growing visibility of health psychology research findings among policy makers and the public should facilitate ongoing support. Compelling findings from highly relevant research such as the Diabetes Prevention Program are perhaps the most essential ingredient for success. The growing importance of cost-effectiveness analyses needs to be recognized among investigators and funders alike.

Most funders do not target their support to specific disciplines like health psychology. Rather, funding tends to be focused on broad research questions, diseases, or public health problems. As a subdiscipline of behavioral science, health psychology is often painted with the same brush as other social and behavioral sciences. Therefore, it is not surprising that as funders like the NIH have expanded support for social and behavioral science, so too have they expanded support for health psychology research. Because so many of today's medical and public health challenges relate to behavior, broadly defined, it has been easier to make the case for support among the leaders of funding agencies and organizations. Within the political realm, however, the assignment of moral blame to victims of disease continues to be a challenge for advocates of health psychology research funding.

Another challenge to adequate research funding is the paucity of health psychologists in leadership positions within the key funding organizations. The relative status of psychology to medicine within academia and government is exacerbated by the underrepresentation of psychologists among leadership. Health psychologists are needed to serve in funding organizations and in advocacy roles before elected officials who allocate appropriations at the national and local levels.

Finally, the ability and willingness of health psychologists to participate in and contribute to large-scale transdisciplinary team science will predict to a large extent the trajectory of funding for health psychology research. Health psychology must be viewed as one of the essential disciplines to be included in broader, integrated efforts to advance knowledge and practice concerning the most significant medical and public health problems of our day. The ability to effectively communicate and integrate our work with other fields is therefore essential. Health psychologists also must embrace rigorous scientific standards, including objective self-criticism, to enable progress and demonstrate the value of funders' investments. As applicants for research funding, health psychologists are most successful when they recognize the limits of their expertise and methods and embrace collaboration with scientists from other disciplines. These are among the grant-writing skills that should be afforded to every young scientist in the field to maximize their likelihood of success.

See Also

Major Funders for Health Psychology Research:

- National Institutes of Health: www.NIH.gov
- Robert Wood Johnson Foundation: www.rwjf.org

Author Biography

Robert T. Croyle, PhD, was appointed director of the Division of Cancer Control and Population Sciences (DCCPS) at the National Cancer Institute (NCI) in July 2003. In this role, he is responsible for overseeing a research portfolio and operating budget of nearly half a billion dollars and serves on NCI's Scientific Program Leaders governance group. As a division, DCCPS covers a wide range of scientific domains and disciplines, including epidemiology, behavioral science, surveillance, cancer survivorship, and health services research. He previously served as the division's associate director for the Behavioral Research Program, leading its development and expansion. Before coming to NCI in 1998, he was professor of psychology and a member of the Huntsman Cancer Institute at the University of Utah in Salt Lake City. Prior to that, he was a visiting investigator at the Fred Hutchinson Cancer Research Center in Seattle, visiting assistant professor of psychology at the University of Washington, and assistant professor of psychology at Williams College in Massachusetts.

Dr. Croyle received his PhD in Social Psychology from Princeton University in 1985 and graduated Phi Beta Kappa with a BA in Psychology from the University of Washington in 1978. His research has examined how individuals process, evaluate, and respond to cancer risk information, including tests for inherited mutations in BRCA1 and BRCA2. His research has been published widely in professional journals in behavioral science, public health, and cancer, and he has edited two volumes: *Mental Representation in Health and Illness* (1991) and *Psychosocial Effects of Screening for Disease Prevention and Detection* (1995). He is co-editor of the *Handbook of Cancer Control and Behavioral Science* (2009) and co-author of *Making Data Talk: Communicating Public Health Data to The Public, Policy Makers, and The Press* (2009).

Genetics and Psychology (Health)

Nathan A. Kimbrel^{1,2,3} and Christopher Fink^{2,4}

¹*Department of Veterans Affairs' VISN 6 Mid-Atlantic MIRECC, Durham, NC, USA*

²*Durham Veterans Affairs Medical Center, Durham, NC, USA*

³*Psychiatry and Behavioral Sciences, Duke University School of Medicine, Durham, NC, USA*

⁴*North Carolina State University, Raleigh, NC, USA*

Overview

Improved understanding of the genetic basis of human disease and human behavior is rapidly changing the field of health psychology. Recent advances in genetic research and technology are permitting researchers to identify the genetic basis of a wide array of health conditions for the first time. Moreover, improved understanding of the field of epigenetics is rapidly shaping understanding of how environmental factors and behavioral changes can fundamentally alter gene expression. Genetic testing also has great potential to improve assessment, optimize the effectiveness of treatments, reduce side effects, and improve safety; however, it can also reveal vulnerabilities for disease that can be difficult for patients to cope with. As such, it is critical that health psychologists recognize the tremendous impact that genetic and epigenetic findings will likely have on their practice, as well as the field of health psychology more broadly, for the foreseeable future.

Central Dogma of Molecular Biology

All cellular processes begin with the genetic code written in every cell known as deoxyribonucleic acid (DNA), which is composed of four different nucleotides: adenine (A), cytosine (C), guanine (G), and thymine (T). Each is made of a phosphate group, a sugar known as 2'-deoxyribose, and a unique base that gives each nucleotide its identity. These bases pair with one another (A with T and G with C) to form DNA's well-known double

helix structure. These physical characteristics allow DNA to carry out its intended function, which is described by the central dogma of molecular biology. Simply put, the central dogma states that genes specify the sequence of messenger ribonucleic acid (mRNA) molecules, which, in turn, specify the sequence of proteins. Proteins perform a variety of functions in the cell such as regulatory functions, transport of small molecules, support, and catalysis of important reactions that would not normally occur under the conditions seen in a cell. DNA is transcribed to mRNA, the intermediate between DNA and proteins, by DNA-dependent RNA polymerases and other relevant proteins. RNA uses the same subunits as DNA, with slight modification of the sugar from deoxyribose to ribose, as well as using uracil (U) rather than thymine. Much of the RNA transcribed from DNA performs a host of various functions in the cell; however, a small selection of RNA, known as mRNA, is translated into proteins by structures known as ribosomes.

Epigenetics

There are many different levels at which gene expression can be controlled. One of the most heavily studied areas is that of epigenetics, which involve changes to the genome that do not affect the specific sequence of the nucleotides that comprise genes (Weinhold, 2006). One of the levels at which epigenetic marks can be seen is the level of histones and chromatin. The term chromatin describes the nucleoprotein complex that is seen when cells compact the 2–3 m of DNA present in a given cell into its nucleus. This packing begins with the DNA being wrapped around protein complexes known as histones. A segment of DNA that is 146 base pairs (bp) long is wrapped around the histone and is known as the core particle. By adding 20 bp and a linker histone known as H1 creates what is known as the chromatosome. Once the length of DNA is greater than 168 bp long, the structure is known as a nucleosome.

As one can imagine, navigating the chromatin can cause problems when the cell begins to transcribe genes. Histones placed over promoters, sequences on the DNA that allow transcription to initiate, can prevent the transcriptional machinery from recognizing the region and initiating transcription. In addition, histones placed throughout a gene can prevent the machinery from traveling the length of the DNA to read and transcribe the gene. Thus, one level at which regulation of gene expression can occur is at that of the histones. Modifications to the chromatin that cause a change in gene expression fit the definition of epigenetic marks because the changes are caused without changing the sequence of the genes affected. Two of the most common forms of epigenetic modification come in the form of histone acetylation and methylation, which involve the addition of two different small molecules (acetate and methane, respectively) to the lysine and arginine tails found on histones after the histone proteins are translated. These modifications can have significant effects on levels of gene expression of the DNA that interacts with modified histones with the general pattern of methylation decreasing gene expression and acetylation increasing it (Allfrey, Faulkner, & Mirsky, 1964).

DNA can also be methylated at cytosine residues. These methylated cytosines are most often found when they neighbor a guanine in what is known as a CpG dinucleotide (Rose & Klose, 2014). Though the specific nucleotides are being modified, the sequence of the nucleotide bases remains the same. Thus, DNA methylation can also be considered an epigenetic modification. As with histone methylation, increased levels of DNA methylation are associated with decreased levels of gene expression. As a result, DNA methylation also plays an important role in cell differentiation.

Genetic and Epigenetic Analysis Techniques

Researchers employ several different techniques to investigate what DNA looks like, the changes that are made to it, and the effects that it may impose on a person. Numerous research designs and techniques for studying the genetic and epigenetic basis of diseases exist, including candidate gene approaches, genome-wide association studies (GWAS), DNA methylation chips, and DNA sequencing. GWAS and candidate gene approaches are used to identify associations between genes and a disease of interest. Candidate gene studies are theoretically driven studies that aim to examine associations between diseases and specific genes that are hypothesized to potentially play a role in the pathogenesis of the disease under study (e.g., because of the known function of a gene). In contrast, GWAS is an atheoretical approach designed to identify novel genetic associations with a particular disease. This is accomplished by comparing individuals with and without the condition of interest on millions of different locations throughout their genome. While GWAS requires very large sample sizes, it has the potential of identifying novel associations that might not otherwise be identified.

Other genome- and epigenome-wide technologies frequently used by modern-day researchers include DNA sequencing and DNA methylation chips. These techniques enable researchers to quickly identify differences in the order of bases or functional groups that may be added on to DNA. For example, methylation chips are constructed with synthetic DNA designed to fluoresce when it binds to methylated cytosines in the sequence of DNA being examined. As a result, researchers are now able to quickly find the locations of methyl groups within a given sequence of DNA. Still other approaches enable researchers to study both the transcriptomes (i.e., all expressed mRNA molecules) and proteomes (i.e., all expressed proteins). Together, these and other techniques enable modern researchers to systematically study the genetic and epigenetic basis of a wide array of health conditions.

Health Psychology Issues Through a Molecular Genetics Lens

Smoking

Tobacco smoking is a global public health problem, as it represents one of the primary preventable risk factors for a wide array of health conditions, including respiratory diseases, cardiovascular diseases, and cancer (Gao, Jia, Breitling, & Brenner, 2015). Notably, tobacco initiation, regular tobacco use, and nicotine dependence all have a substantial genetic component, with heritability estimates of 75, 80, and 60%, respectively (Maes et al., 2004). More recently, a large number of studies have demonstrated that tobacco smoking is associated with substantial DNA methylation modifications throughout the genome (Gao et al., 2015). Indeed, a recent review on this topic found more than 1,000 smoking-associated CpG sites, the most common of which were cg05575921 (*AHRR*), cg03636183 (*F2RL3*), and cg19859270 (*GPR15*; Gao et al., 2015). There is also increasing awareness that smoking-associated DNA methylation changes may also be a key pathway through which tobacco smoking can lead to smoking-induced diseases (Breitling, 2013; Breitling, Salzmann, Rothenbacher, Burwinkelly, & Brenner, 2012). Epigenetic alterations are also known to play a role in cardiovascular disease. Moreover, methylation levels in *F2RL3*—one of the top three genes associated with smoking-associated methylation changes noted above—have also been associated with higher mortality among patients with stable coronary heart disease (Breitling, 2013; Breitling et al., 2012). Thus, it is possible that epigenetic alterations may be one of the

mechanisms through which tobacco smoking—an environmental risk factor with a known genetic component—may lead to increased risk for cardiovascular disease.

Diet

Diet is a behavioral factor that plays an important role in both obesity and cardiovascular disease; however, there is growing evidence that dietary changes, particularly during development, may also affect DNA methylation patterns through biochemical pathways. For example, several nutrients are required for the production of *S*-adenosylmethionine, which is the key methyl donor for DNA, RNA, proteins, and lipids (Glier, Green, & Devlin, 2014). Specifically, both vitamins (folate, riboflavin, vitamin B₁₂, vitamin B₆, choline) and amino acids (methionine, cysteine, serine, glycine) are needed to produce *S*-adenosylmethionine. As a result, imbalances in these nutrients have the potential to impact DNA methylation. While there is growing evidence that DNA changes in nutritional status can lead to methylation changes, this area of research is still in the early stages; however, it is hoped that improved understanding of the association between nutrition and DNA methylation could eventually lead to new biomarkers and new treatments aimed at reversing altered DNA methylation patterns associated with disease (Glier et al., 2014).

Understanding the Overlap Between Physical and Mental Health Conditions

The overlap between physical and mental health conditions has long been of interest to health psychologists. For example, posttraumatic stress disorder (PTSD)—a mental health disorder that has a clear environmental basis (i.e. a traumatic event)—has been linked to a wide range of cardiometabolic conditions, including heart disease, metabolic syndromic, and diabetes (Dedert, Calhoun, Watkins, Sherwood, & Beckham, 2010; Sumner et al., 2017). A variety of behavioral explanations have been posited to account for this association (e.g., poor diet, tobacco smoking, substance use, physical inactivity); however, it is likely that genetic factors also play a significant role in the pathogenesis of PTSD (Duncan et al., 2018). Notably, a novel study recently conducted by Sumner et al. (2017) has provided the first evidence of molecular genetic overlap between PTSD and coronary artery disease using a genome-wide design. This study utilized computational methods that allow researchers to estimate genetic correlations between complex phenotypes using GWAS summary statistics. Through this methodology, Sumner et al. (2017) were able to demonstrate that a variety of cardiometabolic traits were associated with each other. Moreover, they were also able to demonstrate for the first time that the molecular genetic basis of coronary artery disease is significantly correlated with the molecular genetic basis of PTSD ($r = .41$, $p = .01$).

Impact on Assessment and Treatment

Given that the field of genetics is rapidly advancing, it stands to reason that these advancements will continue to have major implications on healthcare for the foreseeable future. One such area will be the domain of personalized medicine, where we see can already see the emergence of this type of approach in relation to the treatment of breast cancer. Specifically, *BRCA1* and *BRCA2* are two genes that are known to be implicated in familial breast cancer. Identification of these genes and their cancer-causing mutations has begun to revolutionize the way in which cancer treatments and screenings are conducted. Already,

individuals who are identified as carrying genetic mutations associated with increased risk can undergo additional screenings and/or elect to have preventative mastectomies in order to reduce the risk of breast cancer. *BRCA* mutation type is also being used to guide treatment decisions in patients, as there is evidence that it may influence risk of relapse and that carriers may be particularly likely to benefit from specific treatment approaches (Li et al., 2016; Soenderstrup et al., 2018). Thus, genetic testing has great potential to improve assessment, optimize the effectiveness of treatments, reduce side effects, and improve safety; however, it can also reveal vulnerabilities for disease that can be difficult for patients to cope with. Since the advent of *BRCA1/2* genetic testing, model communication and counseling protocols have been developed to evaluate the behavioral and psychosocial effects of *BRCA1/2* genetic testing on patients (e.g., Botkin et al., 1996; Tercyak, O'Neill, Roter, & McBride, 2012). In general, studies on this topic have found that *BRCA1/2* genetic counseling and testing is not harmful for the majority of individuals who are found to carry risk variants, although individuals with high levels of preexisting anxiety do appear to respond more poorly (Tercyak et al., 2012). More recent interventions have begun to examine the role of patient knowledge, preferences, and values regarding genetic testing; however, it is clear that more work on this important topic is needed and that health psychologists are uniquely positioned to conduct such work (Tercyak et al., 2012).

Future Directions

It is hoped that advances in our understanding of the genetic and epigenetic basis of cancer and other diseases will eventually lead to a plethora of targeted, evidence-based behavioral health interventions for individuals who are identified as being at increased genetic risk; however, as noted by McBride, Birmingham and Kinney (2015), a significant challenge that health psychologists frequently encounter concerns how to effectively convey to patients the joint contributions of both genetic and environmental factors to disease risk. In addition, there is presently a great need for additional research aimed at (a) determining the optimal communication approaches for providers to employ to help their patients to effectively interpret genetic testing results, (b) identifying the most effective means of leveraging provider–patient communication about genetic testing to increase the patients' engagement in risk-reducing health behaviors (e.g., smoking cessation, weight management) and utilization of appropriate healthcare services, and (c) understanding how best to disseminate risk information to family members in order to further support patients' actions to reduce risk (McBride et al., 2015). Health psychologists are uniquely well suited to make significant contributions to each of these important and growing areas of science and practice (Tercyak et al., 2012).

Author Biographies

Dr. Nathan A. Kimbrel is the assistant director of the Genetics Research Laboratory at the Department of Veterans Affairs' Mid-Atlantic Mental Illness Research, Education, and Clinical Center (VA Mid-Atlantic MIRECC). He is also the assistant director for Implementation Science and Program Evaluation for the VA Mid-Atlantic MIRECC Clinical Core as well as an assistant professor in the Department of Psychiatry at the Duke University School of Medicine. His research primarily focuses on psychiatric genetics and the etiology, assessment, and

treatment of self-injurious behavior. Christopher Fink is a graduate student in the Department of Physiology at North Carolina State University in Raleigh, NC. He received a BS in Biology from the University of North Carolina in 2016 and has served as a clinical research assistant in the Traumatic Stress Laboratory at the Durham VA Medical Center and the VA Mid-Atlantic MIRECC since 2017.

References

- Allfrey, V. G., Faulkner, R., & Mirsky, A. E. (1964). Acetylation and methylation of histones and their possible role in the regulation of RNA synthesis. *Proceedings of the National Academy of Sciences of the United States of America*, 51, 786–794.
- Botkin, J. R., Croyle, R. T., Smith, K. R., Baty, B. J., Lerman, C., Goldgar, D. E., ... Nash, J. E. (1996). A model protocol for evaluating the behavioral and psychosocial effects of BRCA1 testing. *Journal of the National Cancer Institute*, 88, 872–882.
- Breitling, L. P. (2013). Current genetics and epigenetics of smoking/tobacco-related cardiovascular disease. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 33, 1468–1472. doi:10.1161/ATVBAHA.112.300157
- Breitling, L. P., Salzmann, K., Rothenbacher, D., Burwinkelly, B., & Brenner, H. (2012). Smoking, F2RL3 methylation, and prognosis in stable coronary heart disease. *European Heart Journal*, 33, 2841–2848. doi:10.1093/eurheartj/ehs091
- Dedert, E. A., Calhoun, P. S., Watkins, L. L., Sherwood, A., & Beckham, J. (2010). Posttraumatic stress disorder, cardiovascular, and metabolic disease: A review of the evidence. *Annals of Behavioral Medicine*, 39, 61–78.
- Duncan, L. E., Ratanatharathorn, A., Aiello, A. E., Almlí, L. M., Amstadter, A. B., Ashley-Koch, A. E., ... Koenen, K. C. (2018). Largest GWAS of PTSD ($N=20,070$) yields genetic overlap with schizophrenia and sex differences in heritability. *Molecular Psychiatry*, 23, 666–673. doi:10.1038/mp.2017.77
- Gao, X., Jia, M., Breitling, L. P., & Brenner, H. (2015). DNA methylation changes of whole blood cells in response to active smoking exposure in adults: A systematic review of DNA methylation studies. *Clinical Epigenetics*, 7, 113. doi:10.1186/s13148-015-0148-3
- Glier, M. B., Green, T. J., & Devlin, A. M. (2014). Methyl nutrients, DNA methylation, and cardiovascular disease. *Molecular Nutrition & Food Research*, 58, 172–182. doi:10.1002/mnfr.201200636
- Li, X., You, R., Wang, X., Liu, C., Xu, Z., Zhou, J., ... Zou, Q. (2016). Effectiveness of prophylactic surgeries in BRCA1 or BRCA2 mutation carriers: A meta-analysis and systematic review. *Clinical Cancer Research*, 22, 3971–3981. doi:10.1158/1078-0432.CCR-15-1465
- Maes, H. H., Sullivan, P. F., Bulik, C. M., Neale, M. C., Prescott, C. A., Eaves, L. J., & Kendler, K. S. (2004). A twin study of genetic and environmental influences on tobacco initiation, regular tobacco use and nicotine dependence. *Psychological Medicine*, 34, 1251–1261. doi:10.1017/S0033291704002405
- McBride, C. M., Birmingham, W. C., & Kinney, A. Y. (2015). Health psychology and translational genomic research. *American Psychologist*, 70, 91–104. doi:10.1037/a0036568
- Rose, N. R., & Klose, R. J. (2014). Understanding the relationship between DNA methylation and histone lysine methylation. *Biochimica Et Biophysica Acta (BBA) – Gene Regulatory Mechanisms*, 1839(12), 1362–1372. doi:10.1016/j.bbagr.2014.02.007
- Soenderstrup, I. M. H., Laenkholm, A. V., Jensen, M. B., Eriksen, J. O., Gerdes, A. M., Hansen, T. V. O., ... Ejlersen, B. (2018). Clinical and molecular characterization of BRCA-associated breast cancer: Results from the DBCG. *Acta Oncologica*, 57, 95–101. doi:10.1080/0284186X.2017.1398415
- Sumner, J. A., Duncan, L. E., Wolf, E. J., Amstadter, A. B., Baker, D. G., Beckham, J. C., ... Vermetten, E. (2017). Posttraumatic stress disorder has genetic overlap with cardiometabolic traits. *Psychological Medicine*, 47, 2036–2039. doi:10.1017/S0033291717000733

- Tercyak, K. P., O'Neill, S. C., Roter, D. L., & McBride, C. M. (2012). Bridging the communication divide: A role for health psychology in the genomic era. *Professional Psychology Research and Practice*, 43, 568–575. doi:10.1037/a0028971
- Weinhold, B. (2006). Epigenetics: The science of change. *Environmental Health Perspectives*, 114, A160–A167.

Suggested Reading

- Bierut, L. J., & Tyndale, R. F. (2018). Preparing the way: Exploiting genomic medicine to stop smoking. *Trends in Molecular Medicine*, 24(2), 187–196. doi:10.1016/j.molmed.2017.12.001
- Deary, I. J., Harris, S. E., & Hill, W. D. (2018). What genome-wide association studies reveal about the association between intelligence and physical health, illness, and mortality. *Current Opinion in Psychology*, 27, 6–12. doi:10.1016/j.copsyc.2018.07.005
- Pfeiffer, J. R., Mutesa, L., & Uddin, M. (2018). Traumatic stress epigenetics. *Current Behavioral Neuroscience Reports*, 5(1), 81–93. doi:10.1007/s40473-018-0143-z
- Rutter, M. (2006). *Genes and behavior: Nature-Nurture interplay explained*. Oxford, UK: Blackwell Publishing.

The Role of Cultural Mistrust in Health Disparities

Brandon L. Carlisle¹ and Carolyn B. Murray²

¹*Department of Psychiatry, University of California, San Diego, CA, USA*

²*Psychology, University of California, Riverside, CA, USA*

This entry examines the role *cultural mistrust* plays in health disparities experienced by African Americans, in contrast to Whites. While a multitude of individual and environmental factors contribute to trends in health outcomes, the focus of the present entry is the role of trust and its relationship with healthcare disparities, service utilization, adherence to treatment, and patient satisfaction. There are individual differences in the amount of trust potential patients have in the healthcare system, but it is also the case that variation can be partially explained by accounting for patient race/ethnicity. As a result, researchers have conceptualized mistrust as an important cultural variable when interpreting health trends among racial/ethnic minorities, particularly African Americans.

Health Disparities

Health inequality in the United States is a serious problem, and while there has been some improvement in particular areas, the overall picture is bleak. The health status of racial and ethnic minorities continues to lag far behind that of the majority population (Center for Disease Control and Prevention [CDC], 2013), especially for African Americans. In fact, African Americans have much higher disease and death rates than other racial and ethnic groups in this country (Kawachi, Daniels, & Robinson, 2005). The disparity of life expectancy across ethnic groups remains staggering. The gap between the highest and lowest life expectancies for race–county combinations in the United States is currently over 35 years (Murray et al., 2006). In the case of African Americans, disparities in mortality and life expectancy are predicted to increase (Levine et al., 2001). The overall mortality rate was 31% higher for African Americans than for White Americans. African Americans were 46% more likely to die

from a stroke, 32% more likely to die from heart disease, and 23% more likely to die from cancer (CDC, 2013). Even when receiving corrective medical surgery, African American patients are twice as likely as White patients to experience mortality (Nathan, Frederick, Choti, Schulick, & Pawlik, 2008).

Numerous studies have been conducted to identify the cause of health disparities between African Americans and Whites. These studies have identified individual and/or group factors such as obesity, low health literacy (Schwartz, Crossley-May, Vigneau, Brown, & Banerjee, 2003), and lower SES (Mandelblatt, Andrews, Kao, Wallace, & Kerner, 1996), as well as institutional factors, such as health insurance status (Bradley, Given, & Roberts, 2001), patient dumping (Randall, 1993), lack of a primary health provider, less qualified doctors (Bao, Fox, & Escarce, 2007), and other factors that, singly or combined, play a causal role in the creation and persistence of health disparities. However, after controlling for many of these factors, the health disparities across racial groups still remain.

The Role of Cultural Mistrust

The construct of *cultural* mistrust has been defined as a tendency of African Americans to distrust White people, based upon a legacy of direct or vicarious exposure to racism or unfair treatment (Benkert, Peters, Clark, & Keves-Foster, 2006). Terrell and Terrell (1981) first proposed *cultural mistrust* to describe the extent to which African Americans may distrust institutional systems in the United States that are dominated by the White majority; some of the relevant institutions include the education system, politics, business, and interpersonal contexts that may coincide with daily interactions. In addition to these domains, the health-care system has also been identified as an institution that can elicit cultural mistrust (Benkert et al., 2006).

Understanding why African Americans tend to report greater distrust of the healthcare system requires consideration of a specific historical context within the United States. Often, blame for the disproportionate mistrust of the healthcare system held by African Americans is attributed to the infamous Tuskegee syphilis study. This study, conducted by the US Public Health Service, involved the recruitment of hundreds of African American men in order to document the severity of syphilis and its symptoms when left untreated; the study continued long after penicillin was identified as an effective treatment. For many African Americans, the Tuskegee syphilis study serves as a vivid example of African Americans being deliberately victimized by an institution in the healthcare system (Kennedy, Mathis, & Woods, 2007). However, it is important to note that this case of gross mistreatment is “symbolic for the larger problem of African American distrust of the largely White medical establishment which has evolved in the presence of racial discrimination, racial inequalities in quality of care received, and a previous history of medical research misuse” (Shavers, Lynch, & Burmeister, 2000, p. 571).

Historically, those who now *mistrust the dominant group* (Whites) either were once held in captivity (African Americans were *legally* held in chattel slavery), were deprived of their rights to citizenship (Asian Americans), were exploited for their labor and cheated out of their lands (Mexican Americans), or endured genocide and were forced into concentration camps called reservations (Native Americans). While these were governmental policies and practices, often it was medical scientists and practitioners that legitimized and implemented these activities. Specifically, a substantial number of leading American scientists and medical doctors believed in eugenics, a practice they thought would improve the genetic quality of

mankind. The US government, along with the medical profession, embarked on two such programs—one known as positive eugenics and the other known as negative eugenics. Positive eugenic strategies involved urging individuals deemed particularly “fit” (Whites—in particular, Nordics and Aryans) to marry and procreate. Negative eugenics, on the other hand, was meant to actively limit, or control, the “genetically inferior” (e.g., those with low intelligence scores, those believed to be mentally deficient, criminal, and people of color—in particular, those of African American heritage). Negative eugenic practices included abortion, sterilization, and castration, among others. Segregation, incarceration, and terror were employed to keep the “genetically inferior” in their place, and physically limit their ability to increase in number, until such time as it would be acceptable to exterminate them.

By the 1950s, 24 states had anti-miscegenation laws that forbid Whites to marry non-Whites (in particular, African Americans). Additionally, in the United States, the government had compulsory sterilization laws; too often, women of color were sterilized without their knowledge. This strategy was used to control the “genetically inferior” populations, which included immigrants, people of color, poor people, unmarried mothers, the disabled, the criminal, the violent, and the mentally ill. As a matter of fact, government-funded sterilization programs existed in 32 states throughout much of the twentieth century. Driven by prejudiced attitudes, which fostered pseudoscience and social control, these programs were the driving force that fostered ill-conceived policies on immigration and segregation.

Today, if healthcare practitioners’ treatment of African Americans were to reflect a normal bell curve distribution, one would be led to presume that (a) the overwhelming majority of doctors do their job without consideration of race; (b) some doctors mistreat, or even purposefully harm, African Americans; and (c) some doctors make extreme efforts to make sure that African Americans receive treatment consistent with the “gold standard” of medical care (i.e., prescribe the best diagnostic tests or medical treatments available). Unfortunately, evidence indicates that the medical treatment of African Americans is far from a normal distribution but is instead skewed to the low end of treatment (Smedley, Stith, & Nelson, 2003). The evidence indicates that healthcare professionals exhibit the same levels of implicit bias toward African Americans as the wider population. Correlational evidence indicates that biases are likely to influence care in diagnosis, treatment decisions, and levels of medical care given in some circumstances, and these discrepancies need to be further investigated.

According to Dr. J. Corey Williams (2017), a psychiatrist, it is “no surprise that Blacks do not trust doctors or hospitals” (p. 2). He goes on to say that medical practice discrimination takes many forms, including differential prescription patterns such that African American patients, in contrast to Whites, are less likely to receive better-tolerated or more effective medications. African Americans are also more likely to receive an older treatment protocol, with reduced effectiveness and worse (i.e., more severe or deleterious) side effects, rather than the “gold standard” treatment, which has comparatively superior health effects and fewer or milder side effects. In addition, African Americans who go to emergency rooms often wait longer to see a doctor, are permitted less time spent with a doctor, and receive comparably poorer treatment (Williams, 2017).

This type of unfair and abhorrent treatment of African American patients has been noted on more than a few occasions. For instance, in 2003, the Institute of Medicine issued a report titled “Unequal Treatment” (Smedley et al., 2003) after reviewing over 100 studies concluding that African Americans do not receive the same quality of treatment as Whites, even when insurance status, patient income, and access to care were held constant. Specifically, African Americans received fewer referrals, more incorrect diagnoses, poorer provider–patient communication, and so on. One of the many egregious findings was the significantly

higher mortality rate from breast cancer seen in African American women. Despite the lower incidence of breast cancer in African American women and the higher rate of aggressive breast cancer in White women, African American women had a much higher mortality rate even when the number of doctor visits was held constant (Bradley et al., 2001). When searching for a reason for this anomaly, it became clear that physicians employed less aggressive and less appropriate treatment for African American women than for White women (National Cancer Institute, 2003). Discriminatory treatment of African Americans in health-care is well recognized, yet the literature to a certain extent “blames the victim,” namely, African American patients.

In addition to the influence of historical and present abuses, researchers have also investigated modern perceptions of African Americans in order to understand the role of cultural mistrust in health outcomes. In a series of focus groups among African American patients, themes related to medical mistrust emerged when patients reported feeling that their symptoms were discredited and clinicians did not convey respect during their communications (Cuevas, O’Brien, & Saha, 2016). Earlier research had likewise found that racial/ethnic minorities are significantly more likely to report being treated with disrespect during interactions with their healthcare provider (Blanchard & Lurie, 2004). Valid and reliable instruments that specifically measure cultural mistrust can facilitate investigations designed to assess the perceptions of African Americans. Furthermore, tailored measurement can contribute to the identification of correlates and predictors of important health attitudes, behaviors, and, ultimately, health outcomes.

Measuring Cultural Mistrust

The most common measure of cultural mistrust is the Cultural Mistrust Inventory (CMI), which was developed by Terrell and Terrell (1981). The CMI is a 48-item questionnaire assessing the extent to which African American people mistrust White people in societal domains of education (e.g., “If a black student tries, he will get the grade he deserves from a White teacher”), business (e.g., “White storeowners, salesmen, and other White businessmen tend to cheat Blacks whenever they can”), politics and law (e.g., “White politicians will promise Blacks a lot but deliver little”), and interpersonal relations (e.g. “Whites will say one thing and do another”).

Researchers have administered the questionnaire in order to determine if responses are significantly associated with attitudes (e.g., perceived discrimination) and behaviors (e.g., treatment adherence) in the healthcare setting. Despite the well-documented utility of the CMI (Whaley, 2001), the questionnaire does not specifically assess mistrust of the healthcare system; this creates a potential limitation when extending research on cultural mistrust to healthcare settings. Decades after Terrell and Terrell (1981), the Group-Based Medical Mistrust Scale (GBMMS) (Thompson, Valdimarsdottir, Winkel, Jandorf, & Redd, 2004) was developed in order to specifically measure the extent to which respondents exhibit race-based medical mistrust. Thompson et al. argued that an instrument specifically designed to measure mistrust in healthcare settings was warranted, because attitudes and behaviors in healthcare settings may present a unique area of concern given the legacy of medical disparities and abuses experienced by racial/ethnic minorities.

The GBMMS is a 12-item measure that assesses suspicion of mainstream healthcare systems (e.g., “People of my ethnic group should be suspicious of modern medicine”), suspicion of

healthcare professionals (e.g., “People of my ethnic group cannot trust doctors and healthcare workers”), and perceptions about the treatment provided to individuals that identify with the respondent’s racial/ethnic group (e.g., “I have personally been treated poorly or unfairly by doctors or healthcare workers because of my ethnicity”). Previous research has produced evidence demonstrating the validity of the GBMMS, and the measure has been incorporated into several investigations of health attitudes and behaviors exhibited by African Americans (Shelton et al., 2010).

Perception, Utilization, and Adherence

The importance of patient attitudes has been emphasized in research on the perceptions of healthcare providers and patient satisfaction (Hall & Dornan, 1988). Furthermore, researchers have also measured patient attitudes in relation to perceptions about help-seeking and perceived discrimination. Evidence suggests that cultural mistrust can partially explain attitudes reported by African Americans in healthcare settings. In a study of men diagnosed with prostate cancer, African Americans reported greater medical mistrust than Whites, and medical mistrust was negatively associated with physical and emotional well-being (Bustillo et al., 2017). Benkert et al. (2006) collected responses from low-income African Americans across two primary care clinics, and they found that CMI scores were negatively correlated with trust in a patient’s primary healthcare provider. Furthermore, CMI scores were also negatively correlated with indicators of patient satisfaction with healthcare.

Previous research has also revealed racial and ethnic differences in use of healthcare services (i.e., frequency, duration; Weinick, Zuvekas, & Cohen, 2000). Consideration of cultural mistrust can improve understanding of behaviors exhibited by African Americans in the healthcare setting. In a study of preventive care utilization among African American women, greater cultural mistrust predicted a significantly lower likelihood of receiving a physical exam in the previous year (Pullen, Perry, & Oser, 2014). Thompson et al. (2004) found that African American and Latina women who reported that their last breast cancer screening was more than 5 years ago, or who reported no previous breast cancer screening, had significantly higher GBMMS scores. In a related study of preventive health behaviors, Shelton et al. (2010) found that higher scores on the GBMMS were negatively associated with favorable attitudes about prostate cancer screening among African American men. A similar pattern emerged in a systematic review of quantitative and qualitative research on cultural mistrust and colorectal screening, wherein African Americans with higher mistrust scores reported lower rates of receiving colorectal screening (Adams, Richmond, Corbie-Smith, & Powell, 2017).

The importance of patients’ adherence to treatment, and its relationship to positive health outcomes, is well documented in the literature. Furthermore, poor adherence to medical treatment can contribute to racial/ethnic disparities in health outcomes (McQuaid & Landier, 2018). The influence of cultural mistrust is significant because it can potentially impact a patient’s decision to adhere to their medical treatment or follow the recommendations of their physician. For example, in a study of self-care among African Americans diagnosed with type 2 diabetes, greater cultural mistrust was associated with lower adherence to dietary interventions (deGroot et al., 2003). In conclusion, mistrust of the medical profession has been shown to be a major obstacle to African Americans seeking health treatment and properly adhering to health regimens.

Promoting Trust

Cultural mistrust has a deleterious effect on the quality of the relationship between African Americans patients and their physicians. In order to improve the quality and content of their interactions with patients, physicians and medical staff need to understand that cultural mistrust is primarily derived from suspicions at an organizational level, and they must recognize that this mistrust among African Americans is an adaptive defensive strategy stemming both from historical abuses and modern disparities. Cultural competency initiatives have been implemented in systems of physical and mental healthcare. One approach to enhance cultural competency, and to improve patient interactions, is to increase cultural sensitivity and awareness of cultural mistrust so that it can be directly addressed.

Trust can also be fostered through the training and recruitment of racial/ethnic minority care providers. Some African Americans may feel more comfortable in healthcare settings when their provider is African American as well. Doctor–patient race concordance has been associated with an increased likelihood of utilizing health services when needed and a decreased likelihood of delayed help-seeking (LaVeist, Nuru-Jeter, & Jones, 2003). While race concordance does not always produce significant differences in health behaviors, the improved representation of African Americans in healthcare roles can partially address concerns related to cultural mistrust. Based on the existence of several directories that have compiled lists of African American care providers, there appears to be a strong interest in race concordance within segments of the African American population.

Improving trust among African Americans could also involve partnering with the Black church. Among African Americans with strong ties to their community church, disseminating health information in an environment where they feel more comfortable can foster trust. Health campaigns that aim to raise awareness and increase health service utilization can improve outcomes by recognizing the importance of pastors and by allowing them to advise and also play a significant role in the promotion of health behaviors that can potentially mitigate disparities.

Conclusion

Lacking trust in the healthcare system and its providers can influence both service utilization and treatment adherence and thereby influence health outcomes for African Americans. Additionally, differential treatment and disparate outcomes experienced by African Americans could confirm their potential suspicions and consequently exacerbate existing tendencies to lack trust in the healthcare system. Therefore cultural mistrust of healthcare can serve as a predictor of attitudes and behaviors among African Americans, or it can be conceptualized as an outcome that stems from perceived or actual medical mistreatment. These perspectives underscore the importance of examining cultural mistrust and how it may relate to African American health trends. Improving trust among African Americans requires sustained effort on several fronts. Healthcare organizations, providers of care, researchers, and community advocates should give careful consideration to the historical and modern experiences of African Americans in order to inform the development and implementation of interventions. Establishing (or earning) the trust of African Americans will require various methods at individual, situational, and organizational levels, because “just as there are many reasons for the mistrust, there is no one answer that will solve all of the mistrust issues” (Kennedy et al., 2007, p. 60).

Author Biographies

Brandon L. Carlisle, PhD, is a senior mental health researcher in the Department of Psychiatry at the University of California, San Diego. He contributes to research focusing on the evaluation and improvement of behavioral health services provided to children, youth, and families. His research interests include identifying barriers to seeking and receiving care and the importance of cultural competence when providing care to diverse populations.

Carolyn B. Bennett Murray, PhD, is currently a full professor in the Psychology Department at the University of California, Riverside (UCR). She received her PhD from the University of Michigan, Ann Arbor, and has published numerous journal articles and book chapters. Her primary research interest is self-sabotage. A few of her many awards include the Chancellor's Award for Excellence in Undergraduate Research, the Association of Black Psychologists' Distinguished Psychologist Award, and the UCR Distinguished Teaching Award.

References

- Adams, L. B., Richmond, J., Corbie-Smith, G., & Powell, W. (2017). Medical mistrust and colorectal cancer screening among African Americans. *Journal of Community Health, 42*, 1044–1061.
- Bao, Y., Fox, S., & Escarce, J. (2007). Socioeconomic and racial/ethnic differences in the discussion of cancer screening: “Between-” versus “within-” physician differences. *Health Services Research, 42*(3 Pt. 1), 950–970.
- Benkert, R., Peters, R. M., Clark, R., & Keves-Foster, K. (2006). Effects of perceived racism, cultural mistrust and trust in providers on satisfaction with care. *Journal of the National Medical Association, 98*, 1532–1540.
- Blanchard, J., & Lurie, N. (2004). R-E-S-P-E-C-T: Patient reports of disrespect in the health care setting and its impact on care. *Journal of Family Practice, 53*, 721.
- Bradley, C., Given, C., & Roberts, C. (2001). Disparities in cancer diagnosis and survival. *Cancer, 91*, 178–188.
- Bustillo, N. E., McGinty, H. L., Dahn, J. R., Yanez, B., Antoni, M. H., Kava, B. R., & Penedo, F. J. (2017). Fatalism, medical mistrust, and pretreatment health-related quality of life in ethnically diverse prostate cancer patients. *Medical Social Sciences, 26*, 323–329.
- Center for Disease Control and Prevention. (2013). Health disparities and inequalities report—United States. *Morbidity and Mortality Weekly Report Supplement, 62*(3). Retrieved from https://reducedisparity.org/site/wp-content/uploads/2014/09/CDC-Health-Disparities-and-Inequalities-Report_2013.pdf#page=186.
- Cuevas, A. G., O'Brien, K., & Saha, S. (2016). African American experiences in healthcare: “I always feel like I’m getting skipped over”. *Health Psychology, 35*, 987–995.
- deGroot, M., Welch, G., Buckland, G., Fergus, M., Ruggiero, L., & Chipkin, S. (2003). Cultural orientation and diabetes self-care in low-income African Americans with type 2 diabetes mellitus. *Ethnicity & Disease, 13*, 6–14.
- Hall, J. A., & Dornan, M. C. (1988). What patients like about their medical care and how often they are asked: A meta-analysis of the satisfaction literature. *Social Science & Medicine, 27*, 935–939.
- Kawachi, I., Daniels, N., & Robinson, D. E. (2005). Health disparities by race and class: Why both matter. *Health Affairs, 24*, 343–352.
- Kennedy, B. R., Mathis, C. C., & Woods, A. K. (2007). African Americans and their distrust of the health care system: Healthcare for diverse populations. *Journal of Cultural Diversity, 14*, 56–60.
- LaVeist, T. A., Nuru-Jeter, A., & Jones, K. E. (2003). The association of doctor-patient race concordance with health services utilization. *Journal of Public Health Policy, 24*, 312–323.
- Levine, R., Foster, J., Fullilove, R., Griggs, N., Hull, P., Husaini, B., et al. (2001). Black-white inequalities in mortality and life expectancy, 1933–1999: Implication for Healthy People 2010. *Public Health Report, 116*, 474–483.

- Mandelblatt, J., Andrews, H., Kao, R., Wallace, R., & Kerner, J. (1996). The late-stage diagnosis of colorectal cancer: Demographic and socioeconomic factors. *American Journal of Public Health*, 86, 1794–1797.
- McQuaid, E. L., & Landier, W. (2018). Cultural issues in medical adherence: Disparities and directions. *Journal of General Internal Medicine*, 33, 200–206.
- Murray, C., Kulkarni, S., Michaud, C., Tomijima, N., Bulzacchelli, M., Iandiorio, T., et al. (2006). Eight Americas: Investigating mortality disparities across races, counties, and race-counties in the United States. *PLoS Medicine*, 3, 1513–1524.
- Nathan, H., Frederick, W., Choti, M., Schulick, R., & Pawlik, T. (2008). Racial disparity in surgical mortality after major hepatectomy. *Journal of the American College of Surgeons*, 207, 312–319.
- National Cancer Institute. (2003). *Surveillance, epidemiology, and end results (SEERS)*. Retrieved from <https://seer.cancer.gov/archive/studies/epidemiology/study16.html>.
- Pullen, E., Perry, B., & Oser, C. (2014). African American women's preventative care usage: The role of social support and racial experiences and attitudes. *Sociology of Health & Illness*, 36, 1037–1053.
- Randall, V. R. (1993). Racist health care: Reforming an unjust health care system to meet the needs of African Americans. *Health Matrix: The Journal of Law-Medicine*, 3, 127–194.
- Schwartz, K., Crossley-May, H., Vigneau, F., Brown, K., & Banerjee, M. (2003). Race, socioeconomic status and stage at diagnosis for five common malignancies. *Cancer Causes Control*, 14, 761–766.
- Shavers, V. L., Lynch, C. F., & Burmeister, L. F. (2000). Knowledge of the Tuskegee Study and its impact on the willingness to participate in medical research studies. *Journal of the National Medical Association*, 92, 563–572.
- Shelton, R. C., Winkel, G., Davis, S. N., Roberts, N., Valdimarsdottir, H., Hall, S. J., & Thompson, H. S. (2010). Validation of the group-based medical mistrust scale among urban Black men. *Journal of General Internal Medicine*, 25, 549–555.
- Smedley, B. D., Stith, A. Y., & Nelson, A. R. (2003). *Institute of medicine: Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington, DC: National Academies Press.
- Terrell, F., & Terrell, S. L. (1981). An inventory to measure cultural mistrust among Blacks. *Western Journal of Black Studies*, 5, 180–184.
- Thompson, H. S., Valdimarsdottir, H., Winkel, G., Jandorf, L., & Redd, W. (2004). The group based medical mistrust scale: Psychometric properties and association with breast cancer screening. *Preventive Medicine*, 38, 209–218.
- Weinick, R. M., Zuvekas, S. H., & Cohen, J. W. (2000). Racial and ethnic differences in access to and use of health care services, 1977 to 1996. *Medical Care Research and Review*, 57, 36–54.
- Whaley, A. L. (2001). Cultural mistrust: An important psychological construct for diagnosis and treatment of African Americans. *Professional Psychology: Research and Practice*, 32, 555–562.
- Williams, J. C. (2017). Black Americans don't trust our healthcare system—Here's why. *The Hill: Opinion Contributor*, 1–4. Retrieved from <http://thehill.com/blogs/pundits-blog/healthcare/347780-black-americans-dont-have-trust-in-our-healthcare-system>.

Suggested Reading

- Murray, C. B. (2016). Racism and mental health. In H. S. Friedman (Ed.), *Encyclopedia of mental health*. Elsevier: United Kingdom.
- Murray, C. B., Woods, V. D., Khatib, S. M., & McAdoo, H. P. (2009). The psychology of health disparities among African Americans. *Journal of Black Psychology*, (Special Issue), 35.
- Smedley, B. D., Stith, A. Y., & Nelson, A. R. (2003). *Institute of medicine: Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington, DC: National Academies Press.
- Washington, H. A. (2006). *Medical apartheid: The dark history of medical experimentation on Black Americans from colonial times to the present*. New York: Doubleday.
- Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine*, 32, 20–47.

Health Disparities

Melissa Ward-Peterson and Eric F. Wagner

Department of Epidemiology, Florida International University, Miami, FL, USA

Introduction

Racial and ethnic minority groups, rural populations, populations with low socioeconomic status (SES), and other marginalized and medically underserved minority populations persistently experience higher than average rates of illness, impairment, and death. For example, diabetes is more than three times as likely to be a cause of death for Native Americans than for other racial/ethnic groups (66.0 per 100,000 population compared with 20.8 per 100,000) (Indian Health Service, 2018). This health disparity, along with others plaguing Native American communities, has contributed to a life expectancy that is 5.5 years shorter than that of the general US population (Indian Health Service, 2018).

These types of disadvantages in health and longevity suffered by minority groups are referred to as **health disparities** (Braveman, 2014). Compared with non-Hispanic Whites, minority racial and ethnic populations experience earlier onset of illness, more severe disease, and poorer quality of care (Williams, Mohammed, Leavell, & Collins, 2010; Williams, Priest, & Anderson, 2016). Minority racial and ethnic populations also experience dramatically lower SES (e.g., income, education, occupation), which is associated with poorer health and reduced longevity (Williams et al., 2010, 2016). However, the relationship between SES and health varies by race. Research has shown as SES levels increase, the health differences between Blacks and Whites become more pronounced, supporting a *diminishing returns hypothesis* for SES vis-à-vis health among minority groups (Farmer & Ferraro, 2005).

Since health disparities adversely affect groups of people who are socially and/or economically disadvantaged, health disparities reflect social injustices. **Health equity** refers to the ideology, motivation, and actions behind correcting these social injustices and eliminating health disparities (Braveman, 2014). A health equity perspective adheres to the human rights principles of nondiscrimination and equality (Braveman, 2014) and presumes that health disparities ultimately are avoidable if the underlying social structures and arrangements creating and maintaining social injustices are addressed (Pearlin, Schieman, Fazio, & Meersman, 2005).

While the alleviation of health problems experienced by minority individuals may be accomplished through the types of direct interventions common in health psychology, the alleviation of health disparities affecting marginalized minority groups can only be accomplished through social change.

Conceptualizations of Health Disparities

There is consensus among health disparities scholars that contextual exposures, a life-course approach, and the consequences of discrimination, intersectionality, and residential segregation must be taken into account in efforts to understand and eliminate health inequities. Each of these key conceptualizations of health disparities is described next.

Contextual exposures refer to the collection of neighborhood, environmental, socioeconomic, and cultural exposures influencing health (Benn & Goldfeld, 2016; Ruiz & Brondolo, 2016; Williams et al., 2016). Minority individuals are disproportionately exposed to toxic social and environmental contexts that reduce health and longevity (Ruiz & Brondolo, 2016). Toxic contextual exposures directly disrupt biological functioning by producing alterations in gene expression, causing tissue and organ damage and dysregulating stress response and recovery systems (Williams et al., 2016). At least in theory, and hopefully in practice, contextual exposures can be changed in ways that improve health and promote health equity and thus are an increasing focus of health disparities research (Benn & Goldfeld, 2016).

A **life-course approach** is essential in efforts to understand and address the drivers of health disparities. Health disparities emerge from exposure to adversity throughout the life course, including before conception, in utero, and throughout infancy, childhood, and adolescence (Williams et al., 2016). A life-course approach takes into account how risks, resources, and demands linked to race, ethnicity, and SES early in life influence health later in life and systematically considers how these factors combine and accumulate with biological factors to affect health outcomes (Braveman, 2014; Williams et al., 2016). Pearlin et al. (2005) point out that health disparities result from differential exposure across the life course to different demands and hardships. For example, exposure to persistent adversity over time produces greater wear and tear on physiological systems, resulting in physiological dysregulation, with consequent health problems; the various terms used by health disparities scholars for this process include *allostatic load*, *weathering*, *cumulative physiological dysregulation*, and *biological risk profile* (Peek et al., 2010). Among marginalized minority and low SES groups, exposure to multiple adversities across time dramatically increases these individuals' allostatic loads (Pearlin et al., 2005). It is well established that higher allostatic loads are associated with increased mortality, morbidity, cognitive decline, and disability (Peek et al., 2010).

A unique contributor to the allostatic load of individuals from marginalized and stigmatized groups is **discrimination**. Discrimination results from the exertion of power by dominant groups to preserve their relative privilege over subordinate groups (Krieger, 2014). Discrimination falls into several categories of justifying ideologies, including racism, sexism, heterosexism, ableism, nativism, ageism, and class bias (Krieger, 2014). There are two overarching types of discrimination: *de jure* discrimination, which is legally permissible in the societies where it occurs, and *de facto* discrimination, which is not legally allowed but occurs nonetheless because of social and cultural norms (Krieger, 2014). Some of the most frequently cited examples of *de jure* discrimination are Jim Crow laws, which legalized discrimination against African Americans in the Southern states from the late nineteenth century through the mid-twentieth century (Krieger, 2014). Although racial discrimination is now prohibited by

law, de facto discrimination continues to be a rampant and destructive issue problem for minority groups, due in no small part to the norms de jure discrimination cemented into American society during the Jim Crow era (Krieger, 2014).

Studies have found that experiencing discrimination is associated with victimization, poor sexual functioning, less REM stage (deep) sleep, more abdominal fat, elevated hemoglobin A1c, coronary artery calcification, uterine myomas, breast cancer, healthcare underutilization, poor treatment adherence, and alcohol, tobacco, and drug use problems (Williams et al., 2010). Discrimination represents an abject violation of human rights. The experience of discrimination, and the stereotypes and prejudice that go along with it, may foment negative social psychological processes (e.g., internalized racism, status incongruence, rising expectations), emotional dysregulation, and impaired stress reactivity and recovery that pile onto an already burdensome allostatic load (Farmer & Ferraro, 2005; Ruiz & Brondolo, 2016; Williams et al., 2010).

Discriminatory experiences have long half-lives—the psychic pain inflicted by discrimination includes not only the pain of the discriminatory experience itself but also worrisome thoughts and emotions about when it will happen again and why it keeps happening again and again (Pearlin et al., 2005). Moreover, while discrimination is often overt and obvious, there are more subtle discriminatory attitudes and actions that contribute to health disparities experienced by marginalized minorities. **Microaggressions** are subtle insults and subjective reminders to marginalized groups of their oppressed status in society (Sue, 2010). Similar to overt types of discrimination, experiencing microaggressions erodes self-esteem and self-worth and raises the allostatic burden, further compromising the health and longevity of marginalized minority groups.

Of course, it is unrealistic to assume all individuals belong to only one socially subordinate group that is exposed to discrimination. While a White woman will encounter challenges posed by sexism, a Black woman will encounter the combined challenges posed by sexism and racism. Worse still will be the lived experience of a Black lesbian, whose life will be impacted not only by sexism and racism but also by heterosexism. First proposed in 1989, **intersectionality** is a framework for understanding this phenomenon of compounding disadvantage caused by membership in more than one subordinate group (Crenshaw, 1989). Intersectionality has been extended to describe challenges posed by sexual orientation and class as well (Bowleg, 2012). Intersectionality is a critical perspective to take when seeking to understand the causes and effects of health disparities experienced by individuals with more than one identity (Bowleg, 2012).

Enduring **residential segregation** by race/ethnicity and income is a particularly pathogenic example of systemic discrimination and is considered “the most critical distinctive social exposure” driving health disparities (Williams et al., 2016). As enumerated by Williams et al. (2010), the pathways through which residential segregation adversely affect health include:

- 1 socioeconomic immobility by limiting access to quality elementary and high school education, preparation for higher education and employment;
- 2 suboptimal health practices because of conditions created by concentrated poverty;
- 3 economic hardship and compound stressors for individuals, households, and communities;
- 4 difficulties and mistrust among neighbors due to weakened community and neighborhood infrastructure;
- 5 institutional neglect and disinvestment leading to increased exposure to environmental toxins, poor quality housing, and criminal victimization; and
- 6 limited access to and low quality of healthcare.

Historical and Current Examples of Health Inequities

The following section provides some historical and current examples of health inequities and the factors driving them. While the examples offered here are by no means exhaustive and focus only on the social context of the United States, they offer an important glimpse into the complex realities facing marginalized communities. The examples illustrate how structural social injustices have dire implications for the health of minority groups. Additionally, they underscore the relevance of the life-course and intersectionality perspectives and show just how pernicious health disparities are in our society.

Historical Examples

A life-course approach emphasizes the role longitudinal exposure plays in creating health disparities. Therefore, scholars interested in health equity must be actively engaged in a broad historical perspective on the social conditions that have produced current circumstances. Historical events can impact the health and well-being of those who lived through them and of later generations.

One example can be found in the historical practice of “redlining,” in which banks refused to provide home loans in neighborhoods deemed undesirable (Aaronson, Hartley, & Mazumder, 2018). For minority populations, this practice eliminated access to the financial capital needed to purchase homes. Aaronson, Hartley, and Mazumder estimate discriminatory lending practices contributed to up to half of the disparities in Black/African American home ownership between 1950 and 1980. The systematic obstruction of this path to wealth had a cumulative effect on future generations by limiting the assets that were passed from parents to their children and grandchildren.

A second example can be found in the enduring *historical trauma* experienced by Native Americans. Beginning with first contact and continuing through subsequent colonization and the expansion of the United States, indigenous communities have suffered through genocide, dislocation, and other deplorable patterns of violence inflicted upon them by the colonists and their descendants. The cumulative adverse effects of this history are known as “historical trauma,” which describes the profoundly negative, long-term, and intergenerational impact of colonization, cultural suppression, and historical oppression on indigenous peoples (Brave Heart, Elkins, Tafoya, Bird, & Salvador, 2012). The manifold health disparities experienced by Native Americans (e.g., significantly greater morbidity and mortality from diabetes, cancer, substance use, obesity, bronchiolitis, and injuries; Indian Health Service, 2018) are at least partially attributable to massive historical trauma imposed across many generations.

Contemporary Examples

A life-course approach is also valuable when examining contemporary examples of health disparities. A child belonging to a minority group is more likely to be born into racial residential segregation and underprivileged communities, a remnant of redlining and Jim Crow-era discriminatory practices. In the United States, Black, Latino, and Native American children disproportionately grow up in neighborhoods with constrained opportunity structures, under-resourced schools, compound and unrelenting stress, and toxic social and physical environments (Braveman, 2014; Pearlin et al., 2005; Williams et al., 2010).

Environmental exposures are of critical importance during childhood when cognitive and physical development is vulnerable. Lead exposure, which may occur through poor housing conditions or contaminated water, is an especially nefarious environmental toxin because it causes cognitive deficits that limit children's academic potential, thereby altering their futures (Zhang et al., 2013). Racial/ethnic disparities in lead exposure have persisted for decades; low-income Black and Hispanic children residing in older housing continue to have the highest prevalence of exposure (Jacobs, 2011). The most effective way to address lead exposure is to move into a new residence or neighborhood, but this solution is not feasible for the groups most frequently affected. Despite the known risks of even low levels of lead exposure (Lanphear, Dietrich, Auinger, & Cox, 2000) and evidence supporting the effectiveness of interventions that address these risks (Jacobs, 2011), health disparities due to differences in environmental exposures endure. As a result, environmental justice must remain at the forefront of efforts to achieve health equity.

Disparities continue into late adolescence and young adulthood. For minority men in the United States, incarceration becomes an important determinant of health. Incarceration is a "disorderly transitional" stressful event that creates nonnormative, undesired, involuntary, and often irreversible life changes (Williams et al., 2010). Incarceration is associated with subsequent homelessness, employment and financial difficulties, destabilization of relationships, increased health risks, and other impediments to health and well-being (Iguchi, Bell, Ramchand, & Fain, 2005). In 2016, Black individuals were incarcerated at a rate five times higher than that of White individuals (1,608 per 100,000 people compared with 274 per 100,000), and the number of Black children in juvenile detention far exceeds the number of White children in juvenile detention (Gramlich, 2018; Iguchi et al., 2005). The preceding speaks to the putative influence of incarceration on health disparities and the importance of measuring incarceration in studies of health and longevity differences between Black and White individuals (Williams et al., 2010).

Distinctive health disparities are experienced by minority women. In 2011, among Black and Hispanic women, 64 and 50% of pregnancies were unintended, respectively, compared with 38% of pregnancies among White women (Finer & Zolna, 2016). Once pregnant, Black women can expect drastically increased maternal morbidity and mortality compared with White women (Tucker, Berg, Callaghan, & Hsia, 2007). From 2010 to 2014, Black women died at a rate of 40.0 per 100,000 live births, while White women died at a rate of 10.4 per 100,000 live births (Centers for Disease Control and Prevention [CDC], 2018). Potential mediators such as educational achievement do not seem to improve outcomes for Black mothers; a 2016 study conducted by the New York City Department of Health found the rate of severe maternal morbidity for college-educated Black mothers was still more than twice the rate for White mothers who had not graduated from high school (New York City Department of Health and Mental Hygiene, 2016).

Similar to individuals who are racial/ethnic minorities, individuals who are sexual minorities (LGBTQIA) are also subject to de jure and de facto discrimination throughout their lives. While some states have passed laws that explicitly protect LGBTQIA individuals from employment discrimination and hate crimes, many states have not (Krieger, 2014). In fact, as of 2017, 12 states had passed laws allowing for the denial of services to LGBTQIA individuals, largely based on the strongly held religious beliefs of business owners (Raifman, Moscoe, Austin, Hatzenbuehler, & Galea, 2018). Evidence has shown that LGBTQIA individuals in states with de jure discriminatory practices have higher rates of mental distress than LGBTQIA individuals from states without de jure discrimination (Raifman et al., 2018).

Health Disparities Research Priorities

Health disparities are receiving increasing attention in health psychology. Though it has long been recognized that marginalized minority groups experience large disadvantages in health and longevity, only recently have sophisticated models and associated empirical work on health disparities and their alleviation begun to appear in the literature. As the literature has grown, so has extramural funding opportunities for conducting research explicitly intended to understand and eliminate health disparities. The following section enumerates several current research priorities for future research on decreasing health disparities and promoting health equity.

Health Disparities Research Priorities

- 1 Develop multivariate, multilevel, transdisciplinary, integrative, life-course models to guide research on contextual exposures, their epigenetic effects, and the onset and course of illness among vulnerable populations (Ruiz & Brondolo, 2016; Williams et al., 2010, 2016).
- 2 Conduct research focused on modifiable drivers of improved health outcomes among vulnerable populations (Benn & Goldfeld, 2016; Ruiz & Brondolo, 2016).
- 3 Identify influential contextual exposures and optimal intervention strategies at each specific point of the disease continuum based on research conducted with both healthy and clinical populations (Ruiz & Brondolo, 2016; Williams et al., 2010).
- 4 Understand resilience and coping in the context of social disadvantage and stress (Ruiz & Brondolo, 2016; Williams et al., 2010).
- 5 Ascertain the degree to which medical care and genetic factors contribute to racial and SES differences in health (Williams et al., 2010).
- 6 Explore the intersections of race, ethnicity, SES, sex, gender, immigration status, and other variables alongside experiences of discrimination and health outcomes (Ruiz & Brondolo, 2016; Williams et al., 2010).
- 7 Estimate the heretofore under-examined contributions of occupational stress, work environment, community violence, and diet to health disparities (Peek et al., 2010; Williams et al., 2010).
- 8 Attend to the putative influences of acculturation, assimilation, and biological risk on the Hispanic paradox, the health and longevity advantage of foreign-born (vs. US-born) Hispanics (Peek et al., 2010; Williams et al., 2010).

Call to Action

Racial and ethnic minority groups, LGBTQIA groups, rural populations, populations with low SES, and other marginalized and medically underserved minority populations persistently experience earlier onset of illness, more severe disease, poorer quality healthcare, and disproportionate morbidity and mortality from disease. These health disparities are long standing and result from historical and contemporary structural and social injustices such as constrained opportunity structures, under-resourced schools, compound and unrelenting stress, toxic social and physical environments, de jure and de facto discrimination, residential segregation, and the experience of microaggressions. The development and testing of multivariate, multilevel, transdisciplinary, integrative, life-course models of health disparities is urgently needed, especially in regard to modifiable drivers of health outcomes among

vulnerable populations. Although additional research is critically important, advocacy, community engagement, and effective communication of findings to decision makers and the lay public are all indispensable and urgent for advancing health equity and eliminating health disparities. While the health risks and health problems experienced by marginalized minority groups may be modifiable on an individual level through behavior change as per conventional health psychology practices, the long-term elimination of health disparities is dependent on social change.

Acknowledgment

Preparation of this chapter was supported by the National Institute of Minority Health and Health Disparities grant award U54MD012393 for FIU-RCMI. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Author Biographies

Melissa Ward-Peterson, PhD, MPH, is a postdoctoral associate in the Community-Based Research Institute at Florida International University in Miami, Florida, where she manages the Research Infrastructure Core for FIU's Research Center in Minority Institutions (FIU-RCMI). A social epidemiologist by training, her research interests include disparities in access to substance abuse treatment and women's health. She has extensive experience in health equity research and program management and has provided technical support for projects sponsored by a number of federal and international agencies, including HRSA, NIH's Fogarty International Center, the US Department of State, the World Health Organization, the World Bank, and UNAIDS.

Eric F. Wagner, PhD, is the director of Florida International University's (FIU) Community-Based Research Institute, a professor in FIU's Stempel College of Public Health & Social Work, the principal investigator of FIU's NIMHD-funded Health Disparities Research Center in a Minority Institute, and a psychologist licensed to practice in Florida (active) and Rhode Island (inactive). His clinical research expertise is addressing substance use and related problems among teenage, young adult, and underrepresented medically underserved populations. His work has been supported by grants from NIAAA, NIDA, NIMHD, HRSA, SAMHSA, the Ware Foundation, the Aetna Foundation, and Consejo Nacional de Ciencia y Tecnología.

References

- Aaronson, D., Hartley, D., & Mazumder, B. (2018). *The Effects of the 1930s HOLC "Redlining" Maps*. Federal Reserve Bank of Chicago, WP 2017-12.
- Benn, E. K., & Goldfeld, K. S. (2016). Translating context to causality in cardiovascular disparities research. *Health Psychology, 35*, 403–406. doi:10.1037/hea0000309
- Bowleg, L. (2012). The problem with the phrase women and minorities: Intersectionality-an important theoretical framework for public health. *American Journal of Public Health, 102*, 1267–1273. doi:10.2105/AJPH.2012.300750

- Brave Heart, M. Y., Elkins, J., Tafoya, G., Bird, D., & Salvador, M. (2012). Wicasa Was'aka: Restoring the traditional strength of American Indian boys and men. *American Journal of Public Health, 102*, S177–S183. doi:10.2105/AJPH.2011.300511
- Braveman, P. (2014). What is health equity: And how does a life-course approach take us further toward it? *Maternal and Child Health Journal, 18*, 366–372.
- Centers for Disease Control and Prevention. (2018). *Pregnancy mortality surveillance system*. Retrieved from <https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pregnancy-mortality-surveillance-system.htm>
- Crenshaw, K. (1989). Demarginalizing the intersection of race and sex: A black feminist critique of anti-discrimination doctrine, feminist theory and antiracist politics. *The University of Chicago Legal Forum, 1989*, 139–167.
- Farmer, M. M., & Ferraro, K. F. (2005). Are racial disparities in health conditional on socioeconomic status? *Social Science and Medicine, 60*, 191–204. doi:10.1016/j.socscimed.2004.04.026
- Finer, L. B., & Zolna, M. R. (2016). Declines in unintended pregnancy in the United States, 2008–2011. *New England Journal of Medicine, 374*, 843–852. doi:10.1056/NEJMsa1506575
- Gramlich, J. (2018). *The gap between the number of blacks and whites in prison is shrinking*. Pew Research Center. Retrieved from <http://www.pewresearch.org/fact-tank/2018/01/12/shrinking-gap-between-number-of-blacks-and-whites-in-prison/>
- Iguchi, M. Y., Bell, J., Ramchand, R. N., & Fain, T. (2005). How criminal system racial disparities may translate into health disparities. *Journal of Health Care for the Poor and Underserved, 16*, 48–56. doi:10.1353/hpu.2005.0114
- Indian Health Service. (2018). *Disparities*. Retrieved from <https://www.ihs.gov/newsroom/factsheets/disparities/>
- Jacobs, D. E. (2011). Environmental health disparities in housing. *American Journal of Public Health, 101*, S115–S122. doi:10.2105/AJPH.2010.300058
- Krieger, N. (2014). Discrimination and health inequities. In L. F. Berkman, I. Kawachi, & M. M. Glymour (Eds.), *Social epidemiology* (2nd ed.). New York, NY: Oxford University Press.
- Lanphear, B. P., Dietrich, K., Auinger, P., & Cox, C. (2000). Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. *Public Health Reports, 115*, 521–529.
- New York City Department of Health and Mental Hygiene. (2016). *Severe maternal morbidity in New York City, 2008–2012*. New York. Retrieved from <https://www1.nyc.gov/assets/doh/downloads/pdf/data/maternal-morbidity-report-08-12.pdf>
- Pearlin, L. I., Schieman, S., Fazio, E. M., & Meersman, S. C. (2005). Stress, health, and the life course: Some conceptual perspectives. *Journal of Health and Social Behavior, 46*, 205–219. doi:10.1177/002214650504600206
- Peek, M. K., Cutchin, M. P., Salinas, J. J., Sheffield, K. M., Eschbach, K., Stowe, R. P., & Goodwin, J. S. (2010). Allostatic load among non-Hispanic Whites, non-Hispanic Blacks, and people of Mexican origin: Effects of ethnicity, nativity, and acculturation. *American Journal of Public Health, 100*, 940–946. doi:10.2105/AJPH.2007.129312
- Raifman, J., Moscoe, E., Austin, S. B., Hatzenbuehler, M. L., & Galea, S. (2018). Association of state laws permitting denial of services to same-sex couples with mental distress in sexual minority adults: A difference-in-difference-in-differences analysis. *JAMA Psychiatry, 75*, 671–677. doi:10.1001/jamapsychiatry.2018.0757
- Ruiz, J. M., & Brondolo, E. (2016). Introduction to the special issue Disparities in cardiovascular health: Examining the contributions of social and behavioral factors. *Health Psychology, 35*, 309–312. doi:10.1037/hea0000364
- Sue, D. W. (2010). *Microaggressions in everyday life: Race, gender, and sexual orientation*. Hoboken, NJ: Wiley.
- Tucker, M. J., Berg, C. J., Callaghan, W. M., & Hsia, J. (2007). The Black-White disparity in pregnancy-related mortality from 5 conditions: Differences in prevalence and case-fatality rates. *American Journal of Public Health, 97*, 247–251. doi:10.2105/AJPH.2005.072975

- Williams, D. R., Mohammed, S. A., Leavell, J., & Collins, C. (2010). Race, socioeconomic status, and health: Complexities, ongoing challenges, and research opportunities. *Annals of the New York Academy of Sciences*, 1186, 69–101. doi:10.1111/j.1749-6632.2009.05339.x
- Williams, D. R., Priest, N., & Anderson, N. B. (2016). Understanding associations among race, socioeconomic status, and health: Patterns and prospects. *Health Psychology*, 35, 407–411. doi:10.1037/hea0000242
- Zhang, N., Baker, H. W., Tufts, M., Raymond, R. E., Salihu, H., & Elliott, M. R. (2013). Early childhood lead exposure and academic achievement: Evidence from Detroit public schools, 2008-2010. *American Journal of Public Health*, 103, e72–e77. doi:10.2105/AJPH.2012.301164

Suggested Reading

- Barr, D. A. (2014). *Health disparities in the United States: Social class, race, ethnicity, and health* (2nd ed.). Baltimore, MD: Johns Hopkins University Press.
- Berkman, L. F., Kawachi, I., & Glymour, M. M. (Eds.) (2014). *Social epidemiology* (2nd ed.). New York, NY: Oxford University Press.
- Marmot, M., & Wilkinson, R. G. (Eds.) (2006). *Social determinants of health* (2nd ed.). New York, NY: Oxford University Press.
- Oakes, J. M., & Kaufman, J. S. (Eds.) (2017). *Methods in social epidemiology* (2nd ed.). San Francisco, CA: Wiley.

Injury, Accident, and Injury Prevention

Kristi Alexander¹ and Riley Cropper²

¹*Department of Psychology, University of Central Florida, Orlando, FL, USA*

²*Stanford University Counseling and Psychological Services, Stanford Hospital and Clinics, Stanford, CA, USA*

Worldwide, unintentional injuries are one of the greatest threats to the health and well-being of people. Nine percent of all deaths globally are attributable to injuries (World Health Organization [WHO], 2017), and unintentional injury is the leading cause of death for children older than 9 years (Ameratunga & Peden, 2009). In the United States alone, injuries are the leading cause of death for people 1–44 years (Centers for Disease Control and Prevention [CDC], 2018). For every injury death, there are countless more hospitalizations, emergency room visits, and primary care treatments.

Unintentional injuries have been defined in a variety of ways, but for the purposes of this entry, these are unplanned events that result in bodily harm or tissue damage (WHO, 2008). Baker (1992) defined injury as “physical damage that results when a human body is suddenly subjected to energy in amounts that exceed the threshold of physiological tolerance—or else the results of a lack of one or more vital elements such as oxygen.” Although what constitutes an “unintentional injury” varies somewhat by who is defining the term, research and policy has typically limited the definition to harm that occurs without intent such as injuries sustained during a motor vehicle accident, poisonings, drownings, falls, and burns (WHO, 2008).

There are some differences, however, in how the public interprets and defines unintentional injuries and accidents. Girasek (2015), in a survey of over 900 adults, found differences between the public’s and professionals’ interpretation of the word. Specifically, many professionals prefer not to use the term “accident,” as they believe it reinforces the common misconception that injuries are not preventable or predictable. However, Girasek found that the term “accident” evoked associations with preventability in 83% of the public surveyed. Although the term “accident” may imply a lack of predictability, the reality is that injuries almost always involve foreseeable high-risk behaviors and are therefore typically predictable and preventable.

Across the globe, unintentional injuries represent the greatest threat to the health and safety of people across all ages. According to the WHO (2017), 9% of all deaths worldwide are attributable to injuries, which is approximately 1.7 times the number of deaths resulting from HIV/AIDS, tuberculosis, and malaria combined. Millions more people, who are not killed, are injured and incur lifelong disabilities as a result of injury. In the United States alone, unintentional injuries were the third leading cause of death in 2016 across all age groups (National Safety Council [NSC], 2018) demonstrating that unintentional injuries do not only affect children and young adults, but individuals across the lifespan.

Although unintentional injuries occur at every age, around the world, young people are differentially impacted. For people between the ages of 15 and 29 years, road traffic injuries are the number one cause of death for both men and women. Younger children are also at high risk to die as result of a traffic injuries and drowning. Furthermore, poverty increases the risk of injury-related death; about 90% of such deaths occur in low- and lower-middle countries (Wegman, 2017). For example, more than two-thirds of all road traffic child deaths occur in underdeveloped communities of Southeast Asia and Africa (WHO, 2008). Additionally, in the United States, there are significant racial and ethnic differences in mortality rates. For example, in comparison to European Americans, the risk of a fire-related death is significantly greater for Native American and African Americans over 55 years (Bishai & Lee, 2010). Lastly, there are robust gender differences, emerging at birth and lasting until old age, resulting in boys and men being significantly more likely to be fatally injured (NSC, 2018).

Across the lifespan, there are predictable patterns both in the United States and the world in the types of unintentional injuries that occur. Many factors contribute to these differences including cognitive and motor skills. Worldwide, suffocation is the leading cause of death in infants younger than 1 year; falls are the most common *nonfatal* injuries treated in the emergency department (WHO, 2008). The risk of fall injury increases dramatically over the first year of life, reflecting the impact of motor skill development on injury risk.

For children 1–4 years, the leading causes of injury death for children across the world are drowning, followed by road traffic injuries (WHO, 2008). Unfortunately, this trend continues throughout childhood, with drowning and traffic injuries remaining the leading causes of injury death for children across the globe.

In the United States, motor vehicle injuries, including both traffic and pedestrian deaths, are the leading cause of death beginning at age 5. With regard to *nonfatal* unintentional injuries, US children are most likely to sustain an injury due to a fall (5- to 9-year-olds) or being struck by or against an object or person (NSC, 2017). School-age children are more likely to be injured when they are playing outside and away from the home, most commonly on the playground, while playing sports, or as a pedestrian.

Road traffic injuries are the leading cause of death across the world for adolescents and young adults (15–29 years; WHO, 2017). It is during this developmental period that the largest gender difference in motor vehicle deaths is realized with boys and men sustaining significantly more than girls and women (WHO, 2017). In addition, serious nonfatal injuries for adolescents and young adults result from being struck by or against an object or person.

For adults worldwide, road traffic injuries drop to the third cause of death after age 30 (WHO, 2017). During this same developmental period, falls are the leading urgent cause of *nonfatal* injury. In the United States, injury is the seventh leading cause of death for older adults (65 years +) (CDC, 2018). Of these injury deaths, falls are the most prevalent cause for Americans 65 or older. In addition to falls, which account for 85% of injury deaths in this age

group, older adults are vulnerable to motor vehicle injuries, poisonings, and lacerations (DeGrauw, Annest, Stevens, Xu, & Coronado, 2016).

Older adult pedestrians are especially vulnerable to severe injury or death as a result of a pedestrian–vehicle collision. In the United States, the fatality rate for pedestrians over 65 was higher than the rate for all other adults. In addition, in comparison with children or adults less than 60, adults over 60 had a higher injury severity risk in a full-frontal pedestrian–car collision (Niebuhr, Junge, & Rosén, 2016).

Despite the seemingly random nature of unintentional injuries, there are some psychological and temperamental characteristics that have been repeatedly associated with increased risk for injuries. Sensation seeking, or a desire to experience novel situations, is a temperamental characteristic that has been repeatedly implicated as a risk factor for unintentional injuries across the lifespan (Morrongiello, Sandomierski, & Valla, 2010; Schwebel, 2004). For example, children that demonstrate higher levels of sensation seeking tend to pursue novel situations regardless of the presence of physical risk, which in turn increases their risk for injury (Morrongiello et al., 2010). Similar results have been found with adults; multiple studies examining driving behavior have found an association between sensation seeking and an increased risk for motor vehicle accidents (Graziano et al., 2015; O’Jile, Ryan, Parks-Levy, Betz, & Gouvier, 2004). O’Jile et al. (2004) also found that men scored higher in sensation seeking, which may partially explain why males have higher rates of unintentional injuries across the lifespan.

Another temperamental characteristic that has been linked to increased injury risk is impulsivity, or the tendency to quickly react to novel stimuli (Schwebel, 2004; Stavrinos et al., 2011). Although the exact link between impulsivity and injury risk is unclear, it has been proposed that individuals that are more impulsive may overestimate their physical abilities, as they react quickly to stimuli without thoughtfully considering their ability to complete the task. For example, Thomson and Carlson (2015) found that in combination with other variables, high impulsivity was associated with risky skiing and snowboarding in adults. Further, inhibitory control, or the capacity to suppress behavioral responses, has also been shown to play a role in the relationship between impulsivity and unintentional injury. For example, Stavrinos et al. (2011) found that an individual’s ability to inhibit impulsive responses while crossing the street (e.g., darting into the street before assessing safety) significantly impacted his or her risk for pedestrian injury. Thus far, inhibitory control is the only temperamental characteristic that has been shown to reduce an individual’s risk for injury, as those who possess this quality are more likely to perceive potential dangers and avoid them, be controlled and cautious, engage in less dangerous behavior, and comply with rules (Schwebel, 2004).

Prevention

Unintentional injuries have a substantial impact around the world, but ultimately, they are largely predictable and preventable (WHO, 2017). In the United States alone, research suggests the over 90% of pediatric injuries could have been prevented if someone had done something to change the circumstances prior the injury (Rimsza, Schackner, Bowen, & Marshall, 2002). Although research over the past few decades has identified those demographic and behavioral characteristics that increase the risks of unintentional injury, the development and evaluation of effective injury prevention programs has lagged (CDC, 2018).

Unintentional injury prevention has proven to be a difficult issue to address worldwide. Globally, prevention has decreased unintentional injury deaths substantially (Ameratunga & Peden, 2009), but the gains have not been equally realized (Vecino-Ortiz, Jafri, & Hyder, 2018). While high-income countries have realized significant improvements in safety and prevention of injury, low- and middle-income countries have not (WHO, 2017). As previously noted, the vast majority of injury-related deaths occur in low- and middle-income countries where safety and prevention efforts appear to have not caught up with the developments that increase risk. Specifically, low- and middle-income countries have 90% of the world's road traffic deaths but only have half of the vehicles in the world (WHO, 2017). Consequently, road traffic deaths have been steadily increasing in India and Cambodia over the past decade and over two-thirds of all *child* road traffic deaths occur in under-resourced countries in Southeast Asia and Africa (WHO, 2017). In the United States, there are also significant racial and ethnic disparities in unintentional injury death rates (Pollack & LaVeist, 2012).

To date, legislatively mandated and environmental prevention strategies have been among the most successful interventions in decreasing specific injuries (Christoffel & Gallagher, 2016). In the United States, laws that mandated the use of child safety seats, smoke detectors, and graduated driver licensing have each been effective in reducing unintentional injuries (Gardner & the Committee on Injury, Violence, and Poison Prevention, 2007). Similarly, in Asia and Europe, helmet laws have significantly reduced motorcyclist deaths (Araujo, Illanes, Chapman, & Rodrigues, 2017), and Australian pool fencing legislation has decreased child drowning deaths (Franklin & Peden, 2017). Environmental interventions, such as child-resistant packaging for prescription and over-the-counter medicines and air bags, have been effective in decreasing injuries, but there is a perception within the field that these types of interventions may have been "exhausted" (Gielen, Sleet, & DiClemente, 2006). This has led to a current focus on behaviorally based prevention strategies such as teaching swimming and water safety to reduce drownings, the use of safety equipment in sports, and teaching motorists to avoid texting while driving (CDC, 2018; WHO, 2017).

Although effective, behaviorally based prevention strategies require action (Sleet & Gielen, 2008). Even when injury prevention interventions are mandated by law, there remains a behavioral component; children need to wear helmets while bicycling, smoke alarms need to be checked, and seat belts need to be buckled. Unfortunately, many factors influence the adoption of injury prevention behaviors. For example, research has found that even when the efficacy of prevention programs for sports injuries is well established, a minority of coaches employ them with their players (Norcross, Johnson, Bovbjerg, Koester, & Hoffman, 2016). Similarly, other research has demonstrated that over 50% of parents of young children fail to use prevention measures to reduce injuries at home, making even the "most effective" strategies largely ineffective in preventing the occurrence of unintentional injuries in children (Tsoumakas, Dousis, Mavridi, Germou, & Matziou, 2009). Thus, understanding factors influencing compliance is key to increasing the effectiveness of prevention strategies.

However, compliance is not the only issue, as unintentional injuries still occur even when interventions are implemented correctly with absolute compliance. Therefore, the current prevention strategies seem to be failing to address some aspect of this problem (Schwebel & Barton, 2006). In an attempt to resolve this issue, researchers have begun to take a greater multidisciplinary focus as they formulate new interventions. For example, prevention strategies that focus on psychology, public health, medicine, and engineering simultaneously in order to address the beliefs and behaviors of the individual and caregiver as well as the environmental characteristics that contribute to increased injury risk (Schwebel & Barton, 2006).

Author Biographies

Kristi Alexander is a clinical professor and director of clinical training in the Department of Psychology at the University of Central Florida. Her primary research interest is in pediatric unintentional injuries.

Riley Cropper is a staff psychologist at Stanford University's Counseling and Psychological Services.

References

- Ameratunga, S. N., & Peden, M. (2009). World report on child injury prevention: A wake-up call. *Injury*, 40(5), 469–470. doi:10.1016/j.injury.2008.11.015
- Araujo, M., Illanes, E., Chapman, E., & Rodrigues, E. (2017). Effectiveness of interventions to prevent motorcycle injuries: Systematic review of the literature. *International Journal of Injury Control and Safety Promotion*, 24(3), 406–422. doi:10.1080/17457300.2016.1224901
- Baker, S. P. (1992). *The injury fact book*. New York, NY: Oxford University Press.
- Bishai, D., & Lee, S. (2010). Heightened risk of fire deaths among older African Americans and Native Americans. *Public Health Reports*, 225, 406–413.
- Centers for Disease Control and Prevention. (2018). *Injury prevention and control*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Christoffel, T., & Gallagher, S. S. (2016). *Injury prevention and public health: Practical knowledge, skills, and strategies* (2nd ed.). Boston, MA: Jones and Bartlett Publishers.
- DeGrauw, X., Annest, J., Stevens, J. A., Xu, L., & Coronado, V. (2016). Unintentional injuries treated in hospital emergency departments among persons aged 64 years and older, United States, 2006–2011. *Journal of Safety Research*, 56, 105–109. doi:10.1016/j.jsr.2015.11.002
- Franklin, R. C., & Peden, A. E. (2017). Improving pool fencing legislation in Queensland, Australia: Attitudes and impact on child drowning fatalities. *International Journal of Environmental Research and Public Health*, 14(12). doi:10.3390/ijerph 14121450
- Gardner & the Committee on Injury, Violence, and Poison Prevention. (2007). Office-based counseling for unintentional injury prevention. *Pediatrics*, 119(1), 202–206. doi:10.1542/peds.2006-2899
- Gielen, A. C., Sleet, D. A., & DiClemente, R. J. (2006). *Injury and violence prevention: Behavioral science theories, methods and applications*. San Francisco, CA: Jossey-Bass.
- Girasek, D. C. (2015). How members of the public interpret the word accident. *Injury Prevention*, 21(3), 205–210. doi:10.1136/ip.5.1.19rep
- Graziano, P. A., Reid, A., Slavec, J., Paneto, A., McNamara, J. P., & Geffken, G. R. (2015). ADHD symptomatology and risky health, driving, and financial behaviors in college: The mediating role of sensation seeking and effortful control. *Journal of Attention Disorders*, 19(3), 179–190. doi:10.1177/1087054714527792
- Morrongiello, B. A., Sandomierski, M., & Valla, J. (2010). Early identification of children at risk of unintentional injury: A sensation seeking scale for children 2–5 years of age. *Accident Analysis and Prevention*, 42(4), 1332–1337.
- National Safety Council. 2018, March 17. *Injury facts: The source for injury stats*. Retrieved from <https://www.nsc.org/membership/member-resources/injury-facts>
- Niebuhr, T., Junge, M., & Rosén, E. (2016). Pedestrian injury risk and the effect of age. *Accident Analysis and Prevention*, 86, 121–128. doi:10.1016/j.aap.2015.10.026
- Norcross, M. F., Johnson, S. T., Bovbjerg, V. E., Koester, M. C., & Hoffman, M. A. (2016). Factors influencing high school coaches' adoption of injury prevention programs. *Journal of Science and Medicine in Sport*, 19(4), 299–304. doi:10.1016/j.jsams.2015.03.009
- O'Jile, J. R., Ryan, L. M., Parks-Levy, J., Betz, B., & Gouvier, W. D. (2004). Sensation seeking and risk behaviors in young adults with and without a history of head injury. *Applied Neuropsychology*, 11, 107–112.

- Pollack, K. M., & LaVeist, T. (2012). Advancing understanding of racial and ethnic inequalities in injury research. *Injury Prevention*, 18(2), 143–144. doi:10.1136/injuryprev-2012-040344
- Rimsza, M. E., Schackner, R., Bowen, K. A., & Marshall, W. (2002). “Can child deaths be prevented”? The Arizona Child Fatality Review Program experience. *Pediatrics*, 110, 1–7. doi:10.1542/peds.110.1.e11
- Schwebel, D. C. (2004). The role of impulsivity in children’s estimation of physical ability: Implications for children’s unintentional injury risk. *American Journal of Orthopsychiatry*, 74(4), 584–588. doi:10.1037/0002-9432.74.4.584
- Schwebel, D. C., & Barton, B. K. (2006). Temperament and children’s unintentional injuries. In M. E. Vollrath (Ed.), *Handbook of personality and health* (pp. 51–71). New York, NY: Wiley.
- Sleet, D. A., & Gielen, A. C. (2008). Behavioral interventions for injury and violence prevention. In L. S. Doll, S. E. Bonzo, J. A. Mercy, D. A. Sleet, L. S. Doll, S. E. Bonzo, & D. A. Sleet (Eds.), *Handbook of injury and violence prevention* (pp. 397–410). New York, NY: Springer Science and Business Media. doi:10.1007/978-0-387-29457-5_22
- Stavrinos, D., Biasini, F. J., Fine, P. R., Hodgins, J. B., Khatri, S., Mrug, S., & Schwebel, D. C. (2011). Mediating factors associated with pedestrian injury in children with attention deficit/hyperactivity disorder. *Pediatrics*, 128(8), 296–302. doi:10.1542/peds.2010-3829
- Thomson, C. J., & Carlson, S. R. (2015). Increased patterns of risky behaviours among helmet wearers in skiing and snowboarding. *Accident; Analysis and Prevention*, 75, 179–183. doi:10.1016/j.aap.2014.11.024
- Tsoumakas, K., Dousis, E., Mavridi, F., Gremou, A., & Matziou, V. (2009). Parent’s adherence to children’s home-accident preventive measures. *International Nursing Review*, 56(3), 369–374. doi:10.1111/j.1466-7657.2009.00720.x
- Vecino-Ortiz, A. I., Jafri, A., & Hyder, A. A. (2018). Effective interventions for unintentional injuries: A systematic review and morality impact assessment among the poorest billion. *Globalization and Health*, 6(5), e523–e534. doi:10.1016/S2214-109X(18)30107-4
- Wegman, F. (2017). The future of road safety: A worldwide perspective. *IATSS Research*, 40, 66–71.
- World Health Organization. (2008). *World report on child injury prevention*. Geneva, Switzerland: WHO.
- World Health Organization. (2017). *Injury and violence facts*. Geneva, Switzerland: WHO.

Suggested Reading

- Chang, M.-H., Moonesinghe, R., Athar, H. M., & Truman, B. I. (2016). Trends in disparity by sex and race/ethnicity for the leading causes of death in the United States—1999–2010. *Journal of Public Health Management and Practice*, 22, S13–S24. doi:10.1097/PHH.0000000000000267
- Morrongiello, B. A., Sandomierski, M., & Valla, J. (2010). Early identification of children at risk of unintentional injury: A sensation seeking scale for children 2–5 years of age. *Accident Analysis and Prevention*, 42(4), 1332–1337.
- Pollack, K. M., & LaVeist, T. (2012). Advancing understanding of racial and ethnic inequalities in injury research. *Injury Prevention*, 18(2), 143–144. doi:10.1136/injuryprev-2012-040344
- Vecino-Ortiz, A. I., Jafri, A., & Hyder, A. A. (2018). Effective interventions for unintentional injuries: A systematic review and morality impact assessment among the poorest billion. *Globalization and Health*, 6(5), e523–e534. doi:10.1016/S2214-109X(18)30107-4

Integrated Primary Care

Sandra B. Morissette¹ and Brian Konecky²

¹*Department of Psychology, The University of Texas at San Antonio College of Sciences,
San Antonio, TX, USA*

²*Boise VAMC Virtual Integrated Multisite Patient Aligned Care Team Hub, Boise, ID, USA*

Integrated primary care involves the delivery of mental and behavioral health services within primary care settings. There are numerous terms for this care delivery system including primary care mental health integration (PCMHI) or integrated primary care behavioral health (IPCBH). Some contend that the term “behavioral health” may be less stigmatizing than “mental health”; therefore, henceforth the term IPCBH is used. This terminology is important for consideration, as a major aim of IPCBH programs is to reduce stigma and other barriers to care (e.g., location, scheduling separate appointments, longer waiting times) by providing behavioral health services directly within the primary care setting. Integrated care, in which the IPCBH provider is part of the primary care treatment team, is distinguished from co-located care (referral within the same medical center) and coordinated care (behavioral health services referred to a separate location). The rationale for integrating mental and behavioral health services within primary care settings is that often primary care providers (PCPs) are the first point of contact for patients to address their mental and behavioral health concerns. Further, failure to detect mental health conditions along with underutilization of mental health services can lead to negative health outcomes for patients and place significant burden on healthcare systems. Thus, by design, the IPCBH provider works closely with the patient’s medical provider as an integrated part of the primary care team to improve access and deliver seamless coordination of care for both physical and mental/behavioral health conditions. This seamless coordination of services is perhaps best exemplified by “warm handoffs” in which PCPs introduce patients to an IPCBH provider during a medical visit. This decreases barriers to care, the burden of scheduling, and stigma and also allows for a patient to have a same-day visit with an IPCBH provider when needed. Further, warm handoffs increase communication between all treatment team members and the patient and can occur multi-directionally between PCPs, IPCBH providers, and IPCBH prescribers.

IPCBH models focus on population-based care in which fewer, more specific services are offered to more individuals, in contrast to specialty mental health services in which more intensive treatments are offered to patients with more severe clinical presentations. Generally, IPCBH treatments are designed to be brief (one to six sessions) and are shorter in duration (20–30 min) than typical 50- to 60-min specialty psychotherapy sessions. Patients who are perceived as being able to benefit from such brief interventions, including preventative interventions, are seen within primary care. Should it be determined at any point in treatment that the patient needs a higher level of care or more intensive treatment, the patient is referred to specialty mental health services. However, should the patient need care sooner than the availability of specialty services (e.g., waitlist is too long), the IPCBH provider may opt to engage in watchful waiting to observe the progression of symptoms or begin treatment within primary care and then transfer the patient to specialty services when available. Likewise, if a patient needs specialty mental health services but is not ready for such, the IPCBH provider may work with the individual to increase motivation or basic coping strategies. In this manner, patients are seen at the most appropriate level of care, reserving briefer and more targeted interventions for those within the primary care setting and more intensive treatments for those needing specialty services. This can ease burden on the healthcare system by treating mental and behavioral conditions promptly and allocating resources appropriately. This systemic structure also allows for PCPs to feel confident that their patients' behavioral health needs are being addressed while still being able to maintain their clinic schedules. IPCBH programs have been developed within both pediatric and adult primary care settings.

IPCBH Models of Care

Models of IPCBH have been developed dating back to the 1970s, although they were not widely adopted until decades later. Stemming from a growing demand for behavioral healthcare, numerous models have been developed, but none on the scale and scope of the Veterans Health Administration (VHA), the largest healthcare system in the United States. For that reason, this summary focuses on VHA IPCPH programs, which were implemented within medical centers across the nation using three primary models: Behavioral Health Laboratory (BHL) care management model (Tew, Klaus, & Oslin, 2010), White River Model of Colocated Collaborative Care (Pomerantz et al., 2010), and Translating Initiatives for Depression into Effective Solutions (TIDES). VHA medical centers were required to adopt one of these three models and then adapt them to fit within and meet the unique needs of their primary care context and patients. This has led to multiple variations of the below models being implemented and utilized in varying forms. By doing such, a person's health and well-being can be enhanced by interprofessional collaboration aimed at highlighting and intervening at the intersection between physical health, habits, and emotional health, with same-day access to behavioral health services being the norm.

The BHL is a care management model that was designed to help screen and treat common mental health conditions (depression, anxiety, addictions) in the primary care setting. It is a team-based approach involving the patient, the PCP, a care manager (typically a nurse, social worker, or psychologist), and a supervising psychiatrist. BHL staff provide support to PCPs, who are considered central to treatment planning. In particular, BHL staff assist with a structured patient identification process to determine those in need or at risk. Once a patient is identified, they are invited to complete a BHL initial assessment either in person or by telephone, during which patient education is provided and treatment recommendations are

made. Specialized software provides BHL staff with decision support logic, clinical reports, and patient tracking. Recommendations can include care management, prevention and “watchful waiting” (i.e., to determine if symptoms will progress), specialty care services, or no treatment. As needed, BHL staff provide patient-centered services delivered through virtual clinics, telephone assessments, and in-person services. Further, they provide education, behavioral activation (e.g., addressing ambivalence about change and developing an action plan), and assistance with decision making by both patients and providers. Brief evidence-based interventions (e.g., for alcohol misuse, depression/anxiety care management, and monitoring of response to new antidepressants) are also provided as indicated. Throughout this process, care managers work collaboratively with the PCP to attend to the patient’s behavioral health needs.

The White River Model of Colocated Collaborative Care is an open-access model in which patients are seen within primary care on a walk-in basis in order to improve access and reduce wait times. Staffing includes a therapist (psychologist or masters level), psychiatrist or advanced practice nurse, and a clerk. Importantly, staff are co-located directly within primary care space in order to facilitate warm handoffs from the PCP or discussion of patient needs. Patients can self-refer for services or be identified through screening processes, their PCP, or specialty providers. Once identified, the clerk gives the patient an electronic touchpad to complete self-report measures to screen for common presenting concerns (e.g., depression, posttraumatic stress disorder, anxiety, and health and well-being) that can inform the focus of the visit. The therapist then conducts a brief assessment to evaluate psychosocial history and the presenting problem and begins discussing treatment options. The psychiatrist or advanced nurse practitioner then conducts a medical evaluation to determine whether any medical conditions are contributing to the behavioral health symptoms, following which a treatment plan is developed and treatment initiated to include psychotherapy, medication, or both. Psychotherapy is delivered in individual and group formats (e.g., psychoeducation or brief cognitive behavioral therapy [CBT]) and is generally brief, although in keeping with the open-access model patients can opt to conduct sessions in coordination with their PCP visits over time. One week following initiation of the treatment plan, a nurse care manager contacts the patient by telephone to follow up to see how the patient is doing.

The TIDES model was developed in response to the need to help the large number of patients struggling with depression in the primary care settings, including suicide risk management. TIDES was developed using evidence-based quality improvement (EBQI) methods. EBQI is a quality improvement approach with the aim of translating research treatments into routine clinical practice. As with the other models, TIDES includes elements demonstrated to be useful in collaborative care models (i.e., care management, support of patient self-care, assessment and follow-up by care managers, collaboration between PCP and non-physician staff, and referral to specialty mental healthcare). A care manager and mental health specialist play key roles in collaboration with the PCP. The care manager is typically a nurse, given the need for this person to be at ease with monitoring antidepressant medications and medical comorbidities. The care manager conducts initial assessments to determine program eligibility, followed by education about depression for all patients, and behavioral activation. Within the medical record, there is a templated program that aids the provider with determining which patients should be referred for care management. The care manager then assists the PCP with management of depression within primary care, including up to six months of follow-up as needed, or referral to specialty mental health. The mental health specialist likewise plays a key role working with patients who have complex conditions, providing supervision to care managers, as well as delivering educational trainings for PCPs.

IPCBH Treatment Interventions

Because of the varied clinical presentations that occur in primary care settings, the IPCBH provider must have knowledge of a wide variety of health conditions and brief interventions. Conditions treated in IPCBH include those in the realms of mental health, lifestyle changes, and medical psychology. Patients presenting problems can include anxiety, posttraumatic stress disorder, depression, adjustment difficulties, role transitions, relationship problems, attention deficit hyperactivity disorder, grief and loss, dementia, bipolar disorder, personality disorders, and thought-related disorders. As described above, patients are triaged to the appropriate level of care dependent on availability and willingness. However, at times a patient with a serious mental illness may receive a brief intervention within an IPCBH setting. Lifestyle changes can include any issues that are getting in the way of a person's health or well-being, often involving smoking cessation, substance misuse, problems with sexual behavior, problem-solving strategies, chronic pain management, insomnia, weight management, or wanting to change behaviors related to interpersonal relationships. Medical conditions frequently addressed include but are not limited to diabetes, heart disease, hypertension, headaches, spinal cord injury, traumatic brain injury, medication adherence, preparation for stressful medical/medication procedures, age-related decline, adjustment to illness, or terminal illness. Patients are helped to consider and work on the relevant cognitive, behavioral, emotional, social, spiritual, and environmental aspects of their health conditions. Most chronic health conditions have a large behavioral contribution, rendering the role of psychological interventions in the prevention and treatment of such critical. Given the nature of the IPCBH model, it is important to remember that most IPCBH interventions are brief and targeted and involve multi-directional communication among the care team. Below, we describe general mental and behavioral health services offered and review the state of the science in behavioral interventions. Notably, although psychotropic medications are often prescribed by PCPs or IPCBH prescribers (e.g., psychiatrist, psychiatric mental health nurse practitioners), a full summary of such prescription practices is beyond the scope of this summary. Generally, IPCBH prescribers provide expert consultation to PCPs regarding medication management or prescribe medications themselves. IPCBH prescribers may see patients for one to six sessions, triaging them back to their PCP when stable or to specialty mental health if brief intervention/stabilization is unlikely.

Mental and behavioral health interventions are typically aimed at providing psychoeducation, helping patients increase motivation to change, setting adaptive goals, increasing psychological flexibility, decreasing maladaptive avoidance patterns, identifying and challenging irrational or unhelpful thinking patterns, making behavioral changes, clarifying values, coping with stress, and improving problem-solving skills. Patients are also often helped to adhere to medical treatments, engage in appropriate self-care strategies, and best utilize their primary care team. The 5A's framework (Hunter, Goodie, Oordt, & Dobmeyer, 2016) is frequently utilized and is a conceptual model where a therapist will Assess, Advise (options for treatment), Agree (on goals), Assist (in moving toward goals), and Arrange for follow-up. As with most psychotherapy relationships, the core components of empathy, rapport building, alliance, and positive regard are present in IPCBH interventions.

The state of the science for brief interventions being utilized within IPCBH models is in its early stages of development and varies greatly across interventions for differing problems. Several behavioral health interventions have been adapted for use within primary care settings, including depression, anxiety disorders, posttraumatic stress disorder, and health psychology interventions (e.g., chronic pain, sleep, obesity, diabetes, smoking cessation, etc.). Treatments are primarily adapted to fit the briefer models necessary within a primary care context. As these treatments are

developed, there is a push to begin empirical validation of their utility based on the current zeitgeist valuing empirically supported treatments. Many treatments that have been or are being adapted for brief models emanate from CBT, acceptance and commitment therapy, solution-focused/problem-solving therapy, and motivational interviewing. These types of interventions are strong candidates for being adapted for IPCBH as they have an established evidence base when implemented in traditional therapy modalities and are well suited to be modified to fit a brief model. Specifically, certain components of larger treatment protocols are efficacious when used in a dismantled approach (e.g., stimulus control or sleep restriction from a full CBT manual for insomnia). When considering the state of the science for brief interventions in primary care, another challenge is that it is not uncommon for providers and the subsequent research evaluating the interventions to have been conducted with people who meet full diagnostic criteria for disorders, which is not necessarily the population IPCBH models are intended to treat. In an extensive literature review, the Center for Integrated Health's White Paper on embedded behavioral health providers in VA IPCBH concluded that the state of the research on co-located collaborative care is primarily quasi-experimental or in the form of observational studies of programs (Beehler et al., 2015). However, interventions targeting tobacco use and alcohol use have a strong evidence base and can be implemented in a manner consistent with primary care clinical practice. The Society of Clinical Psychology's website section of psychological treatments (<https://www.div12.org/psychological-treatments/>) is a reputable resource that provides information and categorizes the strength of research regarding interventions for specific disorders and currently reflects the state of the research described above. Further research is clearly indicated to foster adoption of IPCBH programs and optimize patient care practices.

In addition to delivering care in person and via telephone, IPCBH interventions are also being delivered via emerging technologies in efforts to decrease barriers to care and reach underserved populations (e.g., those in rural communities). These interventions are typically delivered by what is commonly referred to as clinical video telehealth (CVT). Typically included in CVT encounters are a service provider(s), a service recipient(s), Internet connections of reasonable bandwidth, a software interface that allows for secure connections, and hardware on both ends of the connection to facilitate communication and run the software (e.g., computer, tablet, smartphone, speakers, microphone, webcam technology). Both individual and group sessions can be facilitated and conducted across multiple locations in synchrony. Services can be provided across states lines (theoretically globally) and in a myriad of conglomerations regarding where the provider and recipients are located (e.g., medical center to medical center, medical center to home, home to home, home to medical center). It is worth noting that the legal and ethical aspects of such service delivery are still being established as technology advances.

Within VHA, IPCBH via CVT is being more widely offered in two basic modalities. First, it is being utilized by providers who work traditional IPCBH roles in brick-and-mortar VA settings. These providers typically see veterans in person and then implement CVT as clinically indicated with veterans as needed. Second, Boise VA is rolling out a new telehealth model of healthcare designated as Virtual Integrated Multisite Patient Aligned Care Team (V-IMPACT; for full review see Ambert-Pompey, Konecky, Ahlstrom, & Keefer, 2017). Within V-IMPACT, mental and behavioral health services are implemented through IPCBH. Brief CVT assessment and interventions are provided by psychologists, neuropsychologists, social workers, and psychiatric prescribers (i.e., psychiatrists, psychiatric nurse practitioners) who work in tandem with other staff (e.g., nurse, support assistants, telehealth clinical technician) in distant locations. In this manner, CVT can augment traditional IPCBH practices by facilitating broader outreach and accessibility of behavioral health interventions.

References

- Ambert-Pompey, S., Konecky, B., Ahlstrom, D., & Keefer, A. (2017). Improving access: Team-based primary care via telehealth in the VA. *SGIM Forum*, 40(10).
- Beehler, G. P., Funderburk, J. S., King Jr., P. R., Johnson, E. M., Lilienthal, K., Maisto, S. A., ... Wray, L. O. (2015). *The role and functions of embedded behavioral health providers in VA primary care-mental health integration: Current evidence and future directions for research*. A VA Center for Integrated Healthcare White Paper.
- Hunter, C., Goodie, J. L., Oordt, M. S., & Dobmeyer, A. C. (2016). *Integrated behavioral health in primary care* (2nd ed.). Washington, DC: American Psychological Association.
- Pomerantz, A. S., Shiner, B., Watts, B. V., Detzer, M. J., Kutter, C., Street, B., & Scott, D. (2010). The White River model of colocated collaborative care: A platform for mental and behavioral health care in the medical home. *Families, Systems, & Health*, 28, 114–129. doi:10.1037/a0020261
- Tew, J., Klaus, J., & Oslin, D. W. (2010). The behavioral health laboratory: Building a stronger foundation for the patient-centered medical home. *Families, Systems, & Health*, 28, 130–145. doi:10.1037/a0020249

Suggested Reading

- Augustyn, M. C., Caronna, E., & Zuckerman, B. S. (2010). *The Zuckerman Parker handbook of developmental and behavioral pediatrics for primary care* (3rd ed.). Philadelphia, PA: Lippincott Williams and Wilkins Publishing.
- DiTomasso, R. A., Golden, B. A., & Morris, H. J. (2010). *Handbook of cognitive-behavioral approaches in primary care*. New York, NY: Springer Publishing Company.
- Hunter, C., Goodie, J. L., Oordt, M. S., & Dobmeyer, A. C. (2016). *Integrated behavioral health in primary care* (2nd ed.). Washington, DC: American Psychological Association.
- Robinson, P. J., Gould, D. A., & Strosahl, K. D. (2010). *Real behavior change in primary care*. Oakland, CA: New Harbinger Publishing.
- Robinson, P. J., & Reiter Jeffrey, T. (Eds.) (2016). *Behavioral consultation and primary care: A guide to integrating services* (2nd ed.). New York, NY: Springer International Publishing.
- Strosahl, K. D., Robinson, P. J., & Gustavsson, T. (2012). *Brief interventions for radical change*. Oakland, CA: New Harbinger Publishing.

Neuroimaging

Steven M. Nelson

*VISN 17 Center of Excellence for Research on Returning War Veterans, Waco, TX, USA
Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA
Center for Vital Longevity, School of Behavioral and Brain Sciences,
University of Texas at Dallas, Dallas, TX, USA*

The Beginnings of Neuroimaging

Approximately 30 years ago, one of the first human neuroimaging experiments was published (Petersen, Fox, Posner, Mintun, & Raichle, 1988; Posner, Petersen, Fox, & Raichle, 1988). Researchers used a technique called positron emission tomography (PET) to map locations in the brain that were more active during various language tasks than when the participant was lying still staring at an image of a crosshair. Though revolutionary at the time, these findings represented only a first step in our understanding of how to think about cognition and the human brain by asking a very basic question—“where in the brain do these cognitive processes happen?”

It was not long until the field began to rely less heavily on PET, turning to a technique called functional magnetic resonance imaging (fMRI), which both avoided the need to expose participants to ionizing radiation and promised greater temporal and spatial resolution. In the beginning, fMRI was used much like PET in that participants performed cognitive tasks in a blocked manner, responding to stimuli multiple times over the course of 2 or 3 min. Researchers would then average the signal across that period of time to get an estimate of brain activity. However, not long after, researchers began to wonder if they could estimate brain activity on each separate trial. After all, the assumption that all responses within a block were uniform was surely inaccurate, as participants were certainly responding more quickly during some trials or perhaps answering incorrectly. In 1996 and 1997, two seminal papers (Boynton, Engel, Glover, & Heeger, 1996; Dale & Buckner, 1997) were published that set guidelines for how

fMRI could be used to detect activity at the single-trial level. The details that led to this conclusion are beyond the scope of this chapter, but suffice it to say that the field of cognitive neuroscience had now opened up a whole new set of questions, unlocked by analytical advances made in a very short time span.

In 1998, researchers from Harvard (Wagner et al., 1998) and Stanford (Brewer, Zhao, Desmond, Glover, & Gabrieli, 1998) published a pair of studies that asked the question, “can we predict whether or not a word or picture will be remembered in the future, based on brain activity witnessed at an earlier time?” The answer was a resounding yes and led to the proliferation of what are termed event-related fMRI experiments. The discovery of the neural correlates of cognitive events, captured in single trials, has led to a deeper understanding of how attention, learning, memory, emotion, decision making, language, and a whole host of other domains are instantiated in the human brain.

The Advent of Functional Connectivity

Fast-forward another 5–7 years to two papers published in the *Proceedings of the National Academy of Sciences* (PNAS) (Fox et al., 2005; Greicius, Srivastava, Reiss, & Menon, 2004) that would launch what is still today the most widely researched single topic in fMRI—a technique known as resting-state functional connectivity MRI (RSFC). Instead of measuring discrete brain activity time-locked to an event or stimulus, RSFC measures correlations of slow, or low-frequency, brain activity across regions. For some time, neuroscientists were skeptical that this was a useful technique and instead decided that these slow waves of activity were probably just noise. However, researchers became more convinced of the utility of RSFC because the correlations between brain regions appeared to recapitulate known sets of regions that would typically be deployed in a task setting (Power et al., 2011). In other words, regions that co-activate when performing a task also tend to co-fluctuate in the absence of any overt task. RSFC is a particularly powerful technique because it (a) allows for the identification of all major brain systems with one scan, without having to create a large battery of tasks; (b) is thought to represent the statistical history of brain function; (c) enables study of populations that cannot perform complex tasks; and (d) is not confounded by group differences in task performance. Why hadn’t anyone noticed this phenomenon until now? After all, a great deal about sensory and motor brain systems was understood even before PET and fMRI, and interrogation of these regions in these systems was possible. The simple answer is that someone did notice. In 1995, Bharat Biswal and colleagues (Biswal, Yetkin, Haughton, & Hyde, 1995) showed that low-frequency correlations were present in the motor system of human individuals in the absence of an explicit task. The brain maps showed clear representation of the motor systems not just in the cerebral cortex but also in subcortical structures and the cerebellum. So why did it take so long for RSFC research to become so mainstream on the order of 8–10 years after Biswal’s finding? The answer is likely complex and by no means singular. Perhaps the transition from blocked to event-related design had gotten sufficient attention from the field to keep researchers busy enough asking about cognitive tasks. Perhaps it was not until the early twenty-first century that neuroscientists recognized the promise of RSFC for clinical populations. No matter the reason, the notion that an idea or technique that does not get immediately picked up in any scientific domain does not necessarily spell the end for that line of research. Instead, the adoption of techniques and the ability for ideas to take center stage must sometimes wait their turn until they are championed under the right circumstances.

Is Functional Connectivity Modifiable?

In 2009 and 2010, three papers (Lewis, Baldassarre, Committeri, Romani, & Corbetta, 2009; Stevens, Buckner, & Schacter, 2010; Tambini, Ketz, & Davachi, 2010) were published that addressed whether the correlations between brain regions could be changed with an intervening task tapping learning or attentional processes. The underlying assumption drawn from studies showing the one-to-one correspondence between systems discovered with functional connectivity and those discovered during task-evoked studies was that functional connectivity followed Hebbian principles. Instead of the “fire together, wire together” rule put forth by Hebb (Hebb, 1949), the concept as it pertains to RSFC is that regions that co-activate during tasks co-fluctuate at rest. It would stand to reason, then, that researchers should be able to induce stronger correlations by having participants engage in tasks where specific regions are known to co-activate. Indeed, this has now been consistently shown in the literature, with the field moving from proof-of-principle studies to experiments that aim to discover which regions or networks of regions are modified by a task or intervention. This discovery-based approach lends itself to fMRI studies with a high degree of clinical relevance, because scientists can ask about treatment interventions that have demonstrated success and determine potential mechanisms of brain plasticity. For instance, a study by Liston and colleagues (Liston et al., 2014) examined RSFC patterns in the brains of patients with and without depression, treated the depressed patients using repetitive transcranial magnetic stimulation (rTMS), and asked what brain networks were affected. More specifically, which RSFC patterns that were abnormal before treatment with rTMS looked like the patients without a depression diagnosis? The study found that modulation of connectivity with the subgenual cingulate cortex, a region previously implicated in depression, was one potential mechanism by which rTMS may act to alleviate symptoms.

Functional Connectivity and Health Psychology

Interestingly, although the field of health psychology has not adapted behavioral studies to determine whether the modification of specific brain systems underlie things like physical health, within-subject RSFC designs lend themselves well to this topic of study. For instance, a plethora of research has focused on links between exercise, wellness, and brain function (Geda et al., 2010), asking whether exercise can mediate the effect of aging on cognitive impairment. However, current RSFC studies have not manipulated variables like exercise and ask whether increased (or decreased) exercise causes a change in brain organization. Like other studies focusing on psychiatric disorders, mechanisms of action can be elucidated with such a design because of its interventional nature. Instead, the marriage of health psychology and fMRI has existed inside studies that use brain activity at one timepoint to predict health outcomes related to weight gain (Demos, Kelley, & Heatherton, 2011) or the propensity to engage in behaviors that impact physical health, like smoking (Wagner, Dal Cin, Sargent, Kelley, & Heatherton, 2011). The shift from prediction to within-subject intervention would represent a movement toward a less correlative, more causal scientific framework.

Perhaps the biggest contribution of functional connectivity to the field of health psychology is in the realm of chronic pain, as syndromes such as chronic back pain (CBP), fibromyalgia, diabetic neuropathy, and complex regional pain syndrome (CRPS) have been examined (Thorpe et al., 2018). Studies of chronic pain have shown consistently that regions of insular cortex are abnormal in patients who deal with a variety of related pain disorders. As such, this brain

region, and specifically the extent to which it is functionally connected to other regions that are part of its network in healthy adults, can help to provide a proxy for diagnostics and an understanding of how therapy may modify and strengthen these networks. As we will argue in the next section, the degree to which we can map specific brain regions in the insula onto individual anatomy will determine the degree of precision with which we can make claims and ultimately decide whether a relationship between insula connectivity and pain symptoms can move the field forward to personalized medicine approaches in chronic pain.

How to Make fMRI More Clinically Relevant to Health Psychology

Since its inception, fMRI has held considerable promise as a technique that could revolutionize how we think about and treat psychiatric disorders. However, it is all too clear that little progress has been made in advancing our understanding of how mental health abnormalities translate to brain abnormalities and how that information can inform treatment. A key roadblock toward success may stem from an over-reliance on experiments that ask how two groups differ from one another. Although sampling from large numbers of individuals to determine the average of some brain-based measure is appealing when trying to estimate differences in central tendencies between groups, what often gets lost are idiosyncratic features of data in specific individuals. We know this is the case when, for instance, a group of patients with posttraumatic stress disorder (PTSD) have a wide range of scores on measures that tap different clusters of symptoms. To some degree, this kind of variance may be unavoidable. However, when we map RSFC or activation data onto a group average brain template, as is standard in most fMRI research, neuroscientists also run the risk of distorting *where* differences exist among individuals in an attempt to describe central tendencies of connectivity. In other words, when we add fMRI to the mix, another layer of data must be contorted in order to perform appropriate statistical comparisons in “brain space,” but this comes at a significant cost because functional anatomy is highly heterogeneous. So how do we overcome this added layer of distortion? The answer is to collect far more data from the individual patient than the vast majority of the field currently does in order to greatly reduce within subject variance in data like RSFC so that functional data can be mapped to the patient’s own anatomy (Gordon et al., 2017, 2018; Laumann et al., 2015; Poldrack et al., 2015). The famous neurophysiologist William T. Newsome once said, “in order to understand brain function, we must respect the integrity of cortical organization.” The same can be said in our attempt to understand brain disorders. That is, each individual brain has a specific structural and functional organization that should not be averaged together to arrive at a difference in central tendencies. Instead, in order to make fMRI and human neuroscience clinically relevant, we need to embrace discoveries we make at the individual level, even if at first we do not understand them or we are slogging through data collection because of the increased labor that goes into one participant. This highly sampled individual-based approach provides our best chance of mapping brain to behavior and in the end is likely to guide precision treatments that are tailored to specific patients.

Author Biography

Steven M. Nelson received his bachelor’s degree in psychology and mathematics from Winona State University (Winona, MN). We went on to receive his PhD in Neuroscience at Washington University in St. Louis, where he stayed on as a post-doctoral scholar and research scientist.

He is currently the chief of the Neuroimaging Core at the VISN 17 Center of Excellence for Research on Returning War Veterans, where he uses functional MRI to study veterans with post-traumatic stress disorder and traumatic brain injury.

References

- Biswal, B., Yetkin, F. Z., Haughton, V. M., & Hyde, J. S. (1995). Functional connectivity in the motor cortex of resting human brain using echo-planar MRI. *Magnetic Resonance in Medicine*, 34, 537–541.
- Boynton, G. M., Engel, S. A., Glover, G. H., & Heeger, D. J. (1996). Linear systems analysis of functional magnetic resonance imaging in human V1. *Journal of Neuroscience*, 16, 4207–4221.
- Brewer, J. B., Zhao, Z., Desmond, D. E., Glover, G. H., & Gabrieli, J. D. E. (1998). Making memories: Brain activity that predicts how well visual experience will be remembered. *Science*, 281, 1185–1187.
- Dale, A. M., & Buckner, R. L. (1997). Selective averaging of rapidly presented individual trials using fMRI. *Human Brain Mapping*, 5, 329–340.
- Demos, K. E., Kelley, W. M., & Heatherton, T. F. (2011). Dietary restraint violations influence reward responses in nucleus accumbens and amygdala. *Journal of Cognitive Neuroscience*, 23, 1952–1963.
- Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., Van Essen, D. C., & Raichle, M. E. (2005). The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proceedings of the National Academy of Sciences of the United States of America*, 102, 9673–9678.
- Geda, Y. E., Roberts, R. O., Knopman, D. S., Christianson, T. J., Pankratz, V. S., Ivnik, R. J., ... Rocca, W. A. (2010). Physical exercise, aging, and mild cognitive impairment: A population-based study. *Archives of Neurology*, 67, 80–86.
- Gordon, E. M., Laumann, T. O., Gilmore, A. W., Newbold, D. J., Greene, D. J., Berg, J. J., ... Hampton, J. M. (2017). Precision functional mapping of individual human brains. *Neuron*, 95, 791–807. e797
- Gordon, E. M., Scheibel, R. S., Zambrano-Vazquez, L., Jia-Richards, M., May, G. J., Meyer, E. C., & Nelson, S. M. (2018). High-fidelity measures of whole-brain functional connectivity and white matter integrity mediate relationships between traumatic brain injury and post-traumatic stress disorder symptoms. *Journal of Neurotrauma*, 35, 767–779.
- Greicius, M. D., Srivastava, G., Reiss, A. L., & Menon, V. (2004). Default-mode network activity distinguishes Alzheimer's disease from healthy aging: evidence from functional MRI. *Proceedings of the National Academy of Sciences of the United States of America*, 101, 4637–4642.
- Hebb, D. O. (1949). *The organization of behavior*. New York, NY: Wiley.
- Laumann, T. O., Gordon, E. M., Adeyemo, B., Snyder, A. Z., Joo, S. J., Chen, M. Y., ... Schlaggar, B. L. (2015). Functional system and areal organization of a highly sampled individual human brain. *Neuron*, 87, 657–670.
- Lewis, C. M., Baldassarre, A., Committeri, G., Romani, G. L., & Corbetta, M. (2009). Learning sculpts the spontaneous activity of the resting human brain. *Proceedings of the National Academy of Sciences of the United States of America*, 106, 17558–17563.
- Liston, C., Chen, A. C., Zebly, B. D., Drysdale, A. T., Gordon, R., Leuchter, B., ... Dubin, M. J. (2014). Default mode network mechanisms of transcranial magnetic stimulation in depression. *Biological Psychiatry*, 76, 517–526.
- Petersen, S. E., Fox, P. T., Posner, M. I., Mintun, M., & Raichle, M. E. (1988). Positron emission tomographic studies of the cortical anatomy of single-word processing. *Nature*, 331, 585–589.
- Poldrack, R. A., Laumann, T. O., Koyejo, O., Gregory, B., Hover, A., Chen, M. Y., ... Hunicke-Smith, S. (2015). Long-term neural and physiological phenotyping of a single human. *Nature Communications*, 6, 8885.
- Posner, M. I., Petersen, S. E., Fox, P. T., & Raichle, M. E. (1988). Localization of cognitive operations in the human brain. *Science*, 240, 1627–1631.

- Power, J. D., Cohen, A. L., Nelson, S. M., Vogel, A. C., Church, J. A., Barnes, K. A., ... Petersen, S. E. (2011). Functional network organization in the human brain. *Neuron*, 72, 665–678.
- Stevens, W. D., Buckner, R. L., & Schacter, D. L. (2010). Correlated low-frequency BOLD fluctuations in the resting human brain are modulated by recent experience in category-preferential visual regions. *Cerebral Cortex*, 20, 1997–2006.
- Tambini, A., Ketz, N., & Davachi, L. (2010). Enhanced brain correlations during rest are related to memory for recent experiences. *Neuron*, 65, 280–290.
- Thorp, S. L., Suchy, T., Vadivelu, N., Helander, E. M., Urman, R. D., & Kaye, A. D. (2018). Functional connectivity alterations: Novel therapy and future implications in chronic pain management. *Pain Physician*, 21, E207–E214.
- Wagner, A. D., Schacter, D. L., Rotte, M., Koustaal, W., Maril, A., Dale, A. M., ... Buckner, R. L. (1998). Building memories: Remembering and forgetting of verbal experiences as predicted by brain activity. *Science*, 281, 1188–1191.
- Wagner, D. D., Dal Cin, S., Sargent, J. D., Kelley, W. M., & Heatherton, T. F. (2011). Spontaneous action representation in smokers when watching movie characters smoke. *The Journal of Neuroscience*, 31, 894–898.

Palliative Care

Mamta Bhatnagar and Robert C. Arnold

Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Definition

Palliative care is both a model of care and a specialty focusing on improving quality of life for patients and family with serious illness. It focuses on providing patients with relief from symptoms, pain, and stress of a serious illness—whatever the diagnosis. The goal is to improve quality of life for both the patient and the family. It is appropriate at any age and at any stage of a serious illness. It can be provided at the same time as disease treatment to help people live as well as possible while facing illness (Center to Advance Palliative Care, n.d.).

Psychology and psychologists are an integral part of the palliative care team. During progression of a serious illness, patients and family members have to adjust significantly to the reality of care focused on disease and symptom management without a cure. Psychological concepts such as resilience, coping, grief, anxiety, and depression are thus central to the work of palliative care (Patterson, Croom, Teverovsky, & Arnold, 2014). Psychologists can provide support and psychosocial interventions to assist with anticipatory grief and adjustment reactions, mental disorders, existential and spiritual issues, advance care planning, life review, and unresolved issues that are likely to surface as meaningful concerns (Haley, Larson, Kasl-Godley, Neimeyer, & Kwilosz, 2003).

Models for Palliative Care Delivery and Their Growth in the United States

In the United States much of the growth in specialty palliative care has been in the inpatient setting. This has been fueled by data showing cost avoidance as well as nonrandomized trials that suggest improved patient satisfaction and outcomes (Cassel, Kerr, Pantilat, & Smith, 2010; Morrison et al., 2008, 2011; Norton et al., 2007). The number of hospitals with

palliative care teams increased from 658 to 1,744 between 2000 and 2013 (a growth of 165%) (Dumanovsky et al., 2016). There is however substantial variability in the availability of palliative care throughout the United States. For example, the presence of palliative care team is significantly influenced by geography, hospital size, hospital type (academic medical centers, faith-based institutions), and tax status (for profit versus not for profit) (Dumanovsky et al., 2016). All VA hospitals and over 67% hospitals with more than 50 beds provide palliative care (Spetz et al., 2016).

Hospital-based palliative care consultation is provided by an interprofessional team that can include physicians (including generalists and specialists, as well as psychiatrists), advanced practitioners (nurse practitioners and physician assistants) and nurses, social workers, psychologists, chaplains, pharmacists, and volunteers. A study exploring staffing of palliative care programs registered with the National Palliative Care Registry found only a quarter of the programs had a fully funded team with chaplains and social workers being the most common member who was voluntary or absent. A fully staffed palliative program provides better hospital penetration (percentage of patients who receive a palliative care consultation) and is better able to meet the needs of seriously ill patients with more timely consultations (Spetz et al., 2016). The goals of the palliative care team are to provide expert pain and symptom management, education and communication about achievable goals for care, support for decisions matched to patient and family goals, psychosocial support, and coordination of care. The goal of the consultation service is to support the referring clinician.

While the dominant model for specialty palliative care services is a consultant model, many programs also have a palliative care unit. In the National Palliative Care Registry, 5% of programs had dedicated palliative care units, typically found in larger and more mature programs (Spetz et al., 2016). Palliative care units usually serve patients who have difficult-to-control symptoms; medical needs that cannot be optimally managed in another setting; distressed families in need of a higher level of support; need for transfer out of a critical care setting; patients who are imminently dying. A high-volume palliative care unit may reduce in-hospital care costs by matching treatments provided to patient and family-determined goals for medical care. In one study, a dedicated palliative care unit reduced daily hospital costs by 74% compared with usual care patients (Smith et al., 2003).

Few studies have compared quality of care among different palliative care models. In a phone survey of US Veterans Administration (VA) medical centers that offered both models of inpatient care, family members of patients in a palliative care unit were significantly more likely to report excellent care in the last month of life when compared with families of patients who received a palliative care consultation (Casarett, Johnson, Smith, & Richardson, 2011). The palliative care unit group also scored highest in three of four process measures (do not resuscitate order at the time of death, documented chaplain visit, and bereavement contact after the patient's death).

Bringing palliative care into the emergency department (ED) has also been found to be beneficial. Recently a randomized controlled trial (RCT) of palliative care consultation for patients with advanced cancer showed improved quality of life metrics and median estimated survival (Kavalieratos et al., 2016). Additionally, patients with non-cancer diagnoses such as dementia, congestive heart failure (CHF), and chronic obstructive pulmonary disease (COPD) also benefit from palliative care consultations in the ED with one study showing that these patients had shorter lengths of hospital stay (Wu, Newman, Lasher, & Brody, 2013). Currently, palliative care services in the ED are usually consultations by an inpatient palliative care team. A more comprehensive approach also involves services or training initiated by palliative care

champions in the ED and ED partnerships with hospice providers. The nature of the palliative care intervention across randomized trials has been difficult to compare; however nonrandomized trials continue to show benefit from various ambulatory palliative care services (M. Rabow et al., 2013). A recent meta-analysis of 43 RCTs, of which 14 were conducted in the ambulatory setting, examined whether palliative care interventions improve quality of life, symptom burden, healthcare utilization, and patient/caregiver satisfaction. Palliative care was associated consistently with improvements in advance care planning, patient and caregiver satisfaction, and lower healthcare utilization along with statistically significant improvement in quality of life (Kavalieratos et al., 2016).

Ambulatory palliative care programs focus on delivering care aimed at better quality, improved access, and lower costs. Unlike inpatient consultation teams, ambulatory programs tend to have a wide range of structures. They can be consultative clinics in which specialty palliative care teams take referrals from other providers either as stand-alone clinic that is able to take referrals from different specialties or embedded programs within a specialty service. While the benefits of early palliative care consultations in clinics are being increasingly recognized (see below), the growth of outpatient palliative care programs has been less robust. A survey of cancer centers across America found that the ones designated by the National Cancer Institute (NCI) were more likely to have outpatient palliative care services, specifically 59% as compared with 22% in nondesignated centers (Hui et al., 2010).

There is strong evidence supporting outpatient palliative care programs providing early palliative care. Notable studies include outpatient consultations for patients with advanced cancer that showed improvement in fatigue, pain, depression, anxiety, and sleep after at least one initial visit (Yennurajalingam et al., 2012). Also, a widely publicized study from Temel and colleagues showed improved quality of life and depression as well as 2.7 months' increased survival among patients with metastatic non-small cell lung cancer (Temel, Greer, & Muzikansky, 2010). Similar benefits have also been reported in patients with advanced COPD and CHF (M. W. Rabow, Dibble, Pantilat, & McPhee, 2004).

Other models that have been explored for delivering palliative care are in nursing homes and in home-based primary care programs and collaborative care models in the emergency department. In nursing homes, specialty palliative care can be delivered by hospice organizations, external palliative care consultation team, or an internal palliative care team. The consultative team still comprises a physician accompanied by either a nurse or an advance practice provider. Formal palliative care services in the nursing home are rare because regulatory, payment, and staffing barriers prevent implementation (Meier, Lim, & Carlson, 2010). One study showed improved quality of care with palliative care consult, reducing ER visits and depression, and promoted more appropriate care resulting in more favorable clinical outcomes at the end of life (EOL) (Comart et al., 2013). Palliative care teams in the nursing home can provide valuable education to nursing home staff on recognition of adverse prognostic signs among the frail elderly and incorporating palliative care skills into their care.

Home palliative care models are also evolving. Successful home programs such as the Sutter Health Program of Advanced Illness Management in California have shown an increase in hospice referrals up to 60%, 54% reduction in hospital admissions, 80% reduction in ICU days, and 26% reduction in inpatient length of stay (Ciemens, Stuart, Gerber, Newman, & Bauman, 2006). As accountable care organizations (ACO) continue to grow in the United States, there is a high likelihood that more integrated models of palliative care throughout the care continuum will emerge.

Hospice as a System of Provision of Palliative Care at the EOL in the United States

Although the philosophy of palliative care is nearly congruent with that of hospice care, the scope of hospice care is not identical because it differs in terms of both timing and types of patients served. While both improve quality of life, palliative care is available to patients who continue to benefit from life-prolonging treatments. Access to palliative care is not dependent on prognosis, and there are no federal or commercial insurance benefits specific to palliative care with no formal eligibility criteria.

Hospice is both a philosophy of care and a regulated insurance benefit. The Medicare hospice benefit was enacted by the US Congress in 1982 and is the dominant source of payment for hospice care; 84% of patients who receive hospice services in the United States are covered by Medicare. Patients are eligible to receive hospice care reimbursed under the Medicare hospice benefit if they meet the following criteria: (a) the individual has a life expectancy of 6 months or less if the disease follows its expected course, as certified by two physicians, and (b) the individual forgoes Medicare reimbursement for ongoing therapy or curative medical treatment related to the terminal diagnosis. While there are some general guidelines for certifying the presence of a terminal illness that are applicable to all diseases, there are also specific disease-related guidelines. For example, the hospice eligibility criteria for ALS are vital capacity less than 30%, critical nutritional impairment, and life-threatening complications. The presence of any two of these three can qualify a patient for the Medicare hospice benefit for ALS. While these guidelines are helpful, they are based on clinical opinion rather than data (McCluskey & Houseman, 2004).

Since the first program was established in the United States in 1982, it has grown rapidly. Currently there are 6,100 programs in the country, mostly independent agencies. They vary in size; some hospices serve only 50 patients per year to large national corporate chains that care for thousands of patients each day. They also differ in their for-profit status with only about 28% holding not-for-profit tax status. Simultaneously, the number of patients served by hospice has increased from 25,000 in 1984 to almost 1.4 million in 2015 (National Hospice and Palliative Care Organization, n.d.).

Federal regulations require a core of hospice services be provided by all hospice providers. Core services comprise skilled nursing, physician services, volunteer services, counseling services that include bereavement counseling, spiritual care, dietary, and social services. Services that can be outsourced by hospice providers include physical therapy, occupational therapy, speech language pathology, home healthcare, and homemaker services.

Generalist vs. Specialist Palliative Care

With growing recognition of the benefit of palliative care to seriously ill patients, there is an increasing demand for palliative care services to be provided to all patients with life-threatening illnesses. Although this demand has fostered rapid growth of the palliative care specialty, the current model adds another layer of specialized care for seriously ill patients on top of an already complex, expensive health environment. There is tremendous regional variation in the availability of palliative care—ranging from 20 to 100% across the United States.

Some core elements of palliative care, such as outlining treatment with the patient's goals and basic symptom management, should be routine aspects of care delivered by any practitioner. It is important for primary care providers and other existing specialist or generalist

clinicians to continue to address basic management of pain and symptoms and basic management of depression and anxiety and have basic discussions about prognosis, goals of treatment, suffering, and code status. Other skills such as communicating with difficult families or more refractory symptom management require specialized training and skills.

Most adult clinicians have to be able to perform core items of palliative care such as breaking bad news, handling pain, managing psychological and cognitive symptoms in seriously ill patients, and talking about advance directives. Sadly, practicing clinicians say they have not received adequate training to accomplish these tasks. To equip providers to provide primary palliative care, they need to be taught the primary palliative care curriculum in their training (Barzansky, Veloski, Miller, & Jonas, 1999). Center to Advance Palliative Care, VitalTalk, and the Harvard Serious Illness Conversation provide training for practicing clinicians. Similar courses are developing around the country, both in person and on the Internet, and focused on diseases such as heart failure, chronic lung disease, Alzheimer's disease, or other conditions. Creating a coordinated palliative care model that allows patients to receive basic palliative care from their primary care physician or treating specialist with a palliative care consultation being initiated for more complex or refractory problems reinforces delivery of primary palliative care by everyone caring for the seriously ill patient while leaving room for specialty palliative care consultation where appropriate (Quill & Abernethy, 2013).

Roles of Palliative Care

Symptom Management

Seriously ill patients often have a significant burden of symptoms. In a review of 400 patients admitted to palliative care in England, the 5 most prevalent symptoms were pain (64%), anorexia (34%), constipation (32%), weakness (32%), and dyspnea (31%). Ninety-five percent of these patients had a cancer diagnosis (Potter, Hami, Bryan, & Quigley, 2003). Similar prevalence of symptoms has been reported in patients with heart disease, COPD, renal disease, or acquired immunodeficiency syndrome. The top three symptoms are pain, breathlessness, and fatigue (Solano, Gomes, & Higginson, 2006). Palliative care strives to address all of these symptoms even as it takes into account some inherent differences between the cancer and non-cancer populations. Non-cancer populations tend to be older and follow different patterns of functional decline and dependency than patients suffering from cancer. A brief explanation of evidence-based management strategies employed by palliative care teams to alleviate symptoms of pain, dyspnea, and anorexia is presented below.

Pain

Pain is common among the patients with advanced cancer. The prevalence of moderate to severe pain in a group of advanced cancer patients referred to palliative care services was 51%, ranging from 43% in stomach cancer to 80% in gynecological cancers (Vainio & Auvinen, 1996). Data suggests that mature palliative care teams decrease severe pain in 66% of patients (Currow et al., 2014). They do this by using a combination of medications including opioids and nonopioid adjuvants, interventional pain techniques, radionuclide therapy, and complementary medicine. Using a multimodal strategy helps to decrease systemic opioid doses and address the different mechanisms by which pain can occur including neuropathic and inflammatory pathways.

Breathlessness

Both patients with COPD and patients with progressive cancer especially lung cancer can experience disabling dyspnea. Among advanced cancer patients, dyspnea can occur between 21 and 78.6% of patients and is reported to be from moderate to severe in 10–63% of the patients (Ripamonti, 1999). About 94% of patients with chronic lung disease experience dyspnea in the last year of life (Edmonds, Karlsen, Khan, & Addington-Hall, 2001). Dyspnea is also common in patients with progressive heart failure. One study of outpatients with heart failure found a rate of dyspnea of 55% (Blinderman, Homel, Billings, Portenoy, & Tennstedt, 2008); in other studies the numbers have been as high as 85–90% (Nordgren & Sorensen, 2003).

Palliative care interventions have been shown to improve dyspnea in a general medicine clinic in patients with chronic lung disease and CHF (Lorenz et al., 2008). Along with oxygen supplementation, opioids are often used to treat dyspnea in both patients with COPD as well as with cancer with studies showing improvement in the levels of dyspnea. Other clinical interventions that can be helpful include acupuncture in COPD (Suzuki et al., 2012) as well as targeted exercise program combined with behavioral therapy done at home (Higginson et al., 2014).

Gastrointestinal Symptoms

Nausea, vomiting, and retching are reported in up to 70% of patients with cancer and also occur frequently in non-cancer diagnoses such as CHF, acquired immunodeficiency virus, and hepatic and renal failure. Causation of these symptoms is frequently multifactorial including metabolic derangements, medications, changes in gastric and bowel motility, central nervous system disorders, and autonomic dysfunction. Part of the palliative care assessment is to obtain a careful history that would help to prescribe medications tailored to the suspected cause. Once probable causes are identified, environmental stimuli can be minimized, and antiemetic therapy that is targeted toward the probable cause of emesis/nausea is initiated (Wood, Shega, Lynch, & Von Roenn, 2007).

Cachexia and anorexia are commonly seen in the palliative care population in the setting of chronic progressive diseases that produce weight loss by a combination of loss of appetite, hypercatabolism and systemic inflammation in cancer, and muscle wasting. The estimated prevalence of cachexia ranges from 5 to 15% in advanced COPD or chronic heart failure to as much as 60–80% in advanced cancer (von Haehling, Morley, & Anker, 2010). Cachexia/anorexia requires multimodal management with priority given to a reversal of symptoms contributing to low dietary intake. Potentially correctable problems, for example, pain, dysphagia, nausea, mouth sores or thrush, and dry mouth, are identified and treated. Important psychological factors such as anxiety, depression, and spiritual distress are also carefully looked for and treated.

Discussing Goals of Care

Another important function of palliative care is to work with patients and families to determine their priorities for care and then to help structure subsequent care to best meet those values. Having such discussions in a timely manner allows patients to receive care that is consistent with their preferences, reduces caregiver distress, and increases the use of hospice while decreasing unwanted interventions like cardiopulmonary resuscitation (Apostol et al., 2015;

Mack et al., 2012; Mack, Weeks, Wright, Block, & Prigerson, 2010). Specialists in palliative care are specifically trained in communication skills and often teach others to have these conversations.

Having such conversations is hard, and many clinicians avoid these conversations because they worry about causing distress and taking away hope. However current evidence does not support the belief that communication about goals of care increases patient anxiety, depression, and/or hopelessness (Bernacki & Block, 2014). Early discussions about goals of care are associated with better quality of life, reduced use of nonbeneficial medical care near death, enhanced goal-consistent care, and positive family post-death outcomes (Bernacki & Block, 2014).

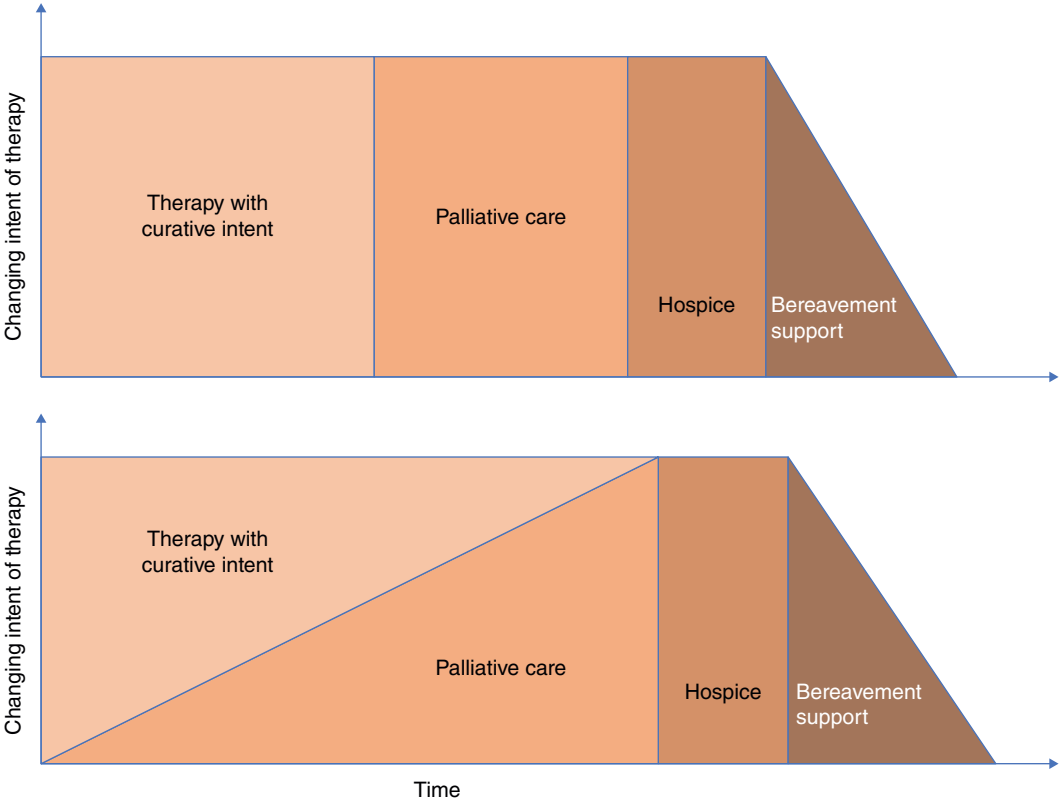
There are growing number of educational interventions to help clinicians develop their palliative care communication skills. Two of the most well known are the Serious Illness Communication Project (Bernacki & Block, 2014) and VitalTalk (n.d.). Common features among them include making sure that the patient understands their medical situation in clear, nonjargon language; attending to patient's emotion and fostering positive coping skills; and eliciting patient values to promote goal-concordant care.

Many of these programs teach mental models for how to give bad news (SPIKES), attend to emotion (NURSE), or talk about goals of care (REMAP). These are summarized in Table 1.

Specialist palliative care-patient interactions lead to improved quality of life, but until recently, how this occurred was unknown. Recent studies suggest that the reason for the positive impact is the effect palliative care specialists have on coping strategies. How one deals with poor prognostic data depends on the patient's coping strategy (Nipp et al., 2016). A secondary analysis of early integration of palliative care among patients with newly diagnosed incurable lung or gastrointestinal cancers found that patients in the palliative care arm used more approach-oriented coping strategies as compared with patients in the usual care group who used more avoidant coping strategies. This was associated with improved quality of life and fewer depressive symptoms (El-Jawahri et al., 2016).

Table 1 Mental models for difficult conversations.

<i>Model</i>	<i>Components</i>	<i>Examples</i>
SPIKES	Setting	Private setting with seating. All important participants present. Minimize interruptions
	Perception	What have you been told of your medical condition so far?
	Invitation	"Would you like me to talk about the treatment plan?"
	Knowledge	Provide information
	Empathy	Respond empathically (see NURSE)
NURSE	Summarize	Summarize patient's concerns and treatment plan
	Name	"This is hard"
	Understand	"I can't even imagine how difficult this must be for you"
	Respect	"You have done a really good job supporting your loved one"
	Support	"I will be available for you"
REMAP	Explore	"Tell me more about....."
	Reframe	Provide bad news using nonjargon language
	Empathize	Respond to emotion with empathic cues (see NURSE)
	Map	"What is important to you?" "What are your worries about the future?"
	Align	"What I have heard you say is...." "Is it ok if I talk about the plan?"
	Plan	"I would recommend"



Caring for the chronically ill individual along the disease continuum

Figure 1 Caring for the chronically ill individual along the disease continuum.

Conclusion

Palliative care is a model of care that focuses on improving quality of life for patients and families by providing relief from symptoms and advocating for care concordant with a patient’s wishes while also encouraging positive coping skills in the face of serious illness. Palliative care programs are growing across the country with special focus on ambulatory settings.

Author Biographies

Mamta Bhatnagar, MD, MS, is a clinical assistant professor of medicine in the Division of General Internal Medicine of the Department of Medicine at the University of Pittsburgh. She completed her medical school training in Lady Hardinge Medical College in Delhi, India, and residency at Case Western Reserve University in Ohio. She subsequently completed fellowships in Geriatrics at the Cleveland Clinic and in Palliative Care at Summa Health System in Akron, Ohio. She currently serves as a palliative care physician at UPMC cancer hospital where she also directs the palliative care services. Additionally, she is the medical director of the UPMC Health Plan home palliative care program.

Dr. Bhatnagar's key interests focus on transitions of care and how to enhance communication among providers across the care continuum. Her quality improvement team has published at the ASCO national meeting. Dr. Bhatnagar serves at the Quality Improvement Committee at AAHPM. She codirects Oncotalk, the annual training of all UPMC oncology fellows. In addition she has participated in several communication training courses throughout the UPMC health system teaching clinicians how to efficiently conduct goals of care discussions.

Robert C. Arnold, MD, is a distinguished professor of medicine in the Division of General Internal Medicine of the Department of Medicine at the University of Pittsburgh and in the University of Pittsburgh Center for Bioethics and Health Law. He completed his medical school training at the University of Missouri–Kansas City and residency at Rhode Island Hospital. Subsequently he has been on the faculty at the University of Pittsburgh. In 2000, Dr. Arnold was named the first Leo H. Creip Chair of Patient Care. The chair emphasizes the importance of the doctor–patient relationship, particularly at the end of life. He is the director of the Institute for Doctor–Patient Communication and the medical director of the UPMC Palliative and Supportive Institute. He is clinically active in palliative care. Dr. Arnold has published on end-of-life care, hospice and palliative care, doctor–patient communication, and ethics education. His current research interests are focused on educational interventions to improve communication in life-limiting illnesses and better understanding how ethical precepts are operationalized in clinical practice. He also is working with the UPMC Health System to develop system-wide, integrative palliative services throughout the health system. He is the past president of the American Society for Bioethics and Humanities as well as the American Academy of Hospice and Palliative Medicine.

References

- Apostol, C. C., Waldfogel, J. M., Pfoh, E. R., List, D., Billing, L. S., Nesbit, S. A., & Dy, S. M. (2015). Association of goals of care meetings for hospitalized cancer patients at risk for critical care with patient outcomes. *Palliative Medicine*, 29(4), 386–390. <http://doi.org/10.1177/0269216314560800>
- Barzansky, B., Veloski, J., Miller, R., & Jonas, H. S. (1999). Education in end-of-life care during medical school and residency training. *Academic Medicine*, 74, S102–S104. Retrieved from <http://131.211.208.19/login?auth=eng&url=http://ovidsp.ovid.com/ovidweb.cgi?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=psyc3&AN=1999-01388-023http://sfx.library.uu.nl/utrecht?sid=OVID:psycdb&id=pmid:&id=doi:10.1097%2F00001888-199910000-00054&issn=1040-2446&isbn=&cv>
- Bernacki, R. E., & Block, S. D. (2014). Communication about serious illness care goals. *JAMA Internal Medicine*, 174(12), 1994. <http://doi.org/10.1001/jamainternmed.2014.5271>
- Blinderman, C. D., Homel, P., Billings, J. A., Portenoy, R. K., & Tennstedt, S. L. (2008). Symptom distress and quality of life in patients with advanced congestive heart failure. *Journal of Pain and Symptom Management*, 35(6), 594–603. <http://doi.org/10.1016/j.jpainsymman.2007.06.007>
- Casarett, D., Johnson, M., Smith, D., & Richardson, D. (2011). The optimal delivery of palliative care. *Archives of Internal Medicine*, 171(7), 649–655.
- Cassel, J. B., Kerr, K., Pantilat, S., & Smith, T. J. (2010). Palliative care consultation and hospital length of stay. *Journal of Palliative Medicine*, 13(6), 761–767. <http://doi.org/10.1089/jpm.2009.0379>
- Center to Advance Palliative Care. (n.d.). About Palliative Care. Retrieved from <https://www.capc.org/about/palliative-care/>.
- Ciemics, E. L., Stuart, B., Gerber, R., Newman, J., & Bauman, M. (2006). An evaluation of the advanced illness management (AIM) program: Increasing hospice utilization in the San Francisco bay area. *Journal of Palliative Medicine*, 9(6), 1401–1411. <http://10.1089/jpm.2006.9.1401>

- Comart, J., Mahler, A., Schreiber, R., Rockett, C., Jones, R. N., & Morris, J. N. (2013). Palliative care for long-term care residents: effect on clinical outcomes. *The Gerontologist*, 53(5), 874–880. <http://doi.org/10.1093/geront/gns154>
- Currow, D. C., Allingham, S., Yates, P., Johnson, C., Clark, K., & Eagar, K. (2014). Improving national hospice/palliative care service symptom outcomes systematically through point-of-care data collection, structured feedback and benchmarking. *Supportive Care in Cancer*, 23(2), 307–315. <http://doi.org/10.1007/s00520-014-2351-8>
- Dumanovsky, T., Augustin, R., Rogers, M., Lettang, K., Meier, D. E., & Morrison, R. S. (2016). The growth of palliative care in U.S. hospitals: A status report. *Journal of Palliative Medicine*, 19(1), 8–15. <http://doi.org/10.1089/jpm.2015.0351>
- Edmonds, P., Karlsen, S., Khan, S., & Addington-Hall, J. (2001). A comparison of the palliative care needs of patients dying from chronic respiratory diseases and lung cancer. *Palliative Medicine*, 15(4), 287–295. <http://doi.org/10.1191/026921601678320278>
- El-Jawahri, A., LeBlanc, T., VanDusen, H., Traeger, L., Greer, J. A., Pirl, W. F., ... Temel, J. S. (2016). Effect of inpatient palliative care on quality of life 2 weeks after hematopoietic stem cell transplantation: A randomized clinical trial. *JAMA*, 316(20), 2094–2103. <http://doi.org/10.1001/jama.2016.16786>
- Haley, W. E., Larson, D. G., Kasl-Godley, J., Neimeyer, R. A., & Kwilosz, D. M. (2003). Roles for psychologists in end-of-life care: Emerging models of practice. *Professional Psychology: Research and Practice*. <http://doi.org/10.1037/0735-7028.34.6.626>
- Higginson, I. J., Bausewein, C., Reilly, C. C., Gao, W., Gysels, M., Dzingina, M., ... Moxham, J. (2014). An integrated palliative and respiratory care service for patients with advanced disease and refractory breathlessness: A randomised controlled trial. *The Lancet Respiratory Medicine*, 2(12), 979–987. [http://doi.org/10.1016/S2213-2600\(14\)70226-7](http://doi.org/10.1016/S2213-2600(14)70226-7)
- Hui, D., Elsayem, A., De la Cruz, M., Berger, A., Zhukovsky, D. S., Palla, S., ... Bruera, E. (2010). Availability and integration of palliative care at US cancer centers. *JAMA*, 303(11), 1054–1061. <http://doi.org/10.1001/jama.2010.258>
- Kavalieratos, D., Corbelli, J., Zhang, D., Dionne-Odom, J. N., Ernecoff, N. C., Hanmer, J., ... Schenker, Y. (2016). Association between palliative care and patient and caregiver outcomes. *JAMA*, 316(20), 2104. <http://doi.org/10.1001/jama.2016.16840>
- Lorenz, K. A., Lynn, J., Dy, S. M., Shugarman, L. R., Wilkinson, A., Mularski, R. A., ... Shekelle, P. G. (2008). Evidence for improving palliative care at the end of life: A systematic review. *Annals of Internal Medicine*. <http://doi.org/10.7326/0003-4819-148-2-200801150-00010>
- Mack, J. W., Cronin, A., Keating, N. L., Taback, N., Huskamp, H. A., Malin, J. L., ... Weeks, J. C. (2012). Associations between end-of-life discussion characteristics and care received near death: A prospective cohort study. *Journal of Clinical Oncology*, 30(35), 4387–4395. <http://doi.org/10.1200/JCO.2012.43.6055>
- Mack, J. W., Weeks, J. C., Wright, A. A., Block, S. D., & Prigerson, H. G. (2010). End-of-life discussions, goal attainment, and distress at the end of life: Predictors and outcomes of receipt of care consistent with preferences. *Journal of Clinical Oncology*, 28(7), 1203–1208. <http://doi.org/10.1200/JCO.2009.25.4672>
- McCluskey, L., & Houseman, G. (2004). Medicare hospice referral criteria for patients with amyotrophic lateral sclerosis: a need for improvement. *Journal of Palliative Medicine*, 7(1), 47–53. doi:10.1089/109662104322737241
- Meier, D. E., Lim, B., & Carlson, M. D. (2010). Raising the standard: Palliative care in nursing homes. *Health Affairs*, 29(1), 136–140. doi:10.1377/hlthaff.2009.0912
- Morrison, R. S., Dietrich, J., Ladwig, S., Quill, T., Sacco, J., Tangeman, J., & Meier, D. E. (2011). Palliative care consultation teams cut hospital costs for medicaid beneficiaries. *Health Affairs*, 30(3), 454–463. <http://doi.org/10.1377/hlthaff.2010.0929>
- Morrison, R. S., Penrod, J. D., Cassel, J. B., Caust-Ellenbogen, M., Litke, A., Spragens, L., & Meier, D. E. (2008). Cost savings associated with US hospital palliative care consultation programs. *Archives of Internal Medicine*, 168(16), 1783–1790. <http://doi.org/10.1001/archinte.168.16.1783>

- National Hospice and Palliative Care Organization. (n.d.). NHPCO Facts and Figures. Retrieved from https://www.nhpc.org/sites/default/files/public/Statistics_Research/2016_Facts_Figures.pdf.
- Nipp, R. D., El-Jawahri, A., Fishbein, J. N., Eusebio, J., Stagl, J. M., Gallagher, E. R., ... Temel, J. S. (2016). The relationship between coping strategies, quality of life, and mood in patients with incurable cancer. *Cancer*, 122(13), 2110–2116. <http://doi.org/10.1002/cncr.30025>
- Nordgren, L., & Sorensen, S. (2003). Symptoms experienced in the last six months of life in patients with end-stage heart failure. *European Journal of Cardiovascular Nursing*, 2(3), 213–217. doi:10.1016/s1474-5151(03)00059-8
- Norton, S. A., Hogan, L. A., Holloway, R. G., Temkin-Greener, H., Buckley, M. J., & Quill, T. E. (2007). Proactive palliative care in the medical intensive care unit: Effects on length of stay for selected high-risk patients. *Critical Care Medicine*, 35(6), 1530–1535. <http://doi.org/10.1097/01.CCM.0000266533.06543.0C>
- Patterson, K. R., Croom, A. R., Teverovsky, E. G., & Arnold, R. (2014). Current state of psychiatric involvement on palliative care consult services: Results of a national survey. *Journal of Pain and Symptom Management*, 47(6), 1019–1027. <http://doi.org/10.1016/j.jpainsymman.2013.06.015>
- Potter, J., Hami, F., Bryan, T., & Quigley, C. (2003). Symptoms in 400 patients referred to palliative care services: Prevalence and patterns. *Palliative Medicine*, 17(4), 310–314. <http://doi.org/10.1191/0269216303pm760oa>
- Quill, T. E., & Abernethy, A. P. (2013). Generalist plus specialist palliative care — Creating a more sustainable model. *New England Journal of Medicine*, 368(13), 1173–1175. <http://doi.org/10.1056/NEJMp1215620>
- Rabow, M., Kvale, E., Barbour, L., Cassel, J. B., Cohen, S., Jackson, V., ... Weissman, D. (2013). Moving upstream: A review of the evidence of the impact of outpatient palliative care. *Journal of Palliative Medicine*, 16(12), 1540–1549. <http://doi.org/10.1089/jpm.2013.0153>
- Rabow, M. W., Dibble, S. L., Pantilat, S. Z., & McPhee, S. J. (2004). The comprehensive care team: A controlled trial of outpatient palliative medicine consultation. *Archives of Internal Medicine*, 164(1), 83–91. <http://doi.org/10.1001/archinte.164.1.83>
- Ripamonti, C. (1999). Management of dyspnea in advanced cancer patients. *Supportive Care in Cancer: Official Journal of the Multinational Association of Supportive Care in Cancer*, 7(4), 233–243. <http://doi.org/10.1007/s005209900037>
- Smith, T. J., Coyne, P., Cassel, B., Penberthy, L., Hager, M. A., & Hopson, A. (2003). A high-volume specialist palliative care unit and team may reduce in-hospital end-of-life care costs. *Journal of Palliative Medicine*, 6(5), 699–705. <http://doi.org/10.1089/109662103322515202>
- Solano, J. P., Gomes, B., & Higginson, I. J. (2006). A comparison of symptom prevalence in far advanced cancer, AIDS, heart disease, chronic obstructive pulmonary disease and renal disease. *Journal of Pain and Symptom Management*. <http://doi.org/10.1016/j.jpainsymman.2005.06.007>
- Spetz, J., Dudley, N., Trupin, L., Rogers, M., Meier, D. E., & Dumanovsky, T. (2016). Few hospital palliative care programs meet national staffing recommendations. *Health Affairs*, 35(9), 1690–1697. <http://doi.org/10.1377/hlthaff.2016.0113>
- Suzuki, M., Muro, S., Ando, Y., Omori, T., Shiota, T., Endo, K., ... Mishima, M. (2012). A randomized, placebo-controlled trial of acupuncture in patients with chronic obstructive pulmonary disease (COPD) the COPD-acupuncture trial (CAT). *Archives of Internal Medicine*, 172(11), 878–886. <http://doi.org/10.1001/archinternmed.2012.1233>
- Temel, J. S., Greer, J. A., & Muzikansky, A. (2010). Early palliative care for patients with metastatic non-small-cell lung cancer. *New England Journal Medicine*, 363, 733–742.
- Vainio, A., & Auvinen, A. (1996). Prevalence of symptoms among patients with advanced cancer: An international collaborative study. *Journal of Pain and Symptom Management*, 12(1), 3–10. [http://doi.org/10.1016/0885-3924\(96\)00042-5](http://doi.org/10.1016/0885-3924(96)00042-5)
- VITALtalk. (n.d.). Retrieved from vitaltalk.org.
- von Haehling, S., Morley, J. E., & Anker, S. D. (2010). An overview of sarcopenia: Facts and numbers on prevalence and clinical impact. *Journal of Cachexia, Sarcopenia and Muscle*, 1(2), 129–133. <http://doi.org/10.1007/s13539-010-0014-2>

- Wood, G. J., Shega, J. W., Lynch, B., & Von Roenn, J. H. (2007). Management of intractable nausea and vomiting in patients at the end of life: "I was feeling nauseous all of the time... nothing was working". *Journal of the American Medical Association*. <http://doi.org/10.1001/jama.298.10.1196>
- Wu, F. M., Newman, J. M., Lasher, A., & Brody, A. A. (2013). Effects of initiating palliative care consultation in the emergency department on inpatient length of stay. *Journal of Palliative Medicine*, 16(11), 1362–1367. <http://doi.org/10.1089/jpm.2012.0352>
- Yennurajalingam, S., Atkinson, B., Masterson, J., Hui, D., Urbauer, D., Tu, S.-M., & Bruera, E. (2012). The impact of an outpatient palliative care consultation on symptom burden in advanced prostate cancer patients. *Journal of Palliative Medicine*, 15(1), 20–24. <http://doi.org/10.1089/jpm.2011.0219>

Suggested Reading

- Back, A., Arnold, R., & Tulsky, J. (2009). *Mastering communication with seriously ill patients: Balancing honesty with empathy and hope*. New York: Cambridge University Press. doi:<http://doi.org/10.1017/CBO9780511576454>
- Gawande, A. (2014). *Being mortal: Medicine, and what matters in the end*. London: Profile Books.
- Qualls, S. H., & Kasl-Godley, J. E. (2010). *End-of-life issues, grief, and bereavement: What clinicians need to know*. Hoboken, NJ: John Wiley & Sons.

Patient Navigation/Community Health Workers

Kristen J. Wells and Janna R. Gordon

San Diego State University and SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA

Community health worker and patient navigation interventions are designed to assist individuals in obtaining healthcare services and in taking action to increase healthy behaviors and decrease unhealthy behaviors. Community health workers have a long history of assisting with the delivery of healthcare in numerous communities and settings globally. In contrast, patient navigation models were initially developed in the 1990s in the United States and Canada to promote patient-centered care for cancer.

There is evidence that community members have been providing basic health services to other members of the community in which they live for more than 60 years (Lehmann & Sanders, 2007). Internationally, community health workers are known by a wide range of titles, including health promoter, outreach educator, paramedical worker, *promotora*, village health worker, and community health representative (Lehmann & Sanders, 2007). A community health worker has been defined as a “frontline public health worker who is a trusted member of and/or has an unusually close understanding of the community served” (Bovbjerg, Eyster, Ormond, Anderson, & Richardson, 2013). Community health workers are trained to deliver a specific intervention, but have not received formal healthcare training as would be obtained as part of tertiary education. Thus, individuals who have studied psychology are eligible to be community health workers if they are trusted by and have a strong understanding of the community that they are assisting.

Unlike community health worker programs, patient navigation programs are a relatively recent development in healthcare. The first patient navigation program was created in response to findings from hearings in the United States that were described in the American Cancer Society’s *Report to the Nation: Cancer in the Poor*. This report indicated that individuals with limited resources face significant obstacles to cancer care, including sociocultural barriers (e.g., limited social support), logistical barriers (e.g., lack of transportation), and financial

barriers (e.g., inability to afford health insurance, Corliss, 1989). In response to this report, Dr. Harold P. Freeman created the first patient navigation program in Harlem, New York, which aimed to improve breast cancer outcomes. The most widely cited definition of patient navigation states that it is a barrier-focused intervention that (a) is provided to individual patients for a defined episode of healthcare, (b) has a definite endpoint when services provided are complete, (c) targets a defined set of health services that are required to complete an episode of healthcare, (d) focuses on the identification of individual patient-level barriers to accessing healthcare, and (e) aims to reduce delays in accessing the continuum of healthcare services (Wells et al., 2008).

There are various conceptualizations regarding the best models of patient navigation interventions (Wells & Nuhaily, 2018), with disagreement on what type of personnel should serve as patient navigators. In one patient navigation model, the most important qualification of a patient navigator is that the patient navigator be a “cultural broker and interpreter” who is from the community served by the patient navigation intervention (Braun et al., 2012). Similar to the definition of a community health worker, this model of patient navigation does not require a “clinical degree” to perform the patient navigation intervention, but emphasizes the patient navigator’s ability to work with both the intended audience and healthcare providers, and to serve as an advocate for patients. Thus, individuals with a wide range of educational backgrounds, including psychology, serve as patient navigators. A second model of patient navigation emphasizes the need for professional training of patient navigators, which is usually completed in the fields of nursing or social work. Finally, a model advocated by numerous individuals and agencies indicates that patient navigation should be provided by a multidisciplinary team of both lay and professional navigators (Freeman, 2012; Oncology Nursing Society, the Association of Oncology Social Work, and the National Association of Social Workers, 2010).

There has been substantial research evaluating the effectiveness of various types of community health worker and patient navigation interventions designed to improve health outcomes. Much of this research has been summarized in both narrative and systematic reviews, as described in detail below. This entry will focus on health outcomes for which there has been substantial research on the effectiveness of patient navigation and community health worker interventions.

Communicable Diseases

HIV/AIDS

There has been some research evaluating whether community health workers are effective in improving outcomes among people living with human immunodeficiency virus (HIV). One narrative synthesis examined the characteristics and impact of community health workers who provided services to people living with HIV in sub-Saharan Africa across 21 studies (Mwai et al., 2013). Community health workers delivered several types of services, such as providing education about HIV-related topics, providing counseling to encourage disclosure of HIV status, facilitating adherence to antiretroviral therapy (ART) via telephone reminders or home visits, and facilitating the retention of patients in HIV care. Although none of the studies evaluated the impact of community health worker interventions on HIV-related knowledge, there is some evidence that community health workers had a positive impact on HIV serostatus disclosure and on improved retention of people living with HIV in HIV-related healthcare.

Only one study included in this review examined the impact of community health workers on adherence, finding that patients in South Africa receiving adherence support were more likely to obtain ART medication refills compared with those without this support. Furthermore, two studies found that community health workers were linked with decreased mortality among people living with HIV in Malawi and South Africa.

Peer health navigation has also emerged as a strategy to enhance health outcomes among people living with HIV. A recent systematic review was conducted to evaluate the efficacy of interventions using HIV-positive peer health navigators to increase health services utilization and medication adherence among people living with HIV (Genberg et al., 2016). This review identified nine studies in the United States and sub-Saharan Africa that examined the use of peer navigation on retention in HIV care and adherence to ART used in the management of HIV. They found mixed results for the effect of peer health navigation on ART adherence; out of seven studies, two found increased adherence among those receiving a peer navigation intervention (1,200 pregnant women in South Africa and 20 nonadherent people in Missouri), and one study found a positive effect that was not maintained at follow-up. The other studies found no difference between peer navigation and control conditions immediately following the intervention. In addition, two studies examined the impact of peer navigation on retention in HIV care and found that peer navigation programs had a positive effect on retention among pregnant women in South Africa, as well as adults in Uganda. The authors postulated that peer navigation interventions serving people living with HIV may be most beneficial in resource-constrained settings with a high patient load and limited availability of healthcare professionals.

Tuberculosis

The World Health Organization (WHO) currently recommends the use of community health worker and community health volunteer (unpaid community health workers) interventions as part of a more comprehensive strategy to control tuberculosis, called directly observed treatment (DOT). DOT seeks to enhance medication adherence by providing direct supervision of patients while they take their daily dose of medication. Clinic-based DOT typically requires that individuals travel to a healthcare institution for each dose, where they are supervised by government health workers. In contrast, community-based DOT utilizes community health workers or community health volunteers that have been trained in tuberculosis prevention and care and directly monitor an individual's dose at locations in the community, such as at patients' homes.

Published systematic reviews have evaluated whether community health workers can improve outcomes related to tuberculosis (Karumbi & Garner, 2015; Lewin et al., 2010; Wright, Westerkamp, Korver, & Dobler, 2015). To date, these reviews have been inconclusive in their findings. A systematic review of four studies, which were mostly conducted in low-income communities, found that there was moderate evidence that community health workers improve cure rates among "new" and "retreated" tuberculosis patients (Lewin et al., 2010). Similarly, in a systematic review and meta-analysis, Wright et al. (2015) compared community-based DOT versus clinic-based DOT for tuberculosis treatment outcomes and treatment retention. They identified eight studies conducted in India, the Middle East, and Africa. Two of the eight studies found that treatment was more effective among those receiving community-based DOT compared with clinic DOT. In contrast, five studies found no difference between community-based DOT and clinic DOT. One study found that treatment outcomes did not differ between community-based DOT and clinic DOT for newly diagnosed, treatment-naïve

patients; however, community-based DOT was more effective for patients already on tuberculosis treatment when compared with clinic-based DOT. When the studies were pooled for meta-analysis, the treatment outcome for community-based DOT was significantly better than for the clinic DOT. Researchers have indicated that community-based DOT may be especially beneficial in contexts in which individuals may have difficulty getting to clinic each day (e.g., long distance to travel, transit is expensive, work responsibilities suffer). However, Wright et al. (2015) found no difference between community-based DOT and clinic DOT with regard to reducing the rate of patients becoming lost to follow-up across seven studies in which this was an outcome.

While Lewin et al. (2010) and Wright et al. (2015) found largely mixed to positive findings regarding the benefit of community health workers in enhancing tuberculosis treatment response, another recent systematic review (Karumbi & Garner, 2015) found limited evidence indicating the effectiveness of this intervention strategy. This study compared the efficacy of various forms of DOT (e.g., family observer, community health worker observer) to self-administered tuberculosis therapy and found no difference between DOT occurring in a healthcare facility and DOT supervised by a community health worker or family member at home (Karumbi & Garner, 2015). Furthermore, they found that DOT did not significantly enhance tuberculosis cure rates or treatment completion rates compared with self-administered tuberculosis therapy, in contrast to the WHO's guidelines for tuberculosis control.

Noncommunicable Diseases

Cancer

Community health worker interventions have been frequently applied to facilitate care related to early detection or prevention for breast, cervical, and colorectal cancer (Kim et al., 2016; Viswanathan et al., 2009; Wells et al., 2011). A 2016 systematic review of randomized controlled trials (Kim et al., 2016) evaluating community health worker interventions designed for vulnerable populations found that the community health worker model was most frequently applied to increase receipt of cervical and breast cancer screening. Several systematic reviews have noted that community health worker interventions are associated with increased rates of uptake of screening mammography (Kim et al., 2016; Viswanathan et al., 2009; Wells et al., 2011). In fact, a 2011 meta-analysis found a small but significant increase in receipt of screening mammography associated with receipt of a community health worker intervention, with a stronger effect noted in randomized trials of these interventions (Wells et al., 2011).

However, fewer high-quality studies have evaluated whether community health worker interventions can improve receipt of cervical cancer screening; thus, there are inconclusive results (Kim et al., 2016; Viswanathan et al., 2009). There have also been few studies included in systematic reviews evaluating whether community health worker interventions improve receipt of colorectal cancer screening. Those studies that have been included in reviews have measured a variety of outcomes (i.e., obtaining an endoscopy appointment, rate of fecal occult blood test) and have yielded mixed findings (Kim et al. 2016; Viswanathan et al., 2009).

Since patient navigation was developed to improve outcomes in cancer and reduce cancer disparities, various patient navigation intervention models have been evaluated to determine their efficacy in improving cancer care. There are multiple published narrative and systematic

reviews of patient navigation research in cancer (Baik, Gallo, & Wells, 2016; Freund et al., 2014; Krok-Schoen, Oliveri, & Paskett, 2016; Paskett, Harrop, & Wells, 2011; Robinson-White, Conroy, Slavish, & Rosenzweig, 2010; Wells et al., 2008). Most narrative reviews have found evidence of the efficacy of patient navigation in improving adherence to recommended screening for breast, cervical, colorectal, and prostate cancer (Krok-Schoen et al., 2016; Paskett et al., 2011; Wells et al., 2008). Similarly, most studies indicate that patient navigation increases adherence to recommended diagnostic care and improves timeliness of diagnostic care for patients who have experienced a breast, cervical, colorectal, or prostate cancer abnormality (Krok-Schoen et al., 2016; Wells et al., 2008). A meta-analysis of data collected from 10,521 patients enrolled in the multisite Patient Navigation Research Program indicated that patient navigation did not improve time to diagnostic resolution of a breast, cervical, colorectal, or prostate cancer abnormality between 0 and 90 days, when compared with usual care. However, there were significant differences between patient navigation and control patients 91 and 365 days following identification of a cancer abnormality, suggesting that patient navigation may prevent patients from being lost to follow-up (Freund et al., 2014).

Similar to the findings related to diagnostic resolution of a cancer-related abnormality, differences between patient navigation and comparison groups in initiation of cancer treatment among participants diagnosed with breast, cervical, colorectal, or prostate cancer in the Patient Navigation Research Program were noted between 91 and 365 days following diagnosis of cancer, with participants receiving patient navigation being more likely to initiate cancer treatment (Freund et al., 2014). On the other hand, narrative and systematic reviews of research evaluating patient navigation as an intervention to improve timely receipt of cancer treatment have yielded mixed results regarding its effectiveness (Baik et al., 2016; Paskett et al., 2011).

Cardiometabolic Diseases

Community health worker interventions have been utilized to promote the management and prevention of cardiometabolic diseases, primarily among underserved populations in the United States. Interventions include those designed to improve glycemic control among individuals living with diabetes, as well as those created to decrease cardiovascular disease risk factors or manage cardiovascular disease. A 2016 systematic review of community health worker interventions designed for vulnerable populations found that cardiometabolic disease risk reduction was the second most common application of community health worker interventions for vulnerable populations (Kim et al., 2016). Most (62%) studies targeting cardiometabolic disease risk found that community health worker interventions yielded a significant reduction in risk of these diseases.

Four of eight studies indicated that community health worker interventions were beneficial for blood pressure control compared with control groups, whereas two found no difference between community health worker and control groups, and two studies lacked statistical power. These findings are in contrast to those of a 2007 systematic review (Brownstein et al., 2007), which indicated that 9 of 10 studies found a positive impact of community health worker interventions on blood pressure.

Additionally, six of eight studies included in the 2016 systematic review (Kim et al., 2016) demonstrated that community health worker interventions were associated with improved glycemic control of diabetes, measured by fasting blood glucose or hemoglobin A1c, a measure of blood glucose over the past 6–8 weeks. This finding is consistent with a 2015

systematic review and meta-analysis (Palmas et al., 2015) specifically evaluating the impact of community health worker interventions on A1c levels among medically underserved patients with diabetes. This study found that community health worker interventions, especially those lasting at least one year, were successful in reducing A1c levels when compared with usual care. Furthermore, they noted that improvement in A1c due to community health worker interventions was greatest for those patients with the highest A1c at the outset. This suggests that individuals with the poorest glycemic control may benefit most from such an intervention.

Maternal and Child Health

In addition to focusing on specific health conditions, community health worker interventions have been implemented to improve health and access to healthcare at specific times in the lifespan, with particular emphasis on maternal and child health. There have been numerous systematic reviews that have summarized the research literature in this domain (Gilmore & McAuliffe, 2013; Lewin et al., 2010; Viswanathan et al., 2009). A narrative synthesis of community health worker interventions for maternal and child health in low- and middle-income countries (Gilmore & McAuliffe, 2013) found that these interventions have had a positive impact on malaria prevention practices, food safety practices that resulted in decreased childhood diarrhea, immunization rates, rates of exclusive breastfeeding, skin-to-skin care for prevention of hypothermia, and maternal mental health. Although Lewin et al. (2010) also found preliminary evidence suggesting that community health worker interventions may also reduce infant and childhood mortality, as well as childhood morbidity, they caution that these studies were of low quality. Thus, more research is needed to ascertain the impact of community health worker interventions on infant and childhood morbidity and mortality.

Breastfeeding

Lewin et al. (2010) provided a comprehensive evaluation of the efficacy of community health worker interventions on various outcomes related to breastfeeding, as reported in 18 studies that were primarily conducted in high-income countries. A meta-analysis of 12 studies indicated that community health worker interventions that promoted breastfeeding had a small but significant effect on initiation of breastfeeding and on any breastfeeding at 6 months postpartum. In addition, meta-analysis of ten studies examining engagement in breastfeeding among those receiving community health worker interventions compared with usual care found that community health worker interventions had a significant positive impact on exclusive breastfeeding by mothers up to 6 months postpartum (Lewin et al., 2010).

Pediatric Immunization

Systematic reviews of research evaluating the effectiveness of community health workers in improving pediatric immunization outcomes have determined that the studies are of mixed quality but suggest that community health worker interventions improve rates of being up to date on pediatric immunizations in global settings (Lewin et al., 2010; Viswanathan et al., 2009). In one review that included three studies, a good-quality trial found no difference between the community health worker and control groups, whereas two studies described as

“fair” or “poor” indicated that community health workers were associated with increased rates of being up to date on childhood immunizations and fewer children being late on immunizations (Viswanathan et al., 2009). Meta-analysis of the outcomes of four studies evaluating uptake of immunizations among children age 5 years or younger with low socioeconomic status living in high-income communities found that community health worker interventions increased the proportion of children who were up to date on their immunizations compared with usual care (Lewin et al., 2010). Similarly, a narrative synthesis of community health worker interventions for maternal and child health in low- and middle-income countries (Gilmore & McAuliffe, 2013) found that community health worker interventions increase immunization rates.

Summary

While community health worker approaches have been evaluated across global settings, populations, and health concerns, patient navigation research has been more limited to North American countries and conducted to improve outcomes related to cancer and HIV. The evidence for the effectiveness of community health worker and patient navigation interventions is mixed, possibly due to the limited number of studies that have been conducted for certain diseases and health outcomes. There is clear indication that both patient navigation and community health worker interventions improve retention in care for people living with HIV as well as receipt of screening mammograms. Patient navigation also improved adherence to cervical and colorectal cancer screening, although it is not clear whether community health worker interventions are effective in improving adherence to these recommended screening exams. Patient navigation also improves adherence to recommended diagnostic testing for breast, cervical, and colorectal cancer. Community health worker interventions are associated with reductions in cardiometabolic disease risks, including blood glucose and blood pressure, as well as increasing rates of initiation of breastfeeding and pediatric immunizations. However, it is not clear whether community health worker interventions improve tuberculosis health outcomes or whether patient navigation improves cancer treatment outcomes.

There are a number of avenues for future research regarding patient navigation and community health worker interventions. To date, the research examining community health worker and patient navigator interventions has mainly focused on certain diseases, like cancer or HIV, and certain periods of human development, like childhood. Even within the most frequently researched diseases, there are still numerous opportunities to study the effectiveness of these two intervention strategies for other outcomes. For example, most patient navigation studies have been conducted in the context of early detection of cancers for which there is a screening test, including breast, cervical, and colorectal cancer. Thus, there is still an opportunity to examine the effectiveness of these intervention strategies for a wide range of health outcomes, such as treatment initiation or completion among individuals diagnosed with cancer.

In addition, there are numerous opportunities for health psychologists, as well as individuals with general psychology training, to contribute to the development, implementation, and evaluation of community health worker and patient navigation interventions. Health psychologists and clinical health psychologists have an important role in developing community health worker and patient navigation interventions that are based on effective theories of behavior change. Furthermore, with their training in evaluation and psychometrics, psychologists are

frequently involved in efforts to evaluate these intervention strategies, both in the context of research and community and healthcare settings. Finally, individuals who have completed undergraduate or graduate training in psychology are well prepared to implement patient navigation and community health worker interventions.

In conclusion, the effectiveness of community health worker and patient navigation interventions has been very well studied for certain health outcomes, but there is very limited research for other health outcomes. Further research is needed to determine the effectiveness of these interventions for other disease outcomes and in other global contexts. Finally, there is a need to regulate, train, support, and credential community health workers and patient navigators who perform this work. There is a significant role for individuals with psychology training in the development, implementation, and evaluation of patient navigation and community health worker interventions.

See Also

Patient satisfaction, Adherence, Screening behavior, Health behavior interventions, Community interventions, Access to healthcare, Health disparities.

Author Biographies

Kristen J. Wells, PhD, MPH, is associate professor in the Department of Psychology at San Diego State University and a participating member of the University of California, San Diego Moores Cancer Center, in San Diego, California. Dr. Wells' research emphasizes the use of community-engaged approaches in the development and evaluation of cancer communication and cancer care coordination interventions. She specializes in the development and evaluation of patient navigation programs focused on cancer outcomes.

Janna R. Gordon, MS, is a doctoral student at the San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology. Ms. Gordon's research has examined cancer across the care continuum, including cancer communication, prevention, detection, and survivorship. She is particularly interested in investigating psychosocial aspects of cancer screening and survivorship among people living with HIV/AIDS (PLWH) and sexual minority individuals.

References

- Baik, S. H., Gallo, L. C., & Wells, K. J. (2016). Patient navigation in breast cancer treatment and survivorship: A systematic review. *Journal of Clinical Oncology*. doi:10.1200/jco.2016.67.5454
- Bovbjerg, R. R., Eyster, L., Ormond, B. A., Anderson, T., & Richardson, E. 2013. *The evolution, expansion, and effectiveness of community health workers*. Retrieved from <http://www.urban.org/sites/default/files/alfresco/publication-pdfs/413072-The-Evolution-Expansion-and-Effectiveness-of-Community-Health-Workers.PDF>.
- Braun, K. L., Kagawa-Singer, M., Holden, A. E., Burhansstipanov, L., Tran, J. H., Seals, B. F., ... Ramirez, A. G. (2012). Cancer patient navigator tasks across the cancer care continuum. *Journal of Health Care for the Poor and Underserved*, 23(1), 398–413. doi:10.1353/hpu.2012.0029
- Brownstein, J. N., Chowdhury, F. M., Norris, S. L., Horsley, T., Jack, L., Jr., Zhang, X., & Satterfield, D. (2007). Effectiveness of community health workers in the care of people with hypertension. *American Journal of Preventive Medicine*, 32(5), 435–447. doi:10.1016/j.amepre.2007.01.011

- Corliss, J. (1989). ACS reports on cancer and the poor. *Journal of the National Cancer Institute*, 81(16), 1206–1207. doi:10.1093/jnci/81.16.1206
- Freeman, H. P. (2012). The origin, evolution, and principles of patient navigation. *Cancer Epidemiology, Biomarkers & Prevention*, 21(10), 1614–1617. doi:10.1158/1055-9965.epi-12-0982
- Freund, K. M., Battaglia, T. A., Calhoun, E., Darnell, J. S., Dudley, D. J., Fiscella, K., ... Paskett, E. D. (2014). Impact of patient navigation on timely cancer care: The Patient Navigation Research Program. *Journal of the National Cancer Institute*, 106(6), dju115. doi:10.1093/jnci/dju115
- Genberg, B. L., Shangani, S., Sabatino, K., Rachlis, B., Wachira, J., Braitstein, P., & Operario, D. (2016). Improving engagement in the HIV care cascade: A systematic review of interventions involving people living with HIV/AIDS as peers. *AIDS and Behavior*. doi:10.1007/s10461-016-1307-z
- Gilmore, B., & McAuliffe, E. (2013). Effectiveness of community health workers delivering preventive interventions for maternal and child health in low- and middle-income countries: A systematic review. *BMC Public Health*, 13, 847. doi:10.1186/1471-2458-13-847
- Karumbi, J., & Garner, P. (2015). Directly observed therapy for treating tuberculosis. *The Cochrane Database of Systematic Reviews*, (5), CD003343. doi:10.1002/14651858.CD003343.pub4
- Kim, K., Choi, J. S., Choi, E., Nieman, C. L., Joo, J. H., Lin, F. R., ... Han, H. R. (2016). Effects of community-based health worker interventions to improve chronic disease management and care among vulnerable populations: A systematic review. *American Journal of Public Health*, 106(4), 671. doi:10.2105/AJPH.2015.302987a
- Krok-Schoen, J. L., Oliveri, J. M., & Paskett, E. D. (2016). Cancer care delivery and women's health: The role of patient navigation. *Frontiers in Oncology*, 6, 2. doi:10.3389/fonc.2016.00002
- Lehmann, U., & Sanders, D. D. (2007). *Community health workers: What do we know about them? The state of the evidence on programmes, activities, costs and impact on health outcomes of using community health workers*. University of the Western Cape. Retrieved from http://www.who.int/hrh/documents/community_health_workers.pdf.
- Lewin, S., Munabi-Babigumira, S., Glenton, C., Daniels, K., Bosch-Capblanch, X., van Wyk, B. E., ... Scheel, I. B. (2010). Lay health workers in primary and community health care for maternal and child health and the management of infectious diseases. *The Cochrane Database of Systematic Reviews*, (3), CD004015. doi:10.1002/14651858.CD004015.pub3
- Mwai, G. W., Mburu, G., Torpey, K., Frost, P., Ford, N., & Seeley, J. (2013). Role and outcomes of community health workers in HIV care in sub-Saharan Africa: A systematic review. *Journal of the International AIDS Society*, 16, 18586. doi:10.7448/ias.16.1.18586
- Oncology Nursing Society, the Association of Oncology Social Work, and the National Association of Social Workers. (2010). Oncology Nursing Society, the Association of Oncology Social Work, and the National Association of Social Workers joint position on the role of oncology nursing and oncology social work in patient navigation. *Oncology Nursing Forum*, 37(3), 251–252.
- Palmas, W., March, D., Darakjy, S., Findley, S. E., Teresi, J., Carrasquillo, O., & Luchsinger, J. A. (2015). Community health worker interventions to improve glycemic control in people with diabetes: A systematic review and meta-analysis. *Journal of General Internal Medicine*, 30(7), 1004–1012. doi:10.1007/s11606-015-3247-0
- Paskett, E. D., Harrop, J. P., & Wells, K. J. (2011). Patient navigation: An update on the state of the science. *CA: A Cancer Journal for Clinicians*, 61(4), 237–249. doi:10.3322/caac.20111
- Robinson-White, S., Conroy, B., Slavish, K. H., & Rosenzweig, M. (2010). Patient navigation in breast cancer: A systematic review. *Cancer Nursing*, 33(2), 127–140. doi:10.1097/NCC.0b013e3181c40401
- Viswanathan, M., Kraschnewski, J., Nishikawa, B., Morgan, L. C., Thieda, P., Honeycutt, A., ..., Jonas, D. (2009). Outcomes of community health worker interventions. *Evidence Report/Technology Assessment* (181):1–144, A1–2, B1–14, passim.
- Wells, K. J., Battaglia, T. A., Dudley, D. J., Garcia, R., Greene, A., Calhoun, E., ... Raich, P. C. (2008). Patient navigation: State of the art or is it science? *Cancer*, 113(8), 1999–2010. doi:10.1002/cncr.23815
- Wells, K. J., Luque, J. S., Miladinovic, B., Vargas, N., Asvat, Y., Roetzheim, R. G., & Kumar, A. (2011). Do community health worker interventions improve rates of screening mammography in the

- United States? A systematic review. *Cancer Epidemiology, Biomarkers & Prevention*, 20(8), 1580–1598. doi:10.1158/1055-9965.epi-11-0276
- Wells, K. J., & Nuhaily, S. (2018). Models of patient navigation. In E. A. Calhoun & A. Esparza (Eds.), *Patient Navigation: Overcoming Barriers to Care*. New York, NY: Springer.
- Wright, C. M., Westerkamp, L., Korver, S., & Dobler, C. C. (2015). Community-based directly observed therapy (DOT) versus clinic DOT for tuberculosis: A systematic review and meta-analysis of comparative effectiveness. *BMC Infectious Diseases*, 15, 210. doi:10.1186/s12879-015-0945-5

Suggested Reading

- Freeman, H. P., Muth, B. J., & Kerner, J. F. (1995). Expanding access to cancer screening and clinical follow-up among the medically underserved. *Cancer Practice*, 3(1), 19–30.

The Patient Protection and Affordable Care Act and Health Psychology

Ronald H. Rozensky

*Department of Clinical and Health Psychology, College of Public Health and Health Professions,
University of Florida, Gainesville, FL, USA*

“Health care costs affect the economy, the federal budget, and virtually every American family’s financial wellbeing. Health insurance enables children to excel at school, adults to work more productively, and Americans of all ages to live longer, healthier lives” (Obama, 2016, p. E2). Ten presidents of the United States, from both political parties—Misters Theodore Roosevelt, Franklin Roosevelt, Harry Truman, Dwight Eisenhower, John Kennedy, Lyndon Johnson, Richard Nixon, Jimmy Carter, Bill Clinton, and Barack Obama—all have advocated for some type of a national healthcare system or changes to the existing system (Jacobs & Skocpol, 2016). There have been various successful modifications to the healthcare system over the years including Medicare, Medicaid, healthcare insurance for children, medication coverage for seniors, and insurance coverage for the uninsured. Each of these changes occurred in the midst of some level of partisan political struggle including philosophical differences about the government’s role in the lives of citizens, in human services, and in healthcare specifically. Debates included which citizens should be covered for health and human services, what concrete services should be provided, which healthcare providers should be reimbursed, and what entity (the federal government, state governments, or private insurance companies) should manage payment for and deliver services. For each change in the US healthcare system, whether part of one of the larger national systemic changes or more local modifications of healthcare rules and coverage, health service psychology in general, and the specialty of clinical health psychology in particular, always has been involved in advocating for its patients, for funding of its science, for inclusion of its services in the evolving care system, and for working to make certain its own providers serve as autonomous professionals on the healthcare team.

This chapter will discuss the recent history that has brought about healthcare reform via the passage and implementation of the *Patient Protection and Affordable Care Act* (ACA; Public

Law No: 111-148, March 23, 2010). The chapter then will emphasize both the details of the *ACA* and ongoing healthcare reform efforts and how those, and will, impact the education, training, science, and practice of health psychology. It is the aim of the chapter to use that history to help prepare the reader for ongoing, and upcoming, changes to the healthcare system. For a broader view of US healthcare policy, health reform, and the *ACA*, readers are encouraged to consult Jacobs and Skocpol (2016). For a look at the evolution of the *ACA*, Gruber (2011), one of the consultants who helped formulate the *Act*, offers details of the policy and concepts adopted within the law. In an editorial in the *Journal of the American Medical Association*, President Obama (2016), author of the *ACA*, provides the reader a review of the impetus for the law and an assessment of progress since the inception of the *Act*. A series of articles focused on various recognized specialties in health service psychology and areas of psychological practice will be discussed in this chapter along with recommendations for interprofessional education, training, and practice. These can be reviewed in depth as background readings (institutional practice—Rozensky, 2011; academic health centers—Rozensky, 2012b; independent practice—Rozensky, 2013a; education and training in professional psychology—Rozensky, 2012c, 2013b, 2014a; primary care—Rozensky, 2014b; family psychology—Rozensky, Celano, & Kaslow, 2013; pediatric psychology—Rozensky & Janicke, 2012; clinical child and adolescent psychology—Janicke, Fritz, & Rozensky, 2015.)

The Patient Protection and Affordable Care Act

As part of the recent history of healthcare reforms in the United States, Berwick (2009) acknowledged the importance of the use of interprofessional, team-based healthcare. He detailed, for both healthcare providers and the public, that this type of a healthcare system can coordinate primary care providers and healthcare specialists both within episodes of care and for services occurring across episodes of care. Berwick described how this team-based approach was key to the actual quality of ongoing healthcare and is the basis for the redesign of a successful, integrated healthcare system. These comments were built upon Berwick, Nolan, and Whittington's (2008) introduction to what has been called the "triple aim" of healthcare reform; the triple aim is often seen as the impetus for the wave of healthcare reforms that lead to the passage of the *ACA* in 2010. Berwick et al. highlighted the importance of three goals—(a) decreasing the number of uninsured Americans, (b) improving the quality and *efficiency* of healthcare, and (c) reducing the overall costs of healthcare.

The actual implementation of the *ACA* was surrounded by a frenzy of political, legal, and technological issues. Along with ongoing political challenges to the *Act* in the US Congress, the Supreme Court of the United States (SCOTUS) responded to the legal storms by ruling that the *ACA* was constitutional (National Federation of Independent Business v. Sebelius, 2012) clearing the way for its implementation. SCOTUS then ruled that governmental subsidies for insurance also were constitutional (King v. Burwell, 2015). This allowed *ACA*'s ongoing growth. Even in the face of a technically challenged "roll out" (Mortiere, 2013, November 15), components of the *ACA* went into effect (Gruber, 2011; McDonough & Adashi, 2014). Initially more than the predicted seven million initial subscribers signed up by the end of the first enrollment period (April 2014; Goldstein & Eilperin, 2014, April 2). Some 12.7 million citizens were enrolled by February 2016 ([https://www.cms.gov/Newsroom/Media Release Database/Fact-sheets/2016-Fact-sheets-items/2016-02-04.html](https://www.cms.gov/Newsroom/Media%20Release%20Database/Fact-sheets/2016-Fact-sheets-items/2016-02-04.html)), and the number of uninsured individuals in the United States declined from 49 million in 2010 to 29 million by 2015 (Obama, 2016).

Oberland (2017) described how, during the Spring and Summer of 2017, Republican members of the US Congress, who controlled both the House and Senate, with a Republican president in the White House, mounted a “crusade to dismantle Obamacare [that], at least for now, collapsed” (p. 1). During that brief time period, the House passed the *American Health Care Act* by a margin of four votes in May and followed by the failure of the *Better Care Reconciliation Act* in August. Oberland described how the Congressional Budget Office projected that these two bills would have substantially increased the number of uninsured and would have provide less affordable and less comprehensive care than the *ACA* provisions already in place. “Still, Obamacare’s fortunes remain uncertain. Senate Republicans fell only one vote short of passing legislation, and with pressure on the GOP to fulfill its promise to overturn Obamacare, the party could try to revive repeal” (Oberland, 2017, p. 1).

It is clear that “whatever else might happen politically overtime regarding the *ACA*, changes in healthcare are to be anticipated and must be responded to ...” by all healthcare disciplines (Rozensky, 2014b, p. 1) given that the *ACA* is the law, it is operational, and millions have sought and will seek healthcare services through it. The various components of the *ACA* will shape healthcare delivery into the future. This then will have direct implications for the education and training within each healthcare discipline and how members of that discipline will practice, including clinical health psychology.

Components of the ACA and Their Implications for Health Psychology

Rozensky (2012a) presented a series of societal, scientific, and healthcare-related trends that set the stage for the *ACA* or developed in anticipation of, or in response to, healthcare reform. Each of these trends has a direct impact on clinical health psychology.

Health in the APA Bylaws

The trend most germane to this discussion is the ongoing changes in healthcare delivery and the healthcare system. Although many psychologists had been carrying out research and direct clinical services in medical hospitals and clinics for many years, it was not until 2001 that the American Psychological Association (APA) reaffirmed its commitment to psychology as a healthcare profession by adding “health” to its bylaws (Rozensky, Johnson, Goodheart, & Hammond, 2004). This was in direct response to ongoing healthcare reform, reaction to changes in the healthcare landscape, and in anticipation of additional changes to come.

Demographic Shifts

Bodenheimer, Chen, and Bennett (2009) pointed out that 133 million Americans had at least one chronic medical condition and this was expected to increase to 157 million by 2020. Sixty-three million citizens had multiple chronic illnesses, and Bodenheimer et al. (2009) predicted that number would rise to 81 million by 2020. They noted that while aging accounted for some of this increase, behaviorally related risk factors such as obesity and tobacco use were responsible for adding to this increase. Leading to the current epoch of healthcare reform, Bodenheimer et al. raised concerns about increased healthcare costs related to multiple chronic healthcare problems and wondered if “robust public health measures” (p. 66) could flatten the cost curve by preventing many behavioral health risk factors.

The population is aging. The US Census Bureau states that “between 2010 and 2050, the United States is projected to experience rapid growth in its older population” (Vincent & Velkoff, 2010, p. 1) with a doubling of the number of those over the age of 65 from 40.2 million in 2010 to 88.5 million by 2050. Further, “an increase in the proportion of the older population that is Hispanic and an increase in the proportion that is a race other than White” (p. 8) are projected. Consistent with the 2010 census (US Census Bureau, 2011), half of the growth in the US population between 2000 and 2010 was due to an increase in the Hispanic population that grew 43% to 50.5 million in 2010. Thirteen percent of the population was African American, and 5% was Asian, an increase of 43% during that same time period. Ortman and Guarneri (2009) state that the “racial and ethnic diversity of the US population is shown to increase” well into the future with the percentage of White-only population decreasing given overall population growth rate and international migration. Plaut (2010) described this changing picture of the US population on healthcare disparities and access to care.

Recognizing the impact of diversity trends (age, chronicity of illness, ethnic and racial changes), clinical health psychologists, educators, scientists, students, and practitioners are presented with the opportunity to build on psychology’s strong commitment to multiculturalism as a core competency (e.g., Rogers, 2009). Assuring this as a focus in the new healthcare system, including the appropriate education and training of our students in preparation for these societal changes, is a key responsibility of all psychologists.

Health Promotion and Disease Prevention

In the section of the *ACA*, Prevention of Chronic Disease and Improving Public Health, the *Act* focuses on issues of public health, health promotion, and disease prevention. Here the law discusses the proposal of evidence-based approaches to health promotion and a national disease prevention model for the public health. Such an emphasis on efficacy, effectiveness, and community-based research and prevention is based on psychology’s research and service traditions and reflects what Raczyński and Leviton (2004) noted, “... the behavioral focus of secondary and tertiary prevention often is not significantly influenced by the nature of the risk factor of disease but is rather based on behavioral objective—for example, losing weight, quitting smoking, and so forth” (p. 3). Further, Schneiderman and Speers (2001) describe the role of psychologists and social scientists across health promotion and disease prevention for both individuals and population health.

Given the importance of the behavioral issues in the *ACA*, the next generation of health psychologists must be prepared to engage in population-based approaches to the scientific study and treatment of the human condition. As our healthcare system evolves, use of psychology’s data from the bedside and consulting room to the community and population (Rozensky, 2008) will position psychology to use its critical thinking and research skills to bring important changes to the delivery system. Both the *ACA*’s current focus on population health and the US Government’s Healthy People 2010 and 2020 (ODPHP, 2020), which has long had a focus on the importance of health behaviors in health promotion, highlight psychology’s leading role in understanding health behaviors and will assure clinical health psychology’s relevancy on the healthcare team.

Technology

The American Reinvestment and Recovery Act infused over one billion dollars into the US economy to stimulate the “meaningful use” of electronic health records (Shachak & Jadad,

2010, p. 452). This reflects the ongoing growth of the digital age and, as Shachak and Jadad suggest, its impact on cost savings with the adoption of an integrated, electronic healthcare record-keeping system, at the local and national level. They further note that this technology will contribute to health promotion and quality care while enhancing patient-centered care. They warn, however, that “excessive preoccupation with privacy may interfere with quality service” (p. 453) that can be enhanced with efficient electronic information sharing. Sittig and Classen (2010) recommend systematic monitoring to assure that adoption of electronic record systems is done securely. Richards (2009) discusses a balance between professional psychology’s focus on its ethical responsibilities for maintaining patient confidentiality, the requirements of the Health Insurance Portability and Accountability Act regarding the sharing of patients’ personal health information (HIPAA, 1996), and the potential complications for psychologists working within an integrated, interprofessional healthcare work environment with medically ill patients versus those patients seeking only psychological care.

Health psychologists embarking on the use of electronic record keep and use of enhanced technologies in healthcare should look at Richards’ suggestions regarding openly addressing information sharing especially regarding electronic healthcare records, when discussing with patients the limits of confidentiality. As the ACA calls for enhanced interprofessional practice, health psychology should provide guidance to itself, per Shackak and Jadad’s concerns about not limiting information exchange, so that psychological services can be truly integrated into the expanding interprofessional healthcare milieu.

Federal Responses to the Healthcare System

Wilson, Rozensky, and Weiss (2010) describe the history and development of federal policy recommendations supporting “the integration of interprofessional education (IPE) into health professions education as a means of assuring a more collaborative healthcare workforce” (p. 210) including the role of the Department of Health and Human Services’ (DHHS) Advisory Committee on Interdisciplinary Community-Based Linkages (ACICBL). The ACICBL (2011), in preparation for the then upcoming writing of the Affordable Care Act, focused its 2010 annual report to the secretary of DHHS and Congress on the importance of the education and training of the entire interprofessional healthcare workforce to adequately address health behavior change in order to enhance the quality and cost effectiveness of the US healthcare system. This document not only helped reinforce the importance of interprofessional education and team-based practice but also highlighted behavioral health as a key component to integrated healthcare. Clearly assessment and treatment of behavioral health issues is a key competency of clinical health psychologists. This trend toward interprofessional education and care provides a welcomed challenge to health psychology educators and practitioners.

Interprofessional Team-Based Care

Possibly the most far reaching functional trend in healthcare is the ACA’s focus on interprofessionalism. The ACA directly recognizes the importance and impact of interprofessional education, training, and care on quality healthcare and cost effectiveness (cf., Section 3502, *Establishing Community Health Teams to Support the Patient-Centered Medical Home*). The ACA makes funding available to establish community-based, interprofessional (primary) healthcare teams that may include behavioral and mental health providers (including psychologists). Also detailed in the Act are interprofessional, integrated disease prevention and health promotion services (including funding for clinical teaching settings

such as academic health science centers and community-based teaching health centers) and interprofessional models of healthcare that can include integration of physical and behavioral/mental health services).

The IOM (2013) notes that interprofessional education (IPE) is not “smudging together the professions and saying they are all the same” but is “about learning together to ultimately improve health and the safety of patients” (p. 2–1). Wilson et al. (2010) noted federal support of interprofessional education, while Frenk et al. (2010) opined about this approach to healthcare being “an enlightened new professionalism that can lead to better services and consequent improvements in the health of patients and populations” (p. 1954). Interprofessionalism is a developmental step for all healthcare disciplines that moves beyond disjointed practices and is defined as “the development of a cohesive practice *between* professionals from different disciplines” (D’Amout & Oandasan, 2005, p. 9). Scheutz, Mann, and Evertt (2010) described the importance of shared competencies developed during professional education and training that are then utilized in actual practice in integrated, team-based healthcare systems (Interprofessional Education Collaborative [IEC], 2011) whether in a primary or tertiary care setting. The success of this team-based focus is based on competencies shared across disciplines and includes values and ethics, clarification of independent and shared roles and responsibilities for collaborative practice, interprofessional communication skills, and competencies that enhance team work and team-based care.

Belar (2016) detailed interprofessional education in health service psychology and provides brief descriptions of psychology education and training programs with an interprofessional focus. That chapter details the categories of interprofessional behaviors elucidated by the Interprofessional Professionalism Collaborative (2014) and that can be measured to review successful behavior in that practice environment—communications, respect, altruism and caring, ethics, excellence, and accountability. The curriculum for the education and training of health service psychologists should include development of competencies for true interprofessional healthcare (IEC, 2011; Interprofessional Education Collaborative Expert Panel, 2011) for traditional mental or behavioral health services and in providing psychological assessment and treatment of patients with medical diagnoses. Contemporary curricula should include a focus on interprofessional communication skills (including developing skills and comfort with the use of electronic health records) and verbal and electronic sharing of clinical information about patients within teams. The patient-centered healthcare system promulgated by the ACA also includes skills needed to routinely explain to patients the importance of sharing sensitive mental health and psychologically focused information with whole healthcare team to enhance continuity and thus quality care (Shachak & Jadad, 2010).

Competency-Based Education and Quality Care

A general trend in health service psychology has been the competency movement and the expectation that a team of verifiably competent providers enhances quality care. Cubic, Mance, Turgesen, and Lamanna (2012), for example, have opined that “interprofessional educational experiences between psychology and other healthcare providers are vital to insure professionals value one another’s disciplines in healthcare reform endeavors, most notably the patient-centered initiatives” (p. 84) including quality improvement efforts. These authors provide descriptions of training opportunities across the graduate and postgraduate education spectrum that includes psychologists and other healthcare professionals learning and working together.

Individual competencies in primary care psychology offered by The Interorganizational Work Group on Competencies for Primary Care Psychology Practice (IWG, 2013) highlight

a set of six broad-based competency domains for primary care psychologists: science, systems, professionalism, relationships, application, and education. France et al. (2008) offer a core competency-based model for clinical health psychology that describes both foundational and functional competencies across all venues of care. More recently, the Health Service Psychology Education Collaborative (2013) published *A Blueprint for Health Service Psychology Education and Training* containing several recommendations that include a need for “clearly articulated and understood” (p. 1) competencies for provision of health services, minimal qualifications for those entering doctoral education in health service psychology, and integration of science and practice to successfully implement evidence-based treatment, including the scientific mindedness required to create new knowledge and not simply “consume research findings,” and the adoption of “a national standard on accreditation” (p. 1). Each of these sets of competencies responds to trends in healthcare education and practice and provides guidance for curriculum development and goals for each student and practitioner as they learn and work within the changing interprofessional healthcare environment.

Structural Changes in Healthcare

HealthCare Home and Accountable Care Organizations. As the healthcare system changes to meet the challenges of the ACA, one trend is the structural reorganization of local healthcare delivery systems and the financial arrangements that support them. The Act is focused on affordable care, but it truly focuses on accountability to assure affordability. It describes the advent of structural changes to the system with accountable care organizations (ACOs) and patient-centered healthcare (medical) homes (PCMH); both built on the foundation of inter-professionalism. Rittenhouse, Shortell, and Fisher (2009) describe ACO aligning financial incentives and accountability across the care continuum, while the PCMH emphasizes strong primary care services as the key to delivery system reforms. For these authors, successful ACOs require a strong primary care base, while the successful PCMH must have a comprehensive delivery system beyond its primary care core. Fisher, Staiger, Bynum, and Gottlieb (2007) recommend that ACOs utilize an extended hospital medical staff model, in concert with hospitals themselves as the hub of the wheel, to assure continuity of care designed to accomplish the mandates of the ACO concept—including performance measures that hold the healthcare professionals (professional staff) and hospitals accountable for quality and cost-effective care. Hospital-based ACOs, most likely, will require enhanced accountability including explicit credentialing of providers. Rozensky (2011, 2012b) noted that credentialing will require graduation from accredited education programs and an increasing expectation of specialty board certification—routine expectations of hospital-based healthcare providers. These system-based expectations should stimulate clinical health psychology to review its national training models and local programs looking at importance of both the general practice and specialized practice. The PCMH is seen as a major improvement to primary care delivery given its focus on access, coordination, and comprehensive/integrated care, and the sustained (long term) personal relationship between patient and provider(s) with patients actively engaged in a healthcare partnership (Nutting et al., 2011). The Carter Center (2011) provided recommendations regarding the education and training of the healthcare workforce to ensure the successful future of primary care. These recommendations include encouraging all health profession education programs to (a) include education about the demographic, socioeconomic, financial, quality, political, and cultural issues affecting healthcare services; (b) educate students about development of high-functioning teams in primary care; and (c) educate providers about the incidence and prevalence of behavioral conditions in primary

care settings. The Carter Center also recommends that the healthcare system in general (d) redirects resources to establish appropriate training settings and curricula to prepare the primary care and interprofessional workforce and (e) the development of a research agenda to inform changes to curricula, methods, and processes of healthcare education including measurement of outcomes.

Specialization

Given the enhanced focus on accountability described above, individual health service psychologists have been encouraged that they seek credentialing at the highest level of independent practice in any healthcare system or organization (Rozensky, 2012b; Rozensky & Kaslow, 2012). Clinical health psychology is one of the specialties recognized by the APA (<http://www.apa.org/ed/graduate/specialize/recognized.aspx>). Individual clinical health psychologists are encouraged to seek board certification (www.abpp.org) in order to have same credential that physician colleagues are expected to obtain to practice in an organization and, as Kaslow, Patterson, and Gottlieb (2011) has noted, consumers expect their healthcare providers to be board certified as a marker of quality care.

Financial Accountability

Clearly a law with affordable care in its title reflects the trend of controlling costs as described by Berwick et al. (2008). When psychological services are studied, healthcare cost savings should be a key research variable in any, and all, applied treatment (efficacy or effectiveness studies) or translational services research (Rozensky, 2011). Translational studies that compare the relative costs for outcomes of various treatment programs can help policymakers determine where to get the “biggest bang for the buck” (e.g., Blount et al., 2007). As such, graduate programs in clinical health psychology, students, and faculty are encouraged to build cost-related variables in their research programs. Curriculum should include both program evaluation competencies as well as measurement of cost offset and cost efficiency skills (Rozensky, 2011). This allows clinical health psychologists to offer a value added skill to the healthcare team based on psychology’s robust history as a measurement and research-based discipline.

Clinical Health Psychology, 2020, and Beyond

Each step in the history of clinical health psychology has been motivated by the spirit of that time—spirit based on politics within and external to the profession, scientific developments in psychology and medicine, technological evolution, vicissitudes in contemporary healthcare and healthcare financing, and changes in educational philosophy. The future of clinical health psychology will be based on contemporary *zeitgeist* composed of those same motivating forces and trends today and tomorrow. The successful health psychologist, whether student, early career, or seasoned veteran, can only contribute to the growth of the field by being aware of these changes and plan for them accordingly. Attention, then, should be paid to Berwick (2016) who described three eras in the evolution of healthcare. Era 1, with roots back to Hippocrates, sees healthcare as a noble profession, self-regulating, and beneficent. Scientific data, however, found “enormous variations in practice” (p. E1) that then lead to Era 2 with its ongoing focus on measurement, scrutiny, and accountability leading to incentives and marketing of healthcare. He argues that healthcare providers must move to Era 3 that rejects

the protectionism of Era 1 and the reductionism of Era 2. Berwick offers nine changes to the future of healthcare that recast measurement, incentives, and a revenue-based strategy to one of quality that listens to the voice of the people served, builds on “improvement science” (p. E2) and transparency, and rejects greed in a spirit of civility. He urges all individual and professional organizations to take up this challenge.

Contemporary clinical health psychology exists within the broader context of healthcare reform as detailed in the *Patient Protection and Affordable Care Act* and its ongoing implementation and the evolution of health systems as presented by Berwick (2016). The *ACA* recognizes the importance of evidence-based healthcare, quality care including credentialing and specialization, team-based, and interprofessionalism in both education and practice (Rozensky, 2011, 2013a, b). And clearly, education and training in all branches of health service psychology, including clinical health psychology, must address the issues highlighted above both in its curricula, practical training, and evolving scope of practice (Rozensky, 2014a).

Maintaining Autonomy in an Interprofessional World. Rozensky (2004) has argued that lessons from health service psychology’s successful history demand that organized psychology maintains its administrative autonomy at the local level so that psychologists always will be in a position to protect their own resources, carry out their own credentialing, and assure noninterference in their academic freedom and scope of practice by another discipline. This is particularly important should such turf battles reignite in times of limited healthcare financing and resources. Such times that might be at hand as the demands of healthcare reform move forward. These lessons of autonomy should be passed down as part of the formal education of the next generation of professional psychologists who will be responsible for psychology’s future. Finding the balance of professional autonomy and self-governance within a healthcare milieu demanding increased interprofessional cooperation can be a challenge but necessary for the survival of health psychology as a recognized, independent profession.

The Psychology Workforce

In order to understand where clinical health psychologists will be employed in the future, a clear national, workforce analysis philosophy must be operationalized, and data collected and studied in order to inform educators and practitioners alike as to societal need and employment opportunities (Rozensky, Grus, Belar, Nelson, & Kohut, 2007). Questions like how many psychologists will be needed in primary care, in cardiac care, in cardiac psychology, in psycho-oncology, etc. will help the field have a picture of the future and provide direction to students and early career psychologists planning their education and practice focus. Workforce data will guide faculty in building their curricula and shape advocacy for research funding and educational and training dollars.

Many scholars have described the history and successful evolution of the specialty of clinical health psychology and highlighted the various individuals and organizations that have helped define the education, training, science, and practice of clinical health psychologists (Postupak & Rozensky, 2016). This history reflects a robust science whose application is key to helping those who wish to learn to prevent disease, improve their health, or seek to manage their illness or their reactions to that illness. As Postupak and Rozensky said, clinical health psychologists work to improve the human condition, and each scientist and clinician recognizes, in their day-to-day efforts, what Shakespeare said some 400 years ago, “What wound does not heal but by degree?”

For any health psychologist to succeed in the future, they must be competent in understanding their work in the context of the scientific and political environments that define practice and

funding of healthcare. The successful health psychologist must apply that knowledge to education, science, and practice in an ever unfolding interprofessional healthcare context to ensure affordable and quality care and build the best future we can for the profession of psychology.

Politics is the gizzard of society, full of grit and gravel, and the two political parties are its opposite halves – sometimes split into quarters – which grind on each other. Not only individuals but states have thus a confirmed dyspepsia.

—Henry David Thoreau

Author Biography

Ronald H. Rozensky, PhD, ABPP—Dr. Rozensky is professor emeritus at the University of Florida where he was chair of the Department of Clinical and Health Psychology and associate dean for International Programs. He is founding editor of *The Journal of Clinical Psychology in Medical Settings*, and his area of scholarly work is in healthcare and health policy. He is recipient of both the *APA Heiser Presidential Award for Advocacy* and *Cynthia D. Belar Education Advocacy Distinguished Service Award*. Dr. Rozensky is a cochair of the 2018 APA presidential initiative, Citizen Psychologist, and has participated in six presidential initiatives.

References

- Advisory Committee on Interdisciplinary Community Based Linkages. (2011). *Preparing the interprofessional healthcare workforce to address health behavior change: Ensuring a high quality and cost-effective healthcare system (10th Annual Report to Congress and the Secretary of DHHS)*. Rockville, MD: Health Resources and Services Administration.
- Belar, C. B. (2016). Interprofessional education and training. In J. C. Norcross, G. R. VandenBos, & D. K. Freedheim (Eds.), *APA handbook of clinical psychology* (pp. 153–160). Washington, DC: American Psychological Association.
- Berwick, D. M. (2009). What “patient-centered” should mean: Confession of an extremist. *Health Affairs*, 28(4), w555–w565.
- Berwick, D. M. (2016). Era 3 for medicine and health care. *Journal of the American Medical Association*, E1–E2. Retrieved from <https://jamanetwork.com/journals/jama/fullarticle/2499845>
- Berwick, D. M., Nolan, T. W., & Whittington, J. (2008). The triple aim: Care, health and cost. *Health Affairs*, 27(3), 759–769.
- Blount, A., Schoenbaum, M., Kathol, R., Rollman, B. L., Thomas, M., O’Donohue, W., & Peek, C. J. (2007). The economics of behavioral health services in medical settings: A summary of the evidence. *Professional Psychology: Research and Practice*, 38(3), 290–297. doi:10.1037/0735-7028.38.3.290
- Bodenheimer, T., Chen, E., & Bennett, H. D. (2009). Confronting the growing burden of chronic disease: Can the US health care workforce do the job? *Health Affairs*, 28, 64–74. doi:10.1377/hlthaff.28.1.64
- Carter Center. (2011). Five prescriptions for ensuring the future of primary care. *Proceedings from the Health Education Summit*, October 5 and 6, 2010. Retrieved from <http://www.cartercenter.org/news/pr/ReinvigoratingPrimaryCareSystem.html>.
- Cubic, B., Mance, J., Turgesen, J. N., & Lamanna, J. D. (2012). Interprofessional education: Preparing psychologists for success in integrated primary care. *Journal of Clinical Psychology in Medical Settings*, 19, 84–92. doi:10.1007/s10880-011-9291-y
- D’Amout, D., & Oandasan, I. (2005). Interprofessionality as the field of interprofessional practice and interprofessional education: An emerging concept. *Journal of Interprofessional Care*, (Suppl. 1), 8–20.

- Fisher, E. S., Staiger, D. O., Bynum, J. P. W., & Gottlieb, D. J. (2007). Creating accountable care organizations: The extended hospital medical staff. *Health Affairs*, 26, w44–w57. doi:10.1377/hlthaff.26.1.w44
- France, C. R., Masters, K. S., Belar, C. D., Kerns, R. D., Klonoff, E. A., Larkin, K. T., ... Thorn, B. E. (2008, 39). Application of the competency model to clinical health psychology. *Professional Psychology: Research and Practice*, 573–580. doi.org/10.1037/0735-7028.39.6.573
- Frenk, J., Chen, L., Bhutta, Z. A., Cohen, J., Crisp, N., Evans, T., ... Zurayk, H. (2010). Health professionals for a new century: transforming education to strengthen health systems in an interdependent world. *The Lancet*, 376(9756), 1923–1958. doi:10.1016/s0140-6736(10)61854-5
- Goldstein, A., & Eilperin, J. (2014, April 1). More than 7 million Americans have enrolled under Affordable Care Act, White House says. *Washington Post*. Retrieved from https://www.washingtonpost.com/national/health-science/enrollment-under-the-affordable-care-act-on-track-to-reach-7-million/2014/04/01/1f6b8b96-b99b-11e3-9a05-c739f29ccb08_story.html
- Gruber, J. (2011). *Health care reform*. New York, NY: Hill and Wang.
- Health Insurance Portability and Accountability Act. (1996). P.L. 104–191, 42 U.S.C. 1320d.
- Health Service Psychology Education Collaborative. (2013). Professional psychology in health care services: A blueprint for education and training. *American Psychologist*, 68, 411–426. doi:10.1037/a0033265
- Institute of Medicine. (2013). Interprofessional Education for Collaboration: Learning how to improve health from interprofessional models across the continuum of education to practice. *Workshop Summary*.
- Interorganizational Work Group on Competencies for Primary Care Psychology Practice. (2013). *Competencies for psychology practice in primary care*. Retrieved from <http://www.apa.org/ed/resources/competencies-practice.pdf>
- Interprofessional Education Collaborative. (2011, February 16 and 17). *Conference proceedings of the team-based competencies: Building a shared foundation for education and clinical practice*. Washington, DC.
- Interprofessional Education Collaborative Expert Panel. (2011). *Core competencies for interprofessional collaborative practice: Report of an expert panel*. Washington, DC: Interprofessional Education Collaborative.
- Interprofessional Professionalism Collaborative. (2014). *Examples of IPP behaviors*. Retrieved from <http://www.interprofessionalprofessionalism.org/behaviors.html>.
- Jacobs, L., & Skocpol, T. (2016). *Health care reform and american politics: What everyone needs to know* (3rd ed.). New York, NY: Oxford University Press.
- Janicke, D., Fritz, A., & Rozensky, R. H. (2015). Healthcare reform and preparing the future clinical child and adolescent psychology workforce. *Journal of Clinical Child and Adolescent Psychology*, 44, 1–10. doi:10.1080/1537416.2015.1050725
- Kaslow, F. W., Patterson, T., & Gottlieb, M. (2011). Ethical dilemmas in psychologists accessing Internet data: Is it justified? *Professional Psychology: Research and Practice*, 42(2), 105–112. doi:10.1037/a0022002
- King v. Burwell. (2015). 576 U.S. 988.
- Mcdonough, J. E., & Adashi, E. Y. (2014). Realizing the Promise of the Affordable Care Act—January 1, 2014. *Jama*, 311(6), 569. doi:10.1001/jama.2013.286067
- Mortiere, P. (2013, November 15). Timeline of botched ObamaCare rollout. *The Hill*. Retrieved from <https://thehill.com/blogs/blog-briefing-room/news/190485-timeline-of-botched-implementation-of-obamacare>
- National Federation of Independent Business v. Sebelius. (2012). 567 U.S. 519.
- Nutting, P. A., Crabtree, B. F., Miller, W. L., Stange, K. C., Stewart, E., & Jaen, C. (2011). Transforming physician practices to patient-centered medical homes: Lessons from the National Demonstration Project. *Health Affairs*, 30, 439–445. doi:10.1377/hlthaff.2010.0159
- Obama, B. (2016). United States health care reform. Progress to date and next steps. *Journal of the American Medical Association*, E1–E8. Retrieved from <http://jama.jamanetwork.com/>. doi:10.1001/jama.2016.9797

- Oberland, J. (2017). Repeal, replace, repair, retreat – Republican's health care quagmire. *The New England Journal of Medicine*. doi:10.1056/NEJMp1710391
- Office of Disease Prevention and Health Promotion. (2020, April 28). Healthy People. Retrieved from <https://www.healthypeople.gov/>
- Ortman, J. M., & Guarneri, C. E. (2009). *United States population projections: 2000 to 2050*. Retrieved from <http://www.census.gov/population/www/projections/analytical-document09.pdf>.
- Plaut, V. C. (2010). Diversity science: Why and how difference makes a difference. *Psychological Inquiry*, 21, 77–99.
- Postupak, R., & Rozensky, R. H. (2016). History of clinical health psychology. In S. Richards & L. Cohen (Eds.), *The Wiley encyclopedia of health psychology, volume III*. Oxford, UK: Wiley.
- Raczynski, J. M., & Leviton, L. C. (2004). Introduction to volume 2: Disorders of behavior and health. In J. M. Raczynski & L. C. Leviton (Eds.), *Handbook of clinical health psychology* (pp. 3–5). Washington, DC: American Psychological Association.
- Richards, M. M. (2009). Electronic medical records: Confidentiality issues in the time of HIPAA. *Professional Psychology: Research and Practice*, 40, 550–556. doi:10.1037/a0016853
- Rittenhouse, D. R., Shortell, S. M., & Fisher, E. S. (2009). Primary care and accountable care—Two essential elements of delivery system reform. *New England Journal of Medicine*, 361, 2301–2303. doi:10.1056/NEJMp0909327
- Rogers, M. (2009). Cultural competency training in professional psychology. In R. H. Dana & J. R. Allen (Eds.), *Cultural competency training in a global society* (pp. 157–173). New York, NY: Springer. doi:10.1007/978-0-387-79822-6_9
- Rozensky, R. H. (2008). Healthy people 2020: Good vision for psychology's future. *Independent Practitioner*, 28, 188–191.
- Rozensky, R. H. (2011). The institution of the institutional practice of psychology: Health care reform and psychology's future workforce. *American Psychologist*, 66, 794–808. doi:10.1037/a0024621
- Rozensky, R. H. (2012a). Health care reform: Preparing the psychological workforce. *The Journal of Clinical Psychology in Medical Settings*, 19, 5–11. doi:10.1007/s10880-011-9287-7
- Rozensky, R. H. (2012b). Psychology in academic health centers: A true healthcare home. *The Journal of Clinical Psychology in Medical Settings*, 19, 1–11. doi:10.1007/s10880-012-9312-5
- Rozensky, R. H. (2012c). A tale of words, the offspring of words, and the specialized use of words: A review of the *APA Dictionary of Clinical Psychology*. *PsycCRITIQUE*. October 17, 2012, Vol. 57, Release 41, Article 1.
- Rozensky, R. H. (2013a). A brief review of the implication of the affordable care act for the practice of psychology. *Journal of Psychology and Christianity*, 32, 353–357.
- Rozensky, R. H. (2013b). Quality education in professional psychology: Flowers blooming, flexner and the future. *American Psychologist*, 68, 701–716. doi:10.1037/a0033771
- Rozensky, R. H. (2014a). Implications of the *Affordable Care Act* for education and training in professional psychology. *Training and Education in Professional Psychology*, 8, 1–12. doi:10.1037/tep0000021
- Rozensky, R. H. (2014b). Implications of *The Patient Protection and Affordable Care Act*: Preparing the professional psychology workforce for primary care. *Professional Psychology: Research and Practice*, 200–211. doi:10.1037/a0036550
- Rozensky, R. H., Celano, M., & Kaslow, N. (2013). Implications of the affordable care act for the practice of family psychology. *Couples and Family Psychology: Research and Practice*, 2, 163–178. doi:10.1037/cfp0000009
- Rozensky, R. H., Grus, C. L., Belar, C. D., Nelson, P. D., & Kohout, J. L. (2007). Using workforce analysis to answer questions related to the internship imbalance and career pipeline in 12 professional psychology. *Training and Education in Professional Psychology*, 1, 238–248.
- Rozensky, R. H., & Janicke, D. M. (2012). Commentary: Health Care Reform and Psychology's Workforce: Preparing for the Future of Pediatric Psychology. *Journal of Pediatric Psychology*, 18, 1–10. doi:10.1093/jpepsy/jsr111

- Rozensky, R. H., Johnson, N. G., Goodheart, C. D., & Hammond, W. R. (2004). *Psychology builds a healthy world: Opportunities for research and practice*. Washington, DC: American Psychological Association.
- Rozensky, R. H., & Kaslow, N. J. (2012). Specialization and life long learning. In G. J. Neimeyer & J. M. Taylor (Eds.), *Continuing education: Types, roles, and societal impacts* (pp. 345–358). Hauppauge, NY: Nova Publishers.
- Scheutz, B., Mann, E., & Evertt, W. (2010). Educating health professionals collaboratively for team-based primary care. *Health Affairs*, 29, 1476–1480. doi:10.1377/hlthaff.2010.00526 78-000
- Schneiderman, N., & Speers, M. A. (2001). Behavioral science, social science and public health in the 21st century. In N. Schneiderman, M. A. Speers, J. M. Silva, H. Tomes, & J. H. Gentry (Eds.), *Integrating behavioral and social sciences with public health* (pp. 3–28). Washington, DC: American Psychological Association.
- Shachak, A., & Jadad, A. R. (2010). Electronic health records in the age of social networks and global telecommunications. *JAMA*, 303, 452–453. doi:10.1001/jama.2010.63
- Sittig, D. F., & Classen, D. C. (2010). Safe electronic health record use requires a comprehensive monitoring and evaluation framework. *JAMA*, 303, 450–451. doi:10.1001/jama.2010.61
- US Census Bureau. (2011). *2010 census shows America's diversity*. Retrieved from https://www.census.gov/newsroom/releases/archives/2010_census/cb11-cn125.html.
- Vincent, G. K., & Velkoff, V. A. (2010). *The next four decades: The older population in the United States: 2010 to 2050*. Current Population Reports (pp. 25–1138). Washington, DC: US Census Bureau.
- Wilson, S. L., Rozensky, R. H., & Weiss, J. (2010). The Advisory Committee on Interdisciplinary Community-based linkages and the federal role in advocating for interprofessional education. *Journal of Allied Health*, 39, 210–215.

Suggested Reading

- American Psychological Association. (2014). *A psychologist's guide to federal advocacy*. Washington, DC: American Psychological Association, Education Government Relations Office & Public Interest Government Relations Office. Retrieved from <http://www.apa.org/about/gr/advocacy/federal-guide.pdf>
- American Psychological Association. (2015). Guidelines for clinical supervision in health service psychology. *American Psychologist*, 70(1), 33–46. <http://dx.doi.org/10.1037/a0038112>
- APA Interprofessional Seminar on Integrated Primary Care Work Group. (2017). *A curriculum for an interprofessional seminar on integrated primary care*. Retrieved from <http://www.apa.org/education/grad/curriculum-seminar.aspx>
- Rozensky, R. H., Grus, C. L., Nutt, R. L., Carlson, C. I., Eisman, E. J., & Nelson, P. D. (2015). A taxonomy for education and training in professional psychology health service specialties. *American Psychologist*, 70, 21–32. doi:10.1037/a0037988
- Sikka, R., Morath, J. M., & Leape, L. (2015). The Quadruple Aim: Care, health, cost and meaning in work. *BMJ Quality and Safety*. doi:10.1136/bmjqs-2015-004160

Pediatric Psychology

Christina M. Amaro, Michael C. Roberts,
Andrea M. Garcia, and Jennifer B. Blossom

Clinical Child Psychology Program, University of Kansas, Lawrence, KS, USA

Pediatric psychology refers to the field of psychological research and clinical practice concerned with the complex interaction of physical health and psychological development and health of children and the families. From early in its development, the field has focused on the assessment and treatment of psychological and behavioral conditions related to pediatric medical conditions and behavioral and emotional concomitants of disease, developmental disorders, and injury. Roberts, Aylward, and Wu (2014a) summarized the activities of pediatric psychologists as providing:

(1) psychosocial services for issues related to pediatric health conditions (e.g., fostering coping and adjustment to the diagnosis of a chronic illness, improving adherence to a medical treatment regimen, pain management, school reintegration); (2) psychological services for mental health problems appearing in medical settings along with a pediatric problem (e.g., behavioral disobedience after hospitalization); (3) assessment and treatment for psychological problems presenting in a medical setting without a concomitant medical condition (e.g., through primary care referrals for attention-deficit/hyperactivity disorder); (4) programs for health promotion, disease and injury prevention, and early intervention; (5) assessment, intervention, and programming to improve functioning for children and adolescents with intellectual and developmental disabilities; and (6) advocacy for public policy supporting children and families and promoting public health advancements. (p. 6)

Pediatric psychologists work in a variety of settings such as inpatient units in children's hospitals, outpatient clinics with pediatricians or family medicine physicians in primary care, interdisciplinary child guidance clinics, specialty clinics and centers for conditions such as developmental disorders or epilepsy, and community agencies and entities to provide services for children outside medical units (e.g., summer camps, after-school programs). Many pediatric psychologists practice in children's hospitals and pediatric clinics to provide psychological services to improve children's functioning with acute and chronic pediatric health conditions. Researchers investigate the relationships of psychological phenomena and health problems,

the outcomes of interventions and psychological programs, and the impact of policies and programs for children's healthy development. These roles and functions of pediatric psychologists, the problems, interventions, practice settings, and integrative science are overviewed in this entry. Roberts and Steele (2017) and Roberts, Aylward, and Wu (2014b) present more comprehensive coverage of these issues.

Historical Foundations

A "role model" article in the *American Psychologist* written by Logan Wright in 1967 is often recognized as a founding document for the field, although many commentators had called for collaboration between pediatrics and psychology as far back as the early 1900s. At that time (and to some extent now), pediatricians had little training, insufficient ability, and limited time to respond to the rise of psychosocial issues related to medical, developmental, psychological, behavioral, emotional, and educational problems that were appearing with greater frequency in pediatric practices. Thus, the field arose to deal more effectively with these multiple challenges (Roberts et al., 2014a). Although pediatric psychology concepts and organization predated those of health psychology, the field shares many considerations in common through a child health psychology orientation.

Wright's article in 1967 provides guideposts for evaluating the field today. He articulated that the developing field needed to (a) establish an identity through a formal organization, (b) focus on professional training in the specialty, and (c) construct new knowledge through applied research. The first component was successfully achieved with the founding of the Society of Pediatric Psychology (SPP) in 1968 with 75 full members and 22 affiliate members; it functioned as a special interest group within the Section on Clinical Child Psychology in the Division of Clinical Psychology of the American Psychological Association (APA). The organization provided an organizational home and information exchange for psychologists scattered across the country. By 1972, SPP had expanded to 650 members and later became a Section in the Division; in 2000 SPP became Division 54 of the APA. In 2016, there were 2,038 members (including 660 students and several international members).

As the organizational home for the field, SPP has provided formal definitions revealing the evolution of concepts. In 1976, the masthead of the *Journal of Pediatric Psychology* indicated: "The field and the contents of this Journal are defined by the interests and concerns of psychologists who work in interdisciplinary settings such as children's hospitals, developmental clinics, and pediatric or medical group practices." In 1988, in recognition that the field had expanded its coverage, pediatric psychology was defined for the journal as an "interdisciplinary field addressing the full range of physical and mental development, health, and illness issues affecting children, adolescents, and families...[such as] the relationship between psychological and physical well-being of children and adolescents including: understanding assessment, and intervention with developmental disorders; evaluation and treatment of behavioral and emotional problems and concomitants of disease and illness; the role of psychology in pediatric medicine; the promotion of health and development; and the prevention of illness and injury among children and youth" (Roberts, La Greca, & Harper, 1988, p. 2). According to the SPP website, an updated "Mission Statement" of the organization presents that "SPP aims to promote the health and psychological well being of children, youth and their families through science and an evidence-based approach to practice, education, training, advocacy, and consultation." An even more concise organizational "Vision Statement" states "Healthier children, youth, and families."

In terms of the professional training needs outlined by Wright (1967), the field started giving specific attention to training issues in the 1980s. Later, an SPP task force focused on the knowledge, attitudes, and skills of a competent pediatric psychologist in terms of general training domains expanded to cover the effects of pediatric medical treatments, children's development and disease processes, and child health psychology (Spirito et al., 2003). More recently, another SPP task force formulated an elaborated set of training sequences for the field even more distinctive from other areas of psychological application (Palermo et al., 2014). Thus, professional training sequences are increasingly formalized through doctoral, internship, and postdoctoral experiences, fulfilling Wright's articulated needs for the field.

In the third component outlined by Wright for building the field, developing a knowledge base, pediatric psychologists have dramatically expanded the scientific basis for understanding children's development, the relationship of psychological constructs and approaches with pediatric conditions, and evidence-based assessment and intervention to improve functioning. Pediatric psychology research is published in many different journals. SPP sponsors two professional journals for reports of research and clinical applications (*Journal of Pediatric Psychology*, *Clinical Practice in Pediatric Psychology*) and the publication of the *Handbook of Pediatric Psychology* (now in its fifth edition; Roberts & Steele, 2017). SPP holds an annual conference with over 600 attendees for interactions and education on the multitude of research and clinical activities of pediatric psychologists. Additionally, pediatric psychology researchers and practitioners participated in interdisciplinary conferences in pain, obesity, diabetes, sleep, and other child health-oriented areas. All of the components that Wright outlined have been successfully achieved. Thanks to contributions of multiple leaders and the ongoing work of pediatric psychologists in multiple locations, the field is thriving with professional opportunities to improve children's mental and physical health.

Perspectives and Systems

Pediatric psychologists take a developmental perspective that recognizes the progression of change over time in childhood. This perspective orients the professional to expect change to occur and be alert to development gone awry, when intervention and prevention services should be provided, and how to provide psychological services that return the child to appropriate developmental functioning. Pediatric psychologists also ascribe to an expanded biopsychosocial model that identifies multiple systems including families, schools, peers, and healthcare, which affect children and their functioning, particularly as these systems might interact over changes in the child and family due to psychological and physical health conditions (Steele & Aylward, 2009; Wu, Aylward, & Roberts, 2014).

Ethical Issues

Psychological practice and research invoke ethical and legal issues such as informed consent and assent, privacy, confidentiality, and record keeping. Pediatric psychologists also frequently deal with ethical issues particular to interprofessional relationships in medical settings and treatment challenges associated with improving psychological functioning of children with medical conditions and their families. These involve decision making and substituted judgment about withholding or changing medical and health treatments, self-responsibility for care, and disability access to care, among others. Pediatric psychologists work within numerous

legal and ethical guidances including federal and state regulations (e.g., educational and healthcare laws, discrimination laws and rulings, professional licensing requirements), institutional policies and practices (e.g., electronic health records), and the Ethical Principles of Psychologists and Code of Conduct (APA, 2010).

Major and Crosscutting Issues

There are several major and crosscutting issues that occur across a range of areas. Outlined below are some exemplars within the field of pediatric psychology: adjustment to chronic illness, consultation–liaison (CL) services, primary care, adherence, sleep, pain, and health promotion and prevention.

Adjustment to Chronic Illness

A large component of pediatric psychology research and clinical practice relates to youth and family's adjustment to and coping with medical conditions and treatments. There are many aspects of treatment of chronic illnesses that can have an impact on children and adolescents, including brief procedures (e.g., injections), invasive methods (e.g., bone marrow aspiration), and adverse side effects (e.g., nausea, sleepiness). Some youth may experience relatively short periods of adjustment (e.g., experiencing brief infection), whereas other illnesses are lifelong and require ongoing adjustment (e.g., diabetes; Kazak, Sood, & Roberts, 2016). Many youth and their families demonstrate good coping mechanisms and adjustment, whereas others may need additional support, and pediatric psychologists work with them to determine appropriate approaches given their specific concerns and needs (Kazak et al., 2016).

Consultation–Liaison Services

As noted earlier, pediatric psychologists work in a variety of settings, such as hospitals and medical academic centers, and perform numerous roles in these environments. One important activity is CL services in the hospital (Kazak et al., 2016). When providing inpatient consultation, a pediatric psychologist may receive a referral from a physician or a specific service to conduct an assessment of a patient and provide recommendations for treatment. Pediatric psychologists typically respond to a referral within 24 hr and communicate impressions and recommendations back to the physician and/or team members in a timely matter. However, they may also be involved in the longer-term implementation of treatment recommendations (Kazak et al., 2016). In a liaison position, a pediatric psychologist may be a team member of subspecialty service (e.g., gastroenterology) and has a portion of time dedicated to providing mental health services. Overall, CL includes a range of assessment (e.g., screening, evaluation of psychological functioning) and indirect or direct treatment (e.g., teaching coping skills, assisting with adjustment to medical condition, enhancing adherence) services to promote the psychosocial well-being of youth with chronic illnesses and their families.

Primary Care

Pediatric psychologists may also work in primary care settings, such as pediatric and family medicine clinics. Within primary care settings, pediatric psychologists provide mental and behavioral health services (e.g., assessment, treatment, consultation) that span a range of

concerns (e.g., prevention, health promotion, psychosocial functioning), and they work alongside other medical providers, such as pediatricians, nurse practitioners, and family medicine physicians to enhance children's mental and physical health (Stancin, Sturm, Tynan, & Ramirez, 2017). The number of psychologists working in this type of setting as well as research in this area is limited compared with tertiary care or hospital practice. However, effective models for pediatric psychology activities in primary care have been developed. For example, Schroeder established a model in the 1970s in which psychologists provided behavioral services to patients and families seen in a pediatric clinic. Psychologists and their trainees served in multiple roles, including therapist, case manager, educator, consultant, and researcher with the pediatricians and their staff (Stancin et al., 2017). Due to recent changes in the United States' healthcare system, greater psychology involvement in primary care appears warranted. Pediatric psychologists, in particular, are prepared to provide services in numerous areas including prevention, screening and early intervention, psychoeducation to parents, brief individual psychotherapy for youth, and care coordination (Stancin et al., 2017). Further, many interventions for youth with chronic illnesses and their families (e.g., self-management training, relaxation skills) are also provided by pediatric psychologists in primary care settings.

Adherence

Adherence to medical regimens is important in the maintenance of health for youth with chronic illnesses, yet nonadherence continues to be a significant behavioral health concern, with a majority of individuals failing to follow treatment recommendations from healthcare providers (Hommel, Ramsey, Loiselle, & Ryan, 2017). Nonadherence has been associated with numerous negative consequences including diminished health-related quality of life, drug resistance, increased use of healthcare, and poorer treatment outcomes (Hommel et al., 2017). Further, research suggests that there are several modifiable and non-modifiable individual factors that influence pediatric adherence including cognitive functioning, illness knowledge, health beliefs, psychological or behavioral problems, and treatment awareness. Family (e.g., parental involvement in self-management, family functioning, parental psychopathology) and peer (e.g., social support, social stigma) factors may also influence youth treatment adherence (Hommel et al., 2017).

Although there are several types of interventions for pediatric nonadherence, recent meta-analyses and systematic reviews suggest that behavioral interventions (e.g., problem solving, rewards, monitoring and goal setting) may be effective in improving adherence to treatment (Hommel et al., 2017; Kazak et al., 2016). Additionally, interventions targeting family concerns (e.g., communication about illness-related responsibilities, parental involvement in disease management) may also be helpful in pediatric adherence (Hommel et al., 2017; Kazak et al., 2016).

Sleep

Sleep is important to children and adolescents' development, and youth may experience a range of pediatric sleep disorders including insomnia (e.g., problems falling and staying asleep) and parasomnias (e.g., events that disrupt sleep, such as sleepwalking). Sleep problems are associated with a range of negative outcomes including worse emotion regulation, increased externalizing symptoms, and diminished cognitive functioning (Honaker, Meltzer, & Mindell, 2017). Youth with chronic illnesses are likely to have comorbid sleep issues, and sleep problems may exacerbate medical symptoms (e.g., a bidirectional relationship between

sleep and pain). Further, research has suggested that sleep problems are particularly pronounced in youth with a range of medical conditions, such as asthma, traumatic brain injury, oncology, epilepsy, and cystic fibrosis (CF) (Honaker et al., 2017). Given that sleep is often an issue for youth, pediatric psychologists require training and a knowledge base in sleep in both typically developing youth and youth with chronic illnesses. Within clinical settings, an important role for pediatric psychologists is to assess pediatric sleep and provide treatment as necessary. There are multiple domains of sleep that a pediatric psychologist should assess for including bedtime routine (e.g., behaviors at bedtime, time it takes to fall asleep), sleep environment (e.g., location, room condition), sleep throughout the night (e.g., number of nighttime wakings, nocturnal enuresis), and how the child feels during the day (e.g., difficulties waking up and staying awake; Honaker et al., 2017). A range of effective behavioral strategies address pediatric sleep problems. For example, pediatric psychologists may help families to establish a regular bedtime routine to decrease bedtime resistance and night wakings (Honaker et al., 2017).

Pain

Similar to sleep, pain often occurs among typically developing children and adolescents as well as youth with chronic illnesses. There are various types of pain, such as procedural pain and recurrent or chronic pain. Children routinely undergo medical procedures (e.g., immunizations, stitches) that may result in pain. There are several strategies that can be implemented before, during, and after the procedure to assist youth with procedural pain and distress (Cohen et al., 2017). Using a developmentally appropriate approach, pediatric psychologists can assist parents with preparing their children for a more invasive medical procedure (e.g., bone marrow aspiration, burn dressing changes). Psychologists may discuss what information to provide to children ahead of time and what language to use. During a procedure, youth may benefit from a range of strategies, such as the use of distraction (e.g., bubbles, pop-up books) and relaxation techniques (e.g., deep breathing; Cohen et al., 2017). After a procedure, use of coping skills can be reinforced, and parents can reward children for good behavior during the procedure.

Chronic or recurrent pain refers to pain that occurs regularly (e.g., for 3 months or more). Recurrent pain may occur as the result of a severe injury or chronic illness (e.g., arthritis, sickle cell disease); however, sometimes an underlying cause is not identified, and this is known as idiopathic chronic pain (Law, Noel, Nagel, & Dahlquist, 2017). Youth with chronic pain often experience significant interferences in their daily lives, such as problems with sleep, school absences, and impaired social and physical functioning. Pediatric psychologists help to promote techniques for self-management, which include identifying specific environmental or psychosocial triggers. Cognitive and/or behavioral interventions are available for chronic pain and may consist of challenging and changing maladaptive thoughts around pain, relaxation strategies, and promotion of physical activity and well-being (e.g., sleep hygiene). Growing evidence exists for the use of acceptance and commitment therapy in the treatment of pediatric chronic pain (Law et al., 2017).

Health Promotion and Prevention

Prevention refers to engaging in behaviors to avoid the development of problems, whereas health promotion refers to the adoption of health behaviors to promote a healthy lifestyle. Further, behaviors developed in childhood may impact youth's quality of life, and those

behaviors also have the potential to persist throughout adulthood. Therefore, it is important for pediatric psychologists to conduct research and administer interventions related to health promotion and prevention in youth. Historically, health promotion and prevention have been topics of interest within pediatric psychology; however, these areas appear to have received less attention in comparison to issues related to pediatric chronic illnesses. Given recent changes in healthcare reform, more opportunities may be available to help support health promotion and prevention activities (Kazak et al., 2016).

There are several examples of prevention applications in pediatric psychology including seat belt safety and the use of immunizations to prevent diseases. Further, pediatric psychologists have conducted research and developed interventions to prevent and control childhood injuries. Health promotion efforts have examined increasing pediatric health behaviors such as physical activity, sleep, and diet to promote healthy lifestyles and decrease the risk of disease (e.g., obesity, type 2 diabetes).

Specific Examples Within Pediatric Psychology

Pediatric psychologists encompass many roles, including consultant, researcher, and an evidence-based practice provider. As clinicians, many pediatric psychologists work with and may conduct interventions for a wide variety of pediatric conditions. However, the approach to any intervention involves conceptualization of the presenting problems and understanding that the child functions within a system. Approaches for case conceptualization often use the biopsychosocial model noted earlier, which examines the extent to which biological, psychological, and social factors as well as their interactions influence health and illness. Understanding such reciprocal multidimensionality of pediatric conditions, pediatric psychologists often work as part of an interdisciplinary or interprofessional team to help manage and intervene with the health issues related to pediatric conditions.

Pediatric psychologists implement various psychosocial treatments focused on individual treatment as well as provide support to the child's family. Moreover, considerations for appropriate interventions include the child's developmental level as well as a biopsychosocial approach. Pediatric psychologists have successfully employed effective treatments with frequently used individual approaches including behavioral techniques, cognitive techniques, cognitive behavioral therapy (CBT), or a multicomponent approach in which multiple strategies are integrated to address the presenting problems. For example, psychological interventions promoting adherence to the medical regimen by children with chronic illness frequently employ the use of multiple components. Behavioral procedures involve contingency management approaches to help improve self-control (e.g., behavioral disobedience after hospitalization), pain management, and self-management in order to increase medical treatment adherence. CBT involves identifying unhelpful thoughts related to either disease or treatment and challenging obstructive thoughts through cognitive restructuring and developing adaptive behaviors (e.g., problem solving and functional coping). Lastly, cognitive techniques involve self-management such as cognitive coping skills to reduce anxiety, impulse control, and depression. Pediatric psychologists also work with families of children with pediatric conditions, providing psychosocial interventions focused extensively on addressing barriers, psychosocial targets, family functioning, and intensive crisis intervention. The following sections discuss examples of how pediatric psychologists apply evidence-based practice in pediatric health conditions: asthma, oncology, CF, and type 1 diabetes (T1D).

Asthma

Asthma is a chronic inflammatory disorder of the airways that causes the airways to swell and narrow leading to shortness of breath, chest tightness, and coughing. Challenges faced by children with asthma and their families include frequent missed school days and hospital visits. Typical treatments for managing symptoms include self-management, control of environmental triggers (e.g., allergens, stress), and prescribed pharmacological treatments. Pediatric psychologists help self-management and adherence to prescribed treatments by providing behavioral educational and technology-based interventions. For example, web-based delivery of intervention content focuses on self-management and symptom management. Similarly, pediatric psychologists also provide the family with educational information on how to manage their child's asthma, as well as behavioral and educational interventions to promote self-management for children with asthma.

Oncology

Advances in modern medicine have significantly increased the survival rate of children with cancer; however, diagnosis, treatment, and surviving cancer are compounded with psychological, social, and biological challenges. Specific challenges include neurological challenges (e.g., problems with executive functioning, processing speed, attention, working memory), health challenges (e.g., endocrine problems, cardiopulmonary), and psychosocial challenges (e.g., isolation, depression, pediatric medical traumatic stress). Interventions for children with cancer focus on social skills (e.g., forming new friendships), psychological well-being (e.g., benefit finding, self-efficacy managing internalizing behaviors), and transitional skills to independent functioning (e.g., independent living, medication adherence). Moreover, interventions help children and adolescents manage the negative side effects of chemotherapy through the use of cognitive coping strategies.

Cystic Fibrosis

CF is a progressive genetic, life-limiting illness, which primarily affects the functioning of the lungs and pancreas through abnormal mucus secretions. Given the progressive and disabling prognosis, CF is associated with many challenges including psychological adjustment, diminished quality of life, and adherence concerns. Treatment is often complex and can be intrusive, involving numerous medications, dietary changes, and exercise; prognosis is largely dependent upon these behavioral changes. However, children frequently do not adhere to their medical regimens. Reasons for this noncompliance include the aversive nature of the treatments, length of the regimen, and the child's developmental level. Interventions to increase the adherence of medical regimens for children with CF often include parental strategies and frequently involve multicomponent incorporating psychoeducational, parent training, exercise, and nutritional management. Further, these interventions encompass cognitive and behavioral strategies for child and parents. For instance, behavioral management interventions seek to decrease negative parent-child interactions around treatment adherence, exercise programs, and nutritional intake.

Diabetes

T1D is an endocrine disorder in pediatric populations, caused by an autoimmune process that destroys insulin-producing cells that regulate glucose, fat, and protein metabolism. Treatment

of T1D encompasses a complex regimen, which includes dietary management, blood glucose checks, frequent daily insulin injections or an insulin pump, and regular exercise. Challenges for children with T1D include frequent hospitalizations due to limited adherence to regimen and poor stress management. Similar to other pediatric conditions, nonadherence with the diabetes treatment program significantly influences prognosis and likelihood of medical complications including hospitalizations. Behavioral interventions and psychosocial interventions may focus on enhancing adherence to treatment regimens, improving family functioning, and reducing psychosocial stress.

Overall, pediatric psychologists provide effective and evidence-based treatments for various pediatric conditions in hospitals and other diverse interdisciplinary settings. Pediatric psychologists receive training that allows them to translate research into practice, conceptualize presenting concerns through a biopsychosocial perspective, and provide individual patient and family support and treatment. Understanding that children and adolescents do not exist in a microcosm, pediatric psychologists work to provide psychological support for children and adolescents in order to help them be successful at home, school, and with peers.

Future Directions in Clinical Practice and Research

eHealth

One future direction in pediatric psychology is the use of digital technologies to improve youth and family health, which is also known as eHealth. Electronic devices can be used to assist with a range of functions within pediatric psychology, including measuring health behaviors (e.g., wearable device that tracks physical activity and sleep) or adherence (e.g., electronic sensors that track the number of times a prescription bottle is opened). Additionally, eHealth interventions (e.g., web based, text messages) have been implemented to manage conditions as well as promote health behaviors (Kazak et al., 2016). However, there are some unique challenges in the use of eHealth interventions, including issues with keeping up with the most current technologies. Additionally, eHealth interventions may be costly and require a significant amount of time to develop initially. However, eHealth interventions may help to increase access to pediatric psychology interventions (Kazak et al., 2016).

Mentorship

From its roots, pediatric psychology has placed substantial emphasis on the role of mentorship within the field, covering all areas of practice from research (both project conceptualization and through the peer-review process) to clinical practice. Recent and developing initiatives continue to shape and contribute to the mentorship process. For example, SPP coordinates a mentorship program between students and career psychologists. Important future directions for such programs might include mentorship of undergraduate students. Further, this process would provide an opportunity for graduate students to develop mentoring experience that they might carry over into their professional careers.

Bridging the Gap and Crosscutting Issues

Fundamental to the field of pediatric psychology is the focus on integration of research and practice. Thus, the future direction of the field of pediatric psychology as a whole assumes these are two interrelated processes, rather than distinct and wholly separate phenomena. In

line with broader healthcare practice, pediatric psychologists often provide clinical services within the context of interdisciplinary, or interprofessional teams, a more common practice than the traditional one-on-one model. However, an ongoing limitation in this area is the lack of strong, clearly defined evidence-based practice protocols across all health conditions (similar to adult health psychology). The integrative research–practice model will likely improve this situation as the field develops.

Another potential method of bridging the potential gap between the research–practice arenas is through quality improvement activities. Within interprofessional programs, quality improvement studies gather information about ongoing practice to make developmental changes and assess for effects within incremental changes made as indicated. For example, quality improvement research may target adherence or examine immunization procedures. Randomized control trials, or large-scale research projects within existing clinics, although important for some areas, face numerous functional limitations. The need for information about process and outcomes can be addressed through quality improvement. Quality improvement projects represent an important focus for future directions in pediatric psychology, providing multiple benefits over traditional research studies, including immediate application within functioning clinics and provision of cost–benefit information.

Although the future of pediatric psychology will likely continue to focus on the psychosocial needs of children with chronic disease, the field will also grow through research and applications in preventive integrated primary care programs. While prevention and primary care efforts have historical roots within pediatric psychology, these also represent essential areas for future research and practice. Continued research and program development are needed to identify effective models of preventive interventions and psychosocially based care within primary care settings. Another important future direction in the field of pediatric psychology will be to understand collaboration processes and to develop interprofessional research and services teams. Many of the challenges facing children and adolescents in physical and mental health require solutions integrated from different disciplines and viewpoints in recognition of the biopsychosocial model including collaboration and coordination, such as examining and facilitating the input and contributions of multiple professionals.

See Also

Child and family health; childhood cancer; childhood obesity; family and health; adherence; health promotion

Author Biographies

Christina M. Amaro, MA, is a doctoral candidate in the Clinical Child Psychology Program at the University of Kansas. Her research interests include pediatric health promotion, child and family adjustment to chronic illness, program evaluation, and professional psychology issues, such as mentoring and training.

Michael C. Roberts, PhD, ABPP, is the dean of Graduate Studies and professor in the Clinical Child Psychology Program at the University of Kansas. His research and professional interests revolve around the application of psychology to improving physical and mental health, particularly for children and adolescents, and in professional issues of health service psychology.

Andrea M. Garcia, MEd, received her MA from the Clinical Child Psychology Program at the University of Kansas. She has interests in autism spectrum disorders and developmental outcomes, neonatal intensive care unit, resilience in children and families with chronic illness, and health promotion.

Jennifer B. Blossom, MS, is a doctoral candidate in the Clinical Child Psychology Program at the University of Kansas and psychology resident at the University of Washington School of Medicine—Child Track. Her research interests include the development, dissemination, and implementation of evidence-based assessment and intervention for youth internalizing problems in community settings, with an emphasis on integrated care settings (e.g., primary care), as a method to reduce mental health disparities and increase service use, access, and efficiency.

References

- American Psychological Association. (2010). Ethical principles of psychologists and code of conduct. *American Psychologist*, 57, 1060–1073.
- Cohen, L. L., Blount, R. L., Chorney, J., Zempsky, W., Rodrigues, N., & Cousins, L. (2017). Management of pediatric pain and distress due to medical procedures. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (5th ed., pp. 146–160). New York, NY: Guilford Press.
- Hommel, K. A., Ramsey, R. R., Loiselle, K., & Ryan, J. L. (2017). Adherence to pediatric treatment regimens. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (5th ed., pp. 119–133). New York, NY: Guilford Press.
- Honaker, S., Meltzer, L. J., & Mindell, J. (2017). Pediatric sleep. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (5th ed., pp. 452–466). New York, NY: Guilford Press.
- Kazak, A. E., Sood, E., & Roberts, M. C. (2016). Pediatric psychology. In J. C. Norcross, G. R. VandenBos, & D. K. Freedman (Eds.), *APA handbook of clinical psychology* (pp. 81–106). Washington, DC: American Psychological Association.
- Law, E. F., Noel, M., Nagel, M. S., & Dahlquist, L. M. (2017). Chronic and recurrent pain. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (5th ed., pp. 134–145). New York, NY: Guilford Press.
- Palermo, T., Janicke, D., McQuaid, E., Mullins, L., Robins, P., & Wu, Y. (2014). Recommendations for training in pediatric psychology: Defining core competencies across training levels. *Journal of Pediatric Psychology*, 39, 965–984.
- Roberts, M. C., Aylward, B. S., & Wu, Y. P. (2014a). Overview of the field of pediatric psychology. In M. C. Roberts, B. Aylward, & Y. P. Wu (Eds.), *Clinical practice of pediatric psychology* (pp. 3–16). New York, NY: Guilford Press.
- Roberts, M. C., Aylward, B. S., & Wu, Y. P. (Eds.) (2014b). *Clinical practice of pediatric psychology*. New York, NY: Guilford Press.
- Roberts, M. C., La Greca, A. M., & Harper, D. C. (1988). *Journal of Pediatric Psychology*: Another stage of development. *Journal of Pediatric Psychology*, 13(1), 1–5.
- Roberts, M. C., & Steele, R. C. (Eds.) (2017). *Handbook of pediatric psychology* (5th ed.). New York, NY: Guilford Press.
- Spirito, A., Brown, R. T., D'Angelo, E., Delamater, A., Rodrigue, J., & Siegel, L. (2003). Society of pediatric psychology task force report: Recommendations for the training of pediatric psychologists. *Journal of Pediatric Psychology*, 28(2), 85–98.
- Stancin, T., Sturm, L., Tynan, D., & Ramirez, L. (2017). Pediatric psychology and primary care. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (5th ed., pp. 550–565). New York, NY: Guilford Press.

- Steele, R. G., & Aylward, B. S. (2009). An overview of systems in pediatric psychology research and practice. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (4th ed., pp. 649–655). New York, NY: Guilford Press.
- Wright, L. (1967). The pediatric psychologist: A role model. *American Psychologist*, 22(4), 323–325.
- Wu, Y. P., Aylward, B. S., & Roberts, M. C. (2014). Cross-cutting issues in pediatric psychology. In M. C. Roberts, B. Aylward, & Y. P. Wu (Eds.), *Clinical practice of pediatric psychology* (pp. 32–45). New York, NY: Guilford Press.

Suggested Reading

- Duncan, C. L., Mentrikoski, J. M., Wu, Y. P., & Fredericks, E. M. (2014). Practice-based approach to assessing and treating nonadherence in pediatric regimens. *Clinical Practice in Pediatric Psychology*, 2(3), 322–336.
- Fisher, E., Heathcote, L., Palermo, T. M., de Williams, A. C., Lau, J., & Eccleston, C. (2014). Systematic review and meta-analysis of psychological therapies for children with chronic pain. *Journal of Pediatric Psychology*, 39(8), 763–782.
- Goethals, E. R., Oris, L., Soenens, B., Berg, C. A., Prikken, S., Van Broeck, N., ... Luyckx, K. (2017). Parenting and treatment adherence in type 1 diabetes throughout adolescence and emerging adulthood. *Journal of Pediatric Psychology*, 42(9), 922–932.
- McGrady, M., & Hommel, K. (2016). Targeting health behaviors to reduce health care costs in pediatric psychology: Descriptive review and recommendations. *Journal of Pediatric Psychology*, 41(8), 835–848.
- Tillery, R., Cohen, R., Berlin, K. S., Long, A., & Phipps, S. (2017). Youth's adjustment to cancer: Examination of patterns of adjustment and the role of peer relations. *Journal of Pediatric Psychology*, 42(10), 1123–1132.

Placebo Effect

Diletta Barbiani¹ and Fabrizio Benedetti²

¹*Department of Neuroscience, University of Turin Medical School, Turin, Italy*

²*Hypoxia Medicine, Plateau Rosà, Switzerland*

Definition

Placebos are often defined as inert substances, devoid of any therapeutic property for the condition being treated. However, this definition is not completely correct, because placebos are made of many things, such as words and rituals, symbols, and meanings, and all these elements are active in shaping the patient's brain. Thus, a better definition of placebo should embrace both the inert substance and the psychosocial context around the patient and the therapy. Although inert substances are of great validity in the clinical trial setting, the clinical trialist has always drawn attention to the inertness of the substance itself, thus diverting it from the real meaning of placebo. If drawing attention to the inert substance is correct in pragmatic clinical trials, whereby the only purpose is to see whether drugs are better than placebos, this surely does not help understand what a placebo is (Benedetti, 2014).

A real placebo effect is a psychobiological phenomenon occurring in the patient's brain following the administration of an inert substance or of a sham physical treatment such as sham surgery, along with verbal suggestions (or any other cue) of clinical benefit (Price, Finniss, and Benedetti, 2008). Saline solutions or sugar pills do not have intrinsic therapeutic properties. Instead, the effect is due to the psychosocial context that surrounds the inert substance and the patient. In this sense, to the clinical trialist and to the neuroscientist, the term "placebo effect" has different meanings. Whereas the former is interested in any improvement that may occur in the group of patients who take the inert substance, regardless of its origin, the latter is only interested in the improvement that derives from active processes occurring in the patient's brain. In fact, the improvement in patients who are given a placebo can be ascribed to a vast array of factors, such as spontaneous remission of the disease (the so-called natural history), regression to the mean (a statistical phenomenon due to selection biases), patient's and doctor's biases, and unidentified effects of co-interventions. In pragmatic clinical trials, the trialist is interested in the improvement irrespective of its cause, because

he/she only needs to establish whether the patients who take the true treatment, be it pharmacological or not, are better off than those who take the placebo. Instead, if the interest is focused on the understanding of what a real placebo effect is and how it works, it becomes necessary to separate it from spontaneous remissions, regression to the mean, and biases (Benedetti, 2014).

Therefore, the true placebo effect is only the psychobiological phenomenon, and all the other phenomena can be ruled out by using the appropriate methodological approach. For example, in order to rule out spontaneous remission, the placebo group must be compared with a no-treatment group that serves as a measure of the natural history of the disease. Similarly, in order to rule out biases, such as those which may occur in subjective symptoms, objective outcome measures must be assessed.

The real placebo response, that is, the real psychobiological phenomenon, is not irrelevant, and its contribution to the clinical improvement is substantial. For example, in antidepressant clinical trials, it has been shown that the natural history of the disease (i.e., spontaneous remission) accounts for 23.87% of the overall effect, the real placebo effect (i.e., expectations of benefit) for 50.97%, and the drug effect for 25.16% only. Thus, placebo responses in depression are usually large, and they have been found to increase over time, with stronger placebo responses in more recent studies. A similar increase over time has been found in pain.

Today we know that there is not a single placebo effect, but many, with different mechanisms at play across different diseases, systems, and therapeutic interventions (Benedetti, 2013, 2014; Finniss, Kaptchuk, Miller, & Benedetti, 2010).

Psychobiological Mechanisms

Two main mechanisms have been the focus of attention: expectation and learning. Expectation is a conscious event whereby the subject expects a therapeutic benefit. The link between expectation and the clinical improvement is twofold. First, positive expectations may reduce anxiety, and anxiety is known to affect different symptoms, such as pain, in opposite directions, that is, either decrease or increase, depending on the circumstances. Second, expectation of a positive event, that is, the therapeutic benefit, may activate reward mechanisms. Learning mechanisms, ranging from behavioral conditioning to social learning, are crucial, because the previous experience of effective treatments leads to powerful placebo responses. Expectation and learning are not necessarily mutually exclusive, since learning can lead to the reinforcement of expectations or can even create *de novo* expectations. The following overview is aimed at briefly describing the main psychological mechanisms that have been identified in the past few years (see Benedetti, 2014, for a detailed review).

The opioid system activated by placebos is the most studied and understood (Figure 1a). The opioid antagonist, naloxone, prevents some types of placebo analgesia, thus indicating that the opioid system plays an important role. The cholecystikinin (CCK) antagonist, proglumide, enhances placebo analgesia on the basis of the anti-opioid action of CCK, whereas the activation of the CCK type 2 receptors with the agonist pentagastrin disrupts placebo analgesia. Therefore, the activation of the CCK type 2 receptors has the same effect as the μ -opioid receptor blockade, indicating that the balance between CCK and opioids is crucial in placebo responsiveness in pain (Figure 1a). Some brain regions in the cerebral cortex and the brainstem are affected by both a placebo and the opioid agonist remifentanyl, which suggests a related mechanism in placebo-induced and opioid-induced analgesia. In vivo receptor-binding techniques show that placebos activate μ -opioid neurotransmission in the

dorsolateral prefrontal cortex, the anterior cingulate cortex, the insula, and the nucleus accumbens. Recent studies in rodents confirm these pharmacological findings in humans.

The CCK pro-nociceptive system has also been found to mediate *nocebo* hyperalgesia (Figure 1b). The *nocebo* response is a phenomenon that is opposite to the placebo response, whereby negative expectations may lead to clinical worsening. For example, expectations of pain increase lead to *nocebo* hyperalgesia, and this increase can be blocked by the CCK antagonist proglumide. Anticipatory anxiety plays a key role here, for *nocebos* are anxiogenic and induce negative expectations. Again, a social defeat model of anxiety in rats supports this view.

When nonopioid drugs, like ketorolac, are administered for 2 days in a row and then replaced with a placebo on the third day, the placebo analgesic response is not reversed by naloxone, whereas the CB1 cannabinoid receptor antagonist, rimonabant, blocks this placebo analgesia completely. Therefore, in some circumstances, for example, following previous exposure to nonopioid drugs, placebo analgesia is mediated by the CB1 cannabinoid receptors. Interestingly, there is compelling experimental evidence that the whole lipidic pathway, involving arachidonic acid, endogenous cannabinoid ligands (e.g., anandamide), and the synthesis of prostaglandins (PG) and thromboxane (TX), is important in the modulation of the placebo response in pain (Figure 1c). For example, the functional missense variant Pro129Thr of the gene coding fatty acid amide hydrolase (FAAH), the major degrading enzyme of endocannabinoids, affects the analgesic responses to placebo as well as placebo-induced μ -opioid neurotransmission. Moreover, cyclooxygenase (COX), which is involved in PG and TX synthesis (Figure 1c), has been found to be modulated by both placebo and *nocebo* in hypobaric hypoxia, or high altitude, headache with a mechanism similar to that of aspirin.

Hypoxia headache has been found to represent an excellent model to investigate these mechanisms. In fact, there are at least two pathways responsible for triggering this type of headache. The first is represented by the acute effects of hypoxia on PG synthesis through the COX enzyme, with the formation of PGD₂, PGF₂, PGE₂, PGI₂ (prostacyclin), and TX A₂. One of the most important effects of PGs, particularly of PGE₂, is represented by vasodilation, which is thought to be the principal factor inducing acute hypoxia headache. The second pathway is represented by hypoxia-related hyperventilation that, in turn, induces the excessive elimination of carbon dioxide (CO₂), with a subsequent increase in blood pH (alkalosis). In a recent study, Benedetti and Dogue (2015) used placebo oxygen inhalation and placebo aspirin administration, both proven to be effective treatments for high altitude headache, to induce placebo responses after repeated associations between headache reduction and either the oxygen mask ritual or the aspirin pill ritual. In other words, both placebos were given after a conditioning procedure, whereby either real oxygen or real aspirin was administered for three consecutive sessions to reduce headache pain. Interestingly, after oxygen conditioning, placebo oxygen induced pain relief along with a reduction in ventilation, blood alkalosis, and salivary PGE₂, though without any increase in blood oxygen saturation (SO₂). Instead, after aspirin conditioning, placebo aspirin induced pain relief through the inhibition of all the products of COX, namely, PGD₂, PGE₂, PGF₂, PGI₂, and TXA₂, yet without affecting ventilation and blood alkalosis. Therefore, two distinct therapeutic rituals used two different and independent pathways to reduce headache pain. Since a decrease in PGE₂ was observed in both situations, PGE₂ reduction might be the final common pathway of the analgesic effect of both the mask and the pill ritual.

Dopamine is involved in placebo responsiveness in at least two conditions: pain and Parkinson's disease. In placebo analgesia, an increase in dopamine binding to D₂/D₃ receptors and in opioid binding to μ -receptors occurs in the nucleus accumbens, whereas a decreased binding to the same receptors is present in *nocebo* hyperalgesia (Figure 1d). Likewise,

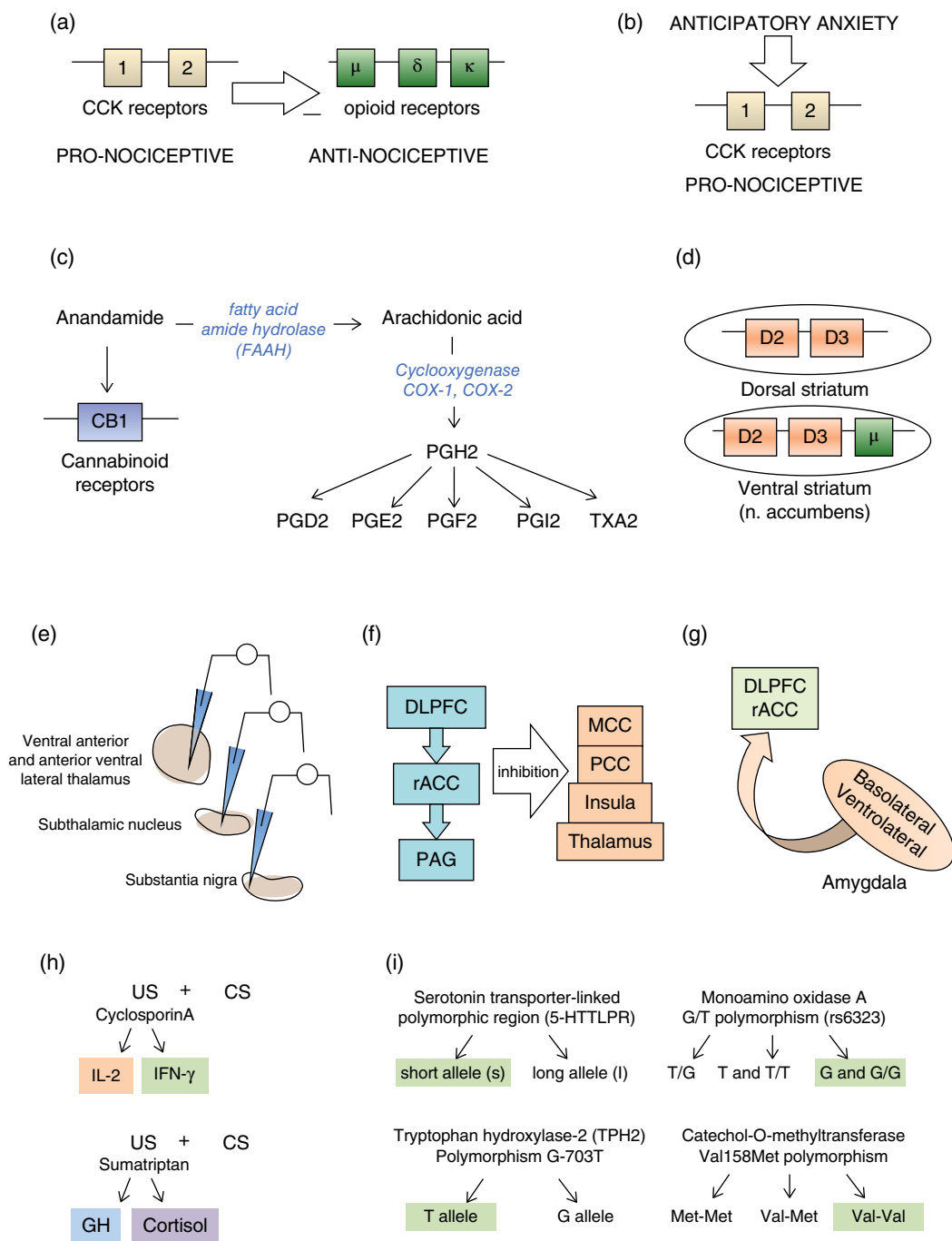


Figure 1 Principal neurobiological mechanisms of the placebo response that have been identified across a variety of conditions. (a) The antinociceptive opioid system is activated in placebo analgesia in some circumstances, and the μ -opioid receptors play a crucial role. The pro-nociceptive cholecystokinin (CCK) system antagonizes the opioid system, thus blocking placebo analgesia. (b) The pro-nociceptive CCK system is activated by anticipatory anxiety in nocebo hyperalgesia, with some evidence that the CCK-2 receptors are more important. (c) Different lipidic mediators have been identified in placebo analgesia and nocebo hyperalgesia. Whereas placebos activate the CB1 cannabinoid receptors and inhibit prostaglandin (PG) synthesis in some circumstances, nocebos increase PG synthesis. In addition, different genetic variants of FAAH affect the magnitude of placebo analgesia. (d) The activation of D2/D3 dopamine receptors in the striatum is related to the placebo response in Parkinson's disease.

dopamine receptors are activated in both ventral (nucleus accumbens) and dorsal striatum when a placebo is administered to patients with Parkinson's disease (De la Fuente-Fernández et al., 2001) (Figure 1d). The release of dopamine corresponds to a change of 200% or more in extracellular dopamine concentration and is comparable with the response to amphetamine in subjects with an intact dopamine system. Dopaminergic activation in the nucleus accumbens in both pain and Parkinson's disease suggests that reward mechanisms could play an important role in many conditions.

Intraoperative single-neuron recording in placebo-treated Parkinsonian patients during the implantation of electrodes for deep brain stimulation (Figure 1e) shows that when placebo is administered, the firing rate of the neurons in the subthalamic nucleus and substantia nigra pars reticulata decreases, whereas the firing rate of thalamic neurons in the ventral anterior and anterior ventral lateral thalamus increases, along with the disappearance of bursting activity in the subthalamic nucleus (Benedetti, 2013, 2014). Although the dopamine findings and the electrophysiological data were obtained in different studies, the changes in firing pattern of the subthalamic and thalamic neurons are likely to be triggered by dopamine release.

Modern brain imaging techniques have been fundamental in the understanding of the placebo response, particularly placebo analgesia, and many brain imaging studies have been carried out to describe the functional neuroanatomy of the placebo analgesic effect (Tracey, 2010; Wager & Atlas, 2015). A meta-analysis of brain imaging data using the activation likelihood estimation method identified two phases: the expectation phase of analgesia and the pain inhibition phase (Amanzio, Benedetti, Porro, Palermo, & Cauda, 2013). During expectation, areas of activation are found in the anterior cingulate, in the precentral and lateral prefrontal cortex, and in the periaqueductal gray. During pain inhibition, deactivations are found in the mid- and posterior cingulate cortex, in the superior temporal and precentral gyri, in the anterior and posterior insula, in the claustrum and putamen, and in the thalamus and caudate body. Overall, many of the regions that are activated during expectation are likely to belong to a descending pain inhibitory system that inhibits different areas involved in pain processing (Figure 1f).

In social anxiety disorder, positron emission tomography has been used to assess regional cerebral blood flow during an anxiogenic public speaking task, before and after 6–8 weeks of treatment with selective serotonin reuptake inhibitors (SSRI) under double-blind conditions. Conjunction analysis reveals a common attenuation of regional cerebral blood flow from

Likewise, in placebo analgesia, there is an activation of D2/D3 receptors and μ -opioid receptors in the nucleus accumbens, whereas in nocebo hyperalgesia there is a deactivation of D2/D3 and μ receptors. (e) Placebo administration in Parkinson patients produces a decrease of firing rate and bursting activity of the subthalamic nucleus neurons. It also produces a decrease of firing rate in the substantia nigra pars reticulata and an increase in the ventral anterior and anterior ventral lateral thalamus. (f) The neuroanatomy of placebo analgesia has been described through brain imaging. Different regions are modulated by both placebos and nocebos, but the most studied and understood regions are the dorsolateral prefrontal cortex (DLPFC), the rostral anterior cingulate cortex (rACC), and the periaqueductal gray (PAG), which represent a descending pain-modulating network. This, in turn, inhibits those regions that are involved in pain processing, such as the mid and posterior cingulate cortex (MCC, PCC), insula, and thalamus. (g) In social anxiety disorder, placebos affect the basolateral and ventrolateral amygdala as well as its projections to DLPFC and rACC. (h) In the immune and endocrine system, the mechanism of the placebo response is classical conditioning, whereby an unconditioned stimulus (US) is paired with a conditioned stimulus (CS). For example, after pairing a CS with either cyclosporine A or sumatriptan, the CS alone can mimic the responses to cyclosporine and sumatriptan. (i) Different polymorphisms have been found to be associated to low (colored squares) or high placebo responsiveness. Source: From Benedetti (2014), Cell Press.

pre- to posttreatment in responders to SSRI and placebo in the left basomedial/basolateral and right ventrolateral amygdala, including amygdala-frontal projections to dorsolateral prefrontal cortex and rostral anterior cingulate cortices (Figure 1g). This pattern correlates with behavioral measures of reduced anxiety and differentiates responders from nonresponders, with no differences between SSRI responders and placebo responders. Therefore, this pattern is capable of differentiating responders from nonresponders to both SSRI and placebos, suggesting that drugs and placebos act on common amygdala targets and amygdala-frontal connections.

High temporal resolution techniques, such as electroencephalography (EEG), have also been used to better understand the effects of expectation and learning. In different studies on laser-evoked potentials (LEPs), placebos have been reported to affect both pain perception and the N2–P2 complex, which represents the largest LEP response with peaks at approximately 200–350 ms after painful stimulation. These data show that placebo effects can be investigated at the electrophysiological level, thus providing in the future an interesting approach to a more detailed temporal analysis.

Immune and endocrine responses can be behaviorally conditioned. When an unconditioned stimulus (US), for example, the effect of a drug, is paired with a conditioned stimulus (CS), for example, a gustatory stimulus, after repeated pairings, the CS alone can mimic the effect of the drug (conditioned response [CR]). Since the CS is a neutral stimulus, it can be conceptualized as a placebo in all respects. Indeed, both immune mediators, like interleukin-2 (IL-2) and interferon- γ (IFN- γ), and hormones, like growth hormone (GH) and cortisol, can be conditioned in humans (Figure 1h). After repeated associations of a CS with cyclosporine A or sumatriptan, which produce IL-2/IFN- γ decrease and GH increase/cortisol decrease, respectively, the CS alone can induce the same immune and hormonal responses.

An association of placebo responsiveness with some genetic variants has been described in some conditions (Figure 1i). Patients with social anxiety disorder have been genotyped with respect to the serotonin transporter-linked polymorphic region (5-HTTLPR) and the G-703T polymorphism in the tryptophan hydroxylase-2 (TPH2) gene promoter. Only patients homozygous for the long allele of the 5-HTTLPR or the G variant of the TPH2 G-703T polymorphism show robust placebo responses and reduced activity in the amygdala, whereas carriers of short or T alleles do not show these effects. In addition, in patients with major depressive disorder, polymorphisms in genes encoding the catabolic enzyme monoamine oxidase A are associated to the magnitude of the placebo response. Patients with monoamine oxidase A G/T polymorphisms (rs6323) coding for the highest activity form of the enzyme (G or G/G) show small placebo responses. Functional Val158Met polymorphism of the catabolic enzyme catechol-O-methyltransferase (COMT) has been found to be associated with the placebo response in irritable bowel syndrome. The lowest placebo responses occur in Val/Val homozygotes. As already described above, also the functional missense variant Pro129Thr of the gene coding FAAH has been found to affect the analgesic responses to placebo.

Treatments Without Expectations

Hidden administration of therapies has provided compelling evidence that expectation is a key element in therapeutic outcome (Benedetti, Carlino, & Pollo, 2011; Colloca, Lopiano, Lanotte, & Benedetti, 2004). If the patient is unaware that a treatment is being performed and has no expectations about any clinical improvement, the therapy is less effective or not effective at all. For example, the effectiveness of the benzodiazepine diazepam is reduced or completely abolished when it is administered unbeknownst to the patient.

This holds true for other conditions as well, such as pain and Parkinson's disease. In post-operative pain following the extraction of the third molar, a hidden intravenous injection of 6–8 mg morphine corresponds to an open intravenous injection of saline solution in full view of the patient (placebo). In other words, telling the patient that a painkiller is being injected (with what is actually a saline solution) is as potent as 6–8 mg of morphine. This holds true for a variety of painkillers, such as morphine, buprenorphine, tramadol, ketorolac, metamizole, and remifentanyl.

Bingel et al. (2011) investigated how divergent expectancies altered the analgesic efficacy of the μ -opioid receptor agonist remifentanyl in healthy volunteers under three experimental conditions: with no expectations of analgesia (told saline, gets remifentanyl), with expectation of a positive analgesic effect (told remifentanyl, gets remifentanyl), and with negative expectation of analgesia (told interruption, gets remifentanyl). Interestingly, expectation of remifentanyl (told remifentanyl, gets remifentanyl), namely, positive treatment expectancies, substantially enhanced, in fact doubled, the analgesic effect of the drug compared with no expectation (told saline, gets remifentanyl). Conversely, expectation of interruption (told interruption, gets remifentanyl) overcame the analgesic potential of remifentanyl to the extent that the effects of drug were completely abolished. The subjective effects were substantiated by fMRI data showing that the enhancement of analgesia in the positive expectation condition was associated with activity in the dorsolateral prefrontal cortex and pregenual anterior cingulate cortex, whereas negative expectation of interruption was matched with activity in the hippocampus. The activity in the hippocampus is thought to stem from an increase in pain perception via anxiety mechanisms. As already pointed out, anxiety represents a powerful modulator in nocebo hyperalgesia, most likely via the activation of the endogenous CCK system.

The understanding of whether the effects of expectation and drugs combine in an additive or synergistically interactive manner is of crucial importance. Atlas et al. (2012) used a balanced placebo design to directly investigate the relationship between opioid analgesia and placebo analgesia by manipulating expectations and remifentanyl concentration. The potential interactions between expectation about treatment (placebo) and remifentanyl were first assessed on pain reports (behavioral experiment). In a separate experiment (fMRI experiment), participants were given intravenous remifentanyl during fMRI scanning in an open–hidden design to test whether the physiological effects of remifentanyl would differ as a function of treatment context (open vs. hidden administration). The results clearly challenge the notion that expectancy effects interact synergistically with opioidergic drugs. Indeed, remifentanyl and expectancy both reduced pain, but drug effects on pain reports and fMRI activity did not interact with expectancy. Regions associated with pain processing showed drug-induced modulation during both open and hidden conditions, with no difference between drug effects as a function of expectation. Instead, expectancy modulated activity in the frontal cortex, with a separable time course from drug effects. Therefore, expectancy seemed to operate alongside, but independent of remifentanyl, without mutual interference.

A natural situation in which hidden therapies are delivered is represented by impaired cognition. Cognitively impaired patients do not have expectations about therapeutic benefits, so the psychological (placebo) component of a treatment is absent. The placebo response of an analgesic therapy has been found to be correlated with both cognitive status and functional connectivity among different brain regions; according to the rule “the more impaired the prefrontal connectivity, the smaller the placebo response.” A number of studies support this view (Benedetti, 2013). First, the individual placebo analgesic effect is correlated with white matter integrity indexed by fractional anisotropy, as assessed through diffusion tensor magnetic resonance imaging; stronger placebo analgesic responses are associated with increased mean fractional

anisotropy values within white matter tracts connecting the periaqueductal gray with the rostral anterior cingulate cortex and the dorsolateral prefrontal cortex. Second, inactivation of the frontal cortex with repetitive transcranial magnetic stimulation completely blocks the analgesic placebo response. Third, the opioid antagonist naloxone blocks placebo analgesia, along with a reduction in the activation of the dorsolateral prefrontal cortex, suggesting that a prefrontal opioidergic mechanism is crucial in the placebo analgesic response. Therefore, both magnetic and pharmacological inactivation of the prefrontal lobes have the same effects as those observed in prefrontal degeneration in Alzheimer's disease and reduced integrity of prefrontal white matter.

The Placebo Effect and the Doctor–Patient Relationship

Doctors, nurses, caregivers, and therapists are essential elements of the whole atmosphere around the therapy, and the doctor–patient relationship may include many placebo and placebo-related effects. Indeed, any element of the psychosocial context is embedded in the doctor–patient interaction and seems to rely on the presence of the doctor, who uses communication, words, and medical instructions, as well as administration of pills, injections, and medications. Words and actions of healing elicit patients' trust, hope, and expectations, and these may, in turn, produce changes in the perception of symptoms or in the progress of illness (Benedetti, 2013). It is therefore not surprising that, if a treatment is surreptitiously administered without a nurse or doctor in the room, it is less effective than when it is given overtly according to routine medical practice (Benedetti et al., 2011; Colloca et al., 2004). Indeed, the decreased effectiveness of hidden therapies indicates that awareness about a treatment affects the therapeutic outcome (see previous section).

Physicians have long known the effects of the relationship with their patients and, accordingly, have used appropriate words and attitudes with them. For example, blood pressure, blood sugar, functional status, and overall health status are consistently related to specific aspects of the physician–patient communication. Moreover, a significant reduction in postoperative pain as well as a lower requirement for narcotics is present in patients who had been informed about the course of their pain and encouraged to overcome it compared with patients who did not achieve any informational or emotional support.

Placebo effects can be experimentally separated through a graded dose escalation of component parts according to the following schema: (a) assignment to a group with only evaluation and observation (waiting list), (b) group with therapeutic ritual alone (placebo acupuncture), and (c) group with therapeutic ritual with an augmented doctor–patient relationship (placebo acupuncture with a patient–practitioner relationship augmented by warmth, attention, and confidence). At 3 weeks, the proportion of the patients reporting adequate relief was 28, 44, and 62%, respectively, indicating that the doctor–patient relationship was a crucial element in the therapeutic outcome.

Positive and negative consultations in medical practice have a great impact on patients who suffer from minor illnesses. Doctors who adopt an empathic behavior are more successful than those who opt for a more formal and detached style of communication. There is also evidence that only a warm and empathic communication combined with positive expectations lead to a relevant decrease in anxiety state, thus suggesting the importance of communication style.

The positive effect of a good relationship is of particular relevance in psychotherapy. Indeed, several authors claim that psychotherapy works only through a benign human relationship. For example, it has been found that students with different psychological problems who were encouraged to interact with practiced psychotherapists with more than 20 years of experience in one case and with teachers of English, mathematics, and philosophy with renowned

reputation for warmth and trustworthiness (though without previous experience as therapists) in the other, recovered in both occasions, regardless of the amount of training the real psychotherapists had achieved. It was therefore concluded that the relief that students had experienced could simply have been due to the healing effect of a positive human contact.

Overall, there are a number of studies that have shown that the doctor–patient relationship plays an important role in the outcome of illness and even diagnostics tests, which have nothing or very little to do with therapy, may induce clinical improvement. Besides expectations of improvement, other factors may be at play. For example, it is possible that a better interaction between the doctor and the patient might lead to a better compliance with drug regimens. In this regard, it is worth mentioning the Coronary Drug Project, a randomized double-controlled trial evaluating the efficacy of lipid lowering drug in the long-term treatment of coronary heart disease. The 5-year in 1,103 men treated with clofibrate was 20%. Those patients who were found to take at least 80% of their prescribed medication (good adherers) had significantly lower mortality (15%) than poor adherers (24.6%). Virtually identical findings occurred in those assigned to the placebo group, in which good adherers had 15.1% mortality and poor adherers had 28.3% mortality. This suggests that placebo may be quite powerful, and its effects may be intertwined with other phenomena, such as compliance and adherence.

The recent advances of neuroscience paved the way for an integration of the doctor–patient relationship within a neurobiological domain, as this unique social interaction involves many physiological processes in the patient’s brain, from the very beginning of the illness to the therapeutic act. From a behavioral and biological viewpoint, to become and to be a patient involves at least four different steps, each of which is amenable to a true neuroscientific investigation (Benedetti, 2013) (Figure 2).

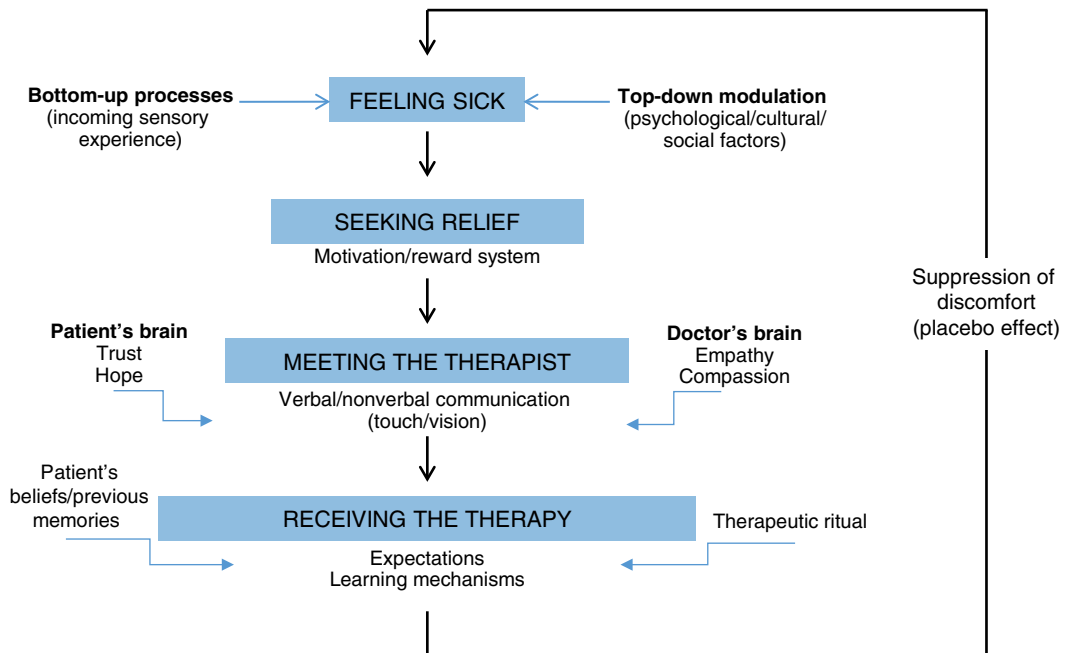


Figure 2 The four different steps of the doctor–patient relationship. The interaction between the healer/therapist and his patient can be considered as a homeostatic system in which the variable to be controlled is represented by the feeling of sickness. The mere ritual of the therapeutic act may generate therapeutic responses through the patient’s expectations and beliefs (placebo responses), which sometimes may be as powerful as those generated by real medical treatments.

The first is the step of “feeling sick,” a crucial starting point that triggers the subsequent behavior. The feeling of sickness is a complex combination of events that may arise from damaged peripheral tissues as well as from their modulation by psychosocial factors. For example, the perception of a symptom such as pain is the product of bottom-up processes taking place in the peripheral and central nervous system and of top-down modulation from cognitive/evaluative and emotional/motivational brain regions (Benedetti, 2013). The physical and psychological unease experienced by the individual triggers motivational and reward mechanisms that are aimed at overcoming discomfort and seeking pleasure. “Seeking relief” is the second step of the doctor–patient relationship and represents a kind of motivated behavior that drives the patient to look for a healer/doctor who himself represents a powerful and active therapeutic agent. Indeed, the whole patient’s reward circuitry is active while he seeks relief.

The third step is when the patient “meets the therapist,” a special and unique social interaction in which the doctor represents the means to suppress discomfort. Here, many intricate mechanisms are at play, such as trust and hope in the patient’s brain and empathy and compassion in the doctor’s brain.

Trust behavior has been found to undergo hormonal modulation by oxytocin. An increase in plasma oxytocin was found in subjects who participated in a trust game whereby cooperative behavior can benefit both parties. The neural circuitry of trustworthy behavior was studied by combining the intranasal administration of oxytocin with functional magnetic resonance imaging, and it was found that oxytocin induced no change in trusting behavior after the subjects learned that their trust had been breached several times, while the control subjects who had not received oxytocin decreased their trust.

Oxytocin has been found to enhance the placebo analgesic response. Male participants were randomly assigned to a double-blind intranasal administration of oxytocin or saline and were then exposed to a set of painful stimuli administered on two different sites of each participant’s forearm. Despite identical thermal stimulation on both sites, pain ratings for the placebo site were significantly lower compared with the control site across both treatment groups, and the placebo analgesic response was significantly higher in the oxytocin group compared with the saline group. The pharmacological enhancement of the placebo analgesic response via intranasal oxytocin administration could be justified by an increased believability of the instructions provided by the experimenter. Indeed, the trust-inducing properties of oxytocin could have more favorably inclined participants to embrace the experimenter’s verbal suggestions, thus reinforcing their belief toward the “healing” properties of the “anesthetic”—actually placebo cream. Moreover, the potential of oxytocin to reduce stress and anxiety might have increased responsiveness to placebo manipulation. Since the brain distribution of oxytocin receptors overlaps with those of arginine vasopressin and the two peptide hormones are both critically involved in regulating social behavior, nonselective vasopressin agonists for both *Avp1a* and *Avp1b* receptors have been found to enhance placebo effects.

The final step of the doctor–patient relationship is when the patient “receives the therapy.” The mere ritual of the therapeutic act may generate therapeutic responses through the patient’s expectations and beliefs (placebo responses), which sometimes may be as powerful as those generated by real medical treatments. As we have seen throughout this review, these placebo responses may be approached from a biological perspective, whereby the biochemical, anatomical, and physiological link between expectation and therapeutic effect has been partially unraveled.

It is worthy to point out that these four steps can be conceived as a homeostatic system that is always at work, regardless of whether the healer administers effective or ineffective therapies. Even if the therapy is totally ineffective, the patient’s expectation of benefit (the placebo response) and the trust toward health professionals may be sufficient to inhibit

discomfort. By contrast, distrust toward health professionals and therapies may elicit unwanted effects as a result of negative expectations, and these may reduce, or even conceal, the efficacy of some treatments (Benedetti, 2013). All this evidence indicates that the therapeutic encounter per se has a profound impact on the patient's brain, so it may be important for doctors, nurses, psychologists, and all other medical and paramedical personnel to enhance their interaction with patients.

Beyond the Healing Context: Physical and Cognitive Performance

All available data indicate athletes' expectations as important elements of physical performance, in spite of the fact that very different experimental conditions have been investigated. These range from short anaerobic sprints to long aerobic endurance cycling, and many different outcome measures have been used, such as time, speed, and weightlifted. These placebo responses acquire a very important meaning if central mechanisms of muscle performance and fatigue are considered, as postulated early in the 1910s through the concept of central command. The notion of central command, or central governor, implies that several physiological parameters like heart rate, arterial blood pressure, pulmonary ventilation, and muscle performance could be altered by manipulating the subject's perception of exercise. Muscle fatigue has also been found to be affected by a central governor. In many studies, athletes are usually asked to perform at their limit, and placebos apparently act by pushing this limit forward. It is interesting to note that the concept of a central governor has been applied to health as well, thus extending from fatigue and physical performance to the healing environment. Physical performance has also been found to be worsened by nocebos.

A number of studies suggest that placebos and expectations can also enhance cognitive performance and other cognition-related tasks, such as reaction times. Unfortunately, nothing is known about the underlying mechanisms, but it is interesting to note that a placebo, which subjects believed to be a memory-boosting drug, has been found to increase short-term memory through the activation of the endogenous opioid systems.

See Also

Health belief model; Treatment seeking/avoidance; Illness representations/perceptions; Placebo/nocebo effects; Patient-provider communication; Embodied health; Social influence; Personality and health outcomes; Complementary and alternative medicine; Psychoneuroimmunology

Author Biographies

Diletta Barbiani, MSc, obtained a Master in Cognitive Psychology at the University of Bologna, Italy, and is now a PhD student at the Department of Neuroscience of the University of Turin Medical School, Italy. The research she is focusing on deals with complex psychological mechanisms, such as expectations, beliefs, and conditioning, particularly those taking part in the placebo response across both clinical and experimental settings. Her interest is mainly related on the integration between neurobiological and behavioral mechanisms underlying the placebo effect and their clinical and social applications.

Fabrizio Benedetti, MD, is professor of neurophysiology and human physiology at the University of Turin Medical School, Turin, Italy, and director of Medicine and Physiology of Hypoxia at Plateau Rosà, Switzerland. He has been nominated member of the *Academy of Europe* and of the *European Dana Alliance for the Brain*. He is author of the book *Placebo Effects* (Oxford University Press, 2nd Edition, 2014), which received the Medical Book Award of the British Medical Association, and *The Patient's Brain* (Oxford University Press 2010). In 2012 he received the Seymour Solomon Award of the American Headache Society, and in 2015 the William S. Kroger Award of the American Society of Clinical Hypnosis. In 2015 he was nominated member of the Council of Scientists of the Human Frontiers Science Program Organization.

References

- Amanzio, M., Benedetti, F., Porro, C. A., Palermo, S., & Cauda, S. (2013). Activation likelihood estimation meta-analysis of brain correlates of placebo analgesia in human experimental pain. *Human Brain Mapping*, 34(3), 738–752. doi:10.1002/hbm.21471
- Atlas, L., Whittington, R. A., Lindquist, M. A., Wielgosz, J., Sonty, N., & Wager, T. D. (2012). Dissociable influences of opiates and expectations on pain. *Journal of Neuroscience*, 32(23), 8053–8064. doi:10.1523/JNEUROSCI.0383-12.2012
- Benedetti, F. (2013). Placebo and the new physiology of the doctor-patient relationship. *Physiological Reviews*, 93(3), 1207–1246.
- Benedetti, F. (2014). Placebo effects: From the neurobiological paradigm to translational implications. *Neuron*, 84(3), 623–637. doi:10.1016/j.neuron.2014.10.023
- Benedetti, F., Carlino, E., & Pollo, A. (2011). Hidden administration of drugs. *Clinical Pharmacology & Therapeutics*, 90(5), 651–661. doi:10.1038/clpt.2011.206
- Benedetti, F., & Dogue, S. (2015). Different placebos, different mechanisms, different outcomes: Lessons for clinical trials. *PLoS One*, 10(11), e0140967.
- Bingel, U., Wanigasekera, V., Wiech, K., Ni Mhuiricheartaigh, R., Lee, M. C., Ploner, M., & Tracey, I. (2011). The effect of treatment expectation on drug efficacy: Imaging the analgesic benefit of the opioid remifentanyl. *Science Translational Medicine*, 3(70), 70ra14. doi:10.1126/scitranslmed.3001244
- Colloca, L., Lopiano, L., Lanotte, M., & Benedetti, F. (2004). Overt versus covert treatment for pain, anxiety, and Parkinson's disease. *Lancet Neurology*, 3(11), 679–684. doi:10.1016/S1474-4422(04)00908-1
- De la Fuente-Fernández, R., Ruth, T. J., Sossi, V., Schulzer, M., Calne, D. B., & Stoessl, A. J. (2001). Expectation and dopamine release: Mechanism of the placebo effect in Parkinson's disease. *Science*, 293(5532), 1164–1166. doi:10.1126/science.1060937
- Finniss, D. G., Kaptchuk, T. J., Miller, F., & Benedetti, F. (2010). Biological, clinical, and ethical advances of placebo effects. *Lancet*, 375(9715), 686–695. doi:10.1016/S0140-6736(09)61706-2
- Price, D. D., Finniss, D. G., & Benedetti, F. (2008). A comprehensive review of the placebo effect: Recent advances and current thought. *Annual Review of Psychology*, 59, 565–590. doi:10.1146/annurev.psych.59.113006.095941
- Tracey, I. (2010). Getting the pain you expect: Mechanisms of placebo, nocebo and reappraisal effects in humans. *Nature Medicine*, 16(11), 1277–1283. doi:10.1038/nm.2229
- Wager, T. D., & Atlas, L. Y. (2015). The neuroscience of placebo effects: Connecting context, learning and health. *Nature Reviews Neuroscience*, 16(7), 403–418. doi:10.1038/nrn3976

Suggested Reading

- Benedetti, F. (2010). *The patient's brain: The neuroscience behind the doctor-patient relationship*. New York, NY: Oxford University Press.
- Benedetti, F. (2014). *Placebo effects* (2nd ed.). New York, NY: Oxford University Press.

Prescriptive Authority for Psychologists

Wendy P. Linda and Robert E. McGrath

School of Psychology, Fairleigh Dickinson University, Teaneck, NJ, USA

Since its inception three decades ago, the movement to obtain prescriptive authority for psychologists, often abbreviated as RxP, has been perhaps the most controversial issue in professional psychology (McGrath, 2010). This has resulted in several preliminary evaluations of prescribing psychologists, particularly in the military, and survey research evaluating support for RxP among psychologists and medical professionals. This description of the RxP movement begins with a history of the movement, followed by a discussion of the controversy surrounding it.

A History of the Prescriptive Authority Movement

Though two task forces of the American Psychological Association (APA) in 1981 and 1985 suggested expanding the scope of practice of healthcare psychology to include biological interventions, the birth of the RxP movement is typically attributed to a speech made in 1984 by Senator Daniel Inouye of Hawaii to the Hawaiian Psychological Association, publicly recommending that psychologists pursue prescriptive authority to increase the availability of quality prescribing providers to serve a much broader patient base (Fox, 2003). As a result of Senator Inouye's interest in RxP, the Department of Defense (DoD) established the Psychopharmacology Demonstration Project (Newman, Phelps, Sammons, Dunivin, & Cullen, 2000). This program trained 10 Army, Navy, and Air Force psychologists to prescribe between the years 1991 and 1997. The evolution of the curriculum of this program is charted by Sammons and Brown (1997). Four governmental and nongovernmental evaluations of this project were conducted with generally positive findings regarding the quality of care provided. In particular, the evaluators failed to identify any adverse patient outcomes for those seeking treatment with a prescribing psychologist (McGrath, 2010).

Though this program was terminated, the three branches of the military with healthcare services all currently have guidelines in place for privileging psychologists to prescribe. The Public Health Service and Indian Health Service have also allowed psychologists to prescribe, though those agencies have so far limited the privilege to psychologists who have obtained a license to prescribe in a state that has approved RxP.

APA has actively supported the effort to extend RxP to the states. In 1990, APA convened a task force on psychopharmacology, and RxP was adopted as official association policy in 1995 (Fox, 2003; Smyer et al., 1993). The task force recommended three levels of preparation in pharmacotherapy for psychologists. Level 1 was appropriate for all healthcare psychologists, Level 2 for those interested in collaborating with medical providers in medication decision making, and Level 3 for those who wanted to pursue independent prescriptive authority. Though training curricula were ultimately developed for all three levels, only the Level 3 curriculum has been considered important enough to merit updating (APA, 2009). In 1997, the APA Practice Organization also began the development of a competency examination called the Psychopharmacology Examination for Psychologists (PEP) that could be used as a separate licensing examination should states authorize psychologists to prescribe. Efforts to achieve this authorization soon followed.

In 1993 the licensing law for psychologists in Indiana was revised to allow prescriptive authority for psychologists participating in federally sponsored training or treatment programs, a bill that would have allowed graduates of the Psychopharmacology Demonstration Project to prescribe in Indiana. Though this was the first instance where a state approved psychologists to prescribe, to date not a single psychologist has used approval by the military to prescribe as a basis for prescribing in Indiana.

In 1999 the territory of Guam passed legislation allowing psychologists to become prescribers as long as they had a collaborative agreement with a physician in the same specialty area. However, because of issues associated with implementation of regulations, it seems that no psychologist in Guam has yet obtained prescriptive authority.

Four states have now approved prescriptive authority for psychologists using the curriculum defined by APA as the basis for their law. All require passing the PEP, licensure as a psychologist, and additional training in psychopharmacology. However, the laws differ in a number of key respects.

New Mexico and Louisiana passed legislation authorizing psychologists to prescribe in 2002 and 2004, respectively. In Louisiana, the legal title for a psychologist prescriber is *medical psychologist*. The Louisiana law requires completion of a master's degree in psychopharmacology and passage of the PEP. The medical psychologist must obtain concurrence from the patient's physician on all prescriptive decisions and is unable to prescribe to patients without a primary care physician. However, the Louisiana legislature subsequently amended this requirement. Now after 3 years of practice without any complaints, a medical psychologist can apply for a certificate of advanced practice that will allow prescribing without physician concurrence.

In the state of New Mexico, after completing coursework and passing the PEP, a psychologist must first complete an additional 480 hr of practicum experience, including 80 hr of physical examination training, and then work as a conditional prescriber under the supervision of a physician for 2 years. Only after this supervisory period may the psychologist in New Mexico prescribe independently, and continued collaboration (not necessarily concurrence) with a primary care physician is required.

In 2014, Illinois approved prescriptive authority for psychologists. This is a particularly restrictive bill, requiring the following in addition to the completion of coursework and the PEP: undergraduate biomedical courses in medical terminology, chemistry or biochemistry,

human physiology, human anatomy, anatomy and physiology, microbiology, and general or cell and molecular biology; a full-time practicum of 14 months comprising at least 36 credit hours, with specific rotations enumerated; and maintenance of a written collaborative agreement with a physician. Also, psychologists are not allowed to prescribe to patients younger than 17 or older than 65, pregnant women, or patients with serious medical conditions. However, the Illinois bill is the only one that allows psychologists to receive psychopharmacology training as part of their doctoral program rather than after licensure.

In 2016, Iowa approved prescriptive authority. In addition to the standard training, the psychologist must complete a supervised practicum. The psychologist then prescribes under a conditional prescription certificate, which requires the supervision of a licensed physician. After 2 years, the psychologist may apply for a prescription certificate, which requires only a collaborative practice agreement with a physician.

In most years, RxP bills are put before the legislature in 5–7 states, with the large majority defeated each year. It is unclear at this point whether recent victories in Illinois and Iowa will represent anomalies as was true for Louisiana and New Mexico or represent a turning point in efforts to achieve prescriptive authority.

The Controversy

Whether the idea becomes more acceptable to legislators, it is an idea that has not been acceptable to medical organizations or to many psychologists. This section will outline the arguments that have been raised for and against RxP.

Training

The depth and quality of training for prescribing psychologists is a central point of contention between opponents and supporters. One issue is whether the training is sufficient to allow for safe practice. In particular, few psychologists complete the undergraduate-level foundational science coursework required of nurse practitioners and physician assistants (Ball, Kratochwill, Johnston, & Fruehling, 2009). Robiner, Tumlin, and Tompkins (2013) argued that such foundational science knowledge is critical to understanding how the body responds to medication, comprehension of the complex interaction of bodily systems, and the impact and interaction of various medications. also argued that a single administration of the PEP is inferior to the United States Medical Licensing Examination, a stepped procedure that evaluates competency at various points in the training sequence.

In response to criticisms focusing on differences between RxP training and training for physicians, it should be noted that basic science coursework is mandated as part of the APA curriculum for prescriptive authority and that the scope of practice being pursued is much more limited than is true for the medical providers referenced by. Proponents of the RxP movement have also pointed to the success of nonphysician prescribers (nurse practitioners, optometrists, dentists), who consistently receive less rigorous training than physicians (Bell, Digman, & McKenna, 1995). When compared with physicians, nurse practitioners who prescribe have demonstrated comparable outcomes and receive higher ratings for satisfaction, signaling that a medical school education is not necessarily a prerequisite for safe and effective prescribing (Lenz, Munding, Kane, Hopkins, & Lin, 2004).

Muse and McGrath (2010) sought to compare training programs for prescribing psychologists with training relevant to prescribing psychotropic medications for physicians and

nurse practitioners up to the point at which they were legally authorized to prescribe such medications. They included topic areas they considered relevant to optimal practice that are not emphasized in traditional medical education, such as research training. While they found physicians received greater instruction in biochemistry and neuroscience, the psychologists' curriculum incorporated much more information specifically about psychopharmacology. For example, psychologists trained to prescribe were found to have received over four times as much coursework in pharmacology as physicians and six times as much as psychiatric nurse practitioners. Additionally, psychologists received much more training in the diagnosis of mental health disorders and in alternative treatments to medication than either physicians or psychiatric nurse practitioners. However, Heiby (2010) noted some flaws in the Muse and McGrath (2010) study. Specifically, though they are not required before a physician can prescribe, the study failed to consider undergraduate prerequisites, apprenticeships, and post-degree training such as residencies that almost all physicians complete before they do prescribe.

Supporters of RxP have criticized the training model from a very different perspective, arguing that the training should be incorporated into the doctoral program. Resnick, Ax, Fagan, and Nussbaum (2012), for example, proposed that a doctoral-level training model might prove to be economically advantageous for psychologists and would encourage the selection of students with a much stronger undergraduate background in natural sciences.

Safety

Concerns relate to a prescribing psychologist's ability to account for drug–drug interactions, or the impact on comorbid health problems when making prescribing decisions (Bell et al., 1995). In response, Fox et al. (2009) reported that over 70 psychologists certified to prescribe in New Mexico and Louisiana had collectively written over 250,000 prescriptions “without incident” (p. 264).

As noted previously, evaluations of the DoD Psychopharmacology Demonstration Project were favorable in terms of patient outcomes. In fact, graduates of the program served in the military as prescribers for a number of years with no untoward outcomes. Though the evaluations of the DoD program provide the most extensive evaluation to date of prescribing psychologists, it has been suggested they have limited generalizability to psychologists in the private sector (Robiner et al., 2002), because the training model was more extensive than that recommended by APA (APA, 2002 [see McGrath, 2010, for a comparison]).

Only two studies have addressed the safety and effectiveness of RxP outside the context of the demonstration project. Shearer, Harmon, Seavey, and Tiu (2012) conducted a survey of 65 medical staff, most of whom were physicians or residents, working with a prescribing psychologist in a primary care clinic. The authors found that medical staff found the presence of the prescribing psychologist helpful, safe, convenient, and overall resulting in better patient care outcomes. Over 90% of the sample reported that they had confidence in the safety of referring their patients to a prescribing psychologist for psychotropic medication management, that the prescribing psychologist would prescribe appropriate medications and dosages, and that the prescribing psychologist could determine when patients needed to be referred for additional medical evaluation.

Unfortunately, the study was flawed in that all ratings were of a single prescriber who was working as part of a team in a primary care clinic in a major US Army medical facility. The study has recently been replicated using a larger sample of both medical providers and prescribing psychologists across New Mexico and Louisiana (Linda & McGrath, 2016).

The researchers found that 30 psychologists and 24 medical colleagues overwhelmingly considered prescribing psychologists competent in the realms of safety, knowledge, and training. The most common benefit reported by medical colleagues of working with prescribing psychologists involved sharing expertise. However, more research is needed before the hypothesis of adequate safety can be considered to have been corroborated.

Another key point made by proponents of the RxP movement, arguing for the safety of the prescribing psychologist, is that a psychologist's strong foundational training in psychosocial intervention may lead prescribing psychologists to prescribe less frequently and may move more patients safely off medication, compared with other prescribers lacking such training (McGrath, 2010). However, Linda and McGrath (2016) did not find evidence supporting this hypothesis.

Access to Care

A key argument for proponents of the RxP movement is that it could increase the number of mental health professionals available to treat underserved populations (Gutierrez & Silk, 1998). At present, a general medical practice is the most frequent treatment setting for individuals with psychological disorders, and in the majority of doctor visits where psychotropic medication is prescribed, the prescriber is a nonpsychiatric prescriber (Mark, Levit, & Buck, 2009). Despite a growing need, the overall number of psychiatrists has actually fallen in recent history (Rao, 2003), especially in remote areas, and Thomas, Ellis, Konrad, Holzer, and Morrissey (2009) conservatively estimated that 96% of US counties do not have enough prescribers with specialty training in mental disorders to meet the need. As a result, psychiatric visits are decreasing in duration, and such visits often last less than 10 min (Olfson, Marcus, & Pincus, 1999). Psychiatry also has the lowest rate of participation in insurance of all medical specialties (Bishop, Press, Keyhani, & Pincus, 2013). Kaplin and Dacunto (2014) made the point that if psychologists continue to see patients weekly, the amount of time they spend with patients would be substantially greater than other prescribers who typically meet with patients monthly or less. Further, prescribing psychology may represent a more efficient model of treatment as patients can receive all of their mental healthcare from one provider.

Moore and McGrath (2007) also argued that, particularly for the military, an increase in prescribing psychologists could reduce the demand placed on nonpsychiatric physicians, freeing medical staff to allocate attention to other urgent medical needs. In a similar vein, Ax, Fagan, and Resnick (2009) proposed that the RxP model could prove beneficial to the incarcerated population. These authors reported that mental health services in correctional settings are already provided by a range of professions, including correctional counselors with minimal and often informal training in mental health issues. Fagan et al. (2004) made the case for the need for psychologist prescribers in Department of Veterans Affairs hospitals, as the number of veteran patients has increased in recent years. In those facilities, prescribing psychologists might improve access to healthcare in a setting where they could easily team with a primary care physician.

In response to concerns that prescribing psychologists are likely to be concentrated in the same metropolitan areas as other prescribers, Vento (2014) found that over 90% of prescribing psychologists surveyed in New Mexico accepted Medicaid payments and 62.6% of patients served were reported by the prescribers to be living in rural areas. However, it is unclear whether the New Mexico experience would generalize to other states. did not see the decrease in the number of psychiatrists as an argument for psychologist prescribers, but rather as a call for enhanced training for primary care physicians, nurse practitioners, and physicians' assistants,

which could possibly create a far larger pool of competent prescribers than would result from allowing psychologists to prescribe. The authors also recommended further training for psychologists to work collaboratively alongside physicians. The authors did not address the issue of how many medical providers could or would pursue additional training without any financial return.

Building on Current Practices

Proponents of the RxP movement are often quick to point out that many treating psychologists are already well aware of their patients' medical regimens, and the psychologist is often included as part of a team making medical decisions or is kept apprised of medication changes or medical issues (Bell et al., 1995). VandenBos and Williams (2000) conducted a survey assessing the professional activities of 596 practicing psychologists. The majority indicated that they were involved with a patient's medication regimen in a variety of ways. It is notable that 99% of the respondents reported that they have at some time collaborated with a physician who prescribed psychotropic and other types of medication. Over 90% indicated that they had made recommendations for medication and that they had consulted with a physician about the need to alter a patient's medication. Finally, 87% of these practitioners reported having been directly involved in the decision-making process about whether to prescribe medication to a patient. Based on these findings, prescriptive authority can be conceptualized as a sequential step in a psychologist's care of a patient, rather than a break with current practice as opponents of RxP have suggested (e.g., Robiner et al., 2002).

Financial and Professional Incentives

McGrath (2010) proposed that prescriptive authority could increase financial and professional opportunities in the field of psychology, similar to the experience of nurses after obtaining prescriptive authority. Linda and McGrath (2016) found that one of the most common changes reported by psychologists since prescribing was increased salary. One unexpected outcome has been the inclusion of psychologists among the authors of some major texts in the field of psychopharmacology (Julien, Advokat, & Comaty, 2011; Stahl & Moore, 2013).

Loss of Identity

Internal concerns among psychologists focus on how the RxP movement might irrevocably change or negatively impact the identity of the profession of psychology as a whole (DeNelsky, 1996; Hayes & Heiby, 1996). Given economic forces to minimize contact with more expensive providers and pressure from insurers for a rapid cure, psychologists worry that their profession could lose the historic focus on psychosocial intervention, and as Shearer et al. (2012) noted, psychologists could become "junior psychiatrists." There is also concern that psychologists might surrender psychosocial intervention to master's level providers such as social workers or other counselors (McGrath, 2010). Further, DeNelsky (1996) predicted that because there is a greater risk of legal action for medication-related decisions than for psychological interventions, more time may need to be devoted to learning about medication in the continuing education of prescribing psychologists at the expense of psychosocial intervention strategies.

DeNelsky (1996) also argued that medications cannot be conceptualized as simply another "tool" in the psychologist's toolbox. The concern is that, as occurred with psychiatrists, this

one tool would come to dominate practice, in part because of aggressive marketing by the pharmaceutical industry but also because of increased demands on the psychologist's time. LeVine, Wiggins, and Masse (2011) found that 13 of 17 prescribing psychologists working in private practice in the states of New Mexico and Louisiana reported that sales representatives from pharmaceutical companies had not particularly targeted them, but a few did receive drug samples and small gifts such as pens. It is reasonable to hypothesize, however, that if the RxP movement expands, psychologists will experience greater pressure from the pharmaceutical industry. This also does not address the influence of more subtle marketing such as manipulation of publications.

In line with this concern, McGrath (2004) argued that it would be naive to believe that prescribing psychologists will not experience similar pressure to emphasize medication over other interventions, particularly from insurers, and in anticipation of such pressure, psychologists will need to develop strategies for resistance. He suggested one possible response to those external forces would be for psychology to develop an ethical guideline forbidding psychologists from prescribing to individuals for whom they are not also providing psychosocial interventions. However, this may stand in the way of the RxP's goal of overall increasing access to psychopharmacological care.

Ethical Implications

Buelow and Chafetz (1996) argued that the RxP movement comes with a range of ethical considerations for a "subfield" that touches on territory not previously considered or entered into by psychologists. The authors proposed some ethical standards appropriate to the situation, including the importance of clear therapeutic contracts, transparent and delineated treatment objectives, and agreement by the patient about which modalities of treatment will be used. Psychologists will need to be open about the risks and the benefits of different forms of treatment. Parameters of informed consent will need to be reconsidered as well, and professional relationships will need to readjust along with the shifting role of the psychologist within the healthcare system. Mantell, Ortiz, and Planthara (2004) raised the lack of empirically based research approaches for the appropriate combination of medical and psychological interventions as an ethical issue. However, an increase in the number of psychologist prescribers may foster the development and validation of such treatment protocols.

Though ethical standards specific to prescribing have not yet emerged in psychology, APA (2011) has adopted practice guidelines relevant to psychologists' involvement in pharmacotherapy. Unlike ethical standards, which for psychologists are mandatory and enforceable, practice guidelines are intended to represent optimal practice related to a domain of practice, are aspirational in nature, and represent optimal practice recommendations.

Conclusions

Opinion related to psychologists prescribing is also still evolving. Research demonstrates that the majority of psychologists favor prescriptive authority for psychologists (RxP; Fagan et al., 2004; Rae, Jensen-Doss, Bowden, Mendoza, & Banda, 2008; Walters 2001), though there is still opposition within the field (Robiner et al., 2013). This opposition has often centered on how prescriptive authority will impact the practice of psychology, leading to questions regarding the abandonment of psychosocial intervention (DeNelsky, 1996; Hayes & Heiby, 1996). However, with additional legislation for RxP, more research on the practices of prescribing

psychologists, and new guidelines for their practice, opinions may shift. Organized psychiatry continues to oppose RxP, and both the American Psychiatric Association and the American Medical Association have lobbied against legislation (Robiner et al., 2013). Again, these views of RxP may evolve over time with more prescribing psychologists working in integrated healthcare settings and more research related to patient outcomes.

In one important way, the debate about whether psychologists should prescribe is irrelevant, in that psychologists have been prescribing for over 20 years, with no evidence of incompetence, and therefore unlikely to evaporate. Whether it will reshape the nature of the profession and whether tensions will emerge over time between prescribing and non-prescribing psychologists are questions to be followed closely.

Author Biographies

Wendy P. Linda, PhD, received her doctorate in clinical psychology from Fairleigh Dickinson University, Teaneck, NJ. Currently, she is finishing a postdoctoral fellowship focused on inpatient psychology at Long Island Jewish Medical Center–Zucker Hillside Hospital of Northwell Health, Glen Oaks, New York. Her clinical and research interests include the treatment of chronic and severe mental illness, trauma and substance use, and professional issues in health-care psychology.

Robert E. McGrath, PhD, received his doctorate in clinical psychology from Auburn University, Auburn, AL. He is a professor of psychology at Fairleigh Dickinson University in Teaneck, NJ. He is also a senior scientist for the VIA Institute on Character in Cincinnati, OH. He maintains an active research program in professional issues in health service psychology, character, and program evaluation.

References

- American Psychological Association. (2002). New Mexico becomes first state to gain Rx privileges. *APA Monitor*, 33, 24. Retrieved from <http://www.apa.org/monitor/apr02/newmexico.aspx>.
- American Psychological Association. (2009). *Recommended postdoctoral education and training program in psychopharmacology for prescriptive authority*. Washington, DC: Author.
- American Psychological Association. (2011). Practice guidelines regarding psychologists' involvement in pharmacological issues. *American Psychologist*, 66, 835–849.
- Ax, R. K., Fagan, T. J., & Resnick, R. J. (2009). Predoctoral prescriptive authority training: The rationale and a combined model. *Psychological Services*, 6, 85–95.
- Ball, C. R., Kratochwill, T. R., Johnston, H. F., & Fruehling, J. J. (2009). Limited prescription privileges for psychologists: Review and implications for the practice of psychology in the schools. *Psychology in Schools*, 46, 836–845.
- Bell, P. F., Digman, R. H., Jr., & McKenna, J. P. (1995). Should psychologists obtain prescribing privileges? A survey of family physicians. *Professional Psychology: Research and Practice*, 26, 371–376.
- Bishop, T. F., Press, M. J., Keyhani, S., & Pincus, H. A. (2013). Acceptance of insurance by psychiatrists and the implications for access to mental health care. *JAMA Psychiatry*, 71, 176–181.
- Buelow, G. D., & Chafetz, M. D. (1996). Proposed ethical practice guidelines for clinical pharmacopsychology: Sharpening a new focus in psychology. *Professional Psychology: Research and Practice*, 27, 53–58.
- DeNelsky, G. Y. (1996). The case against prescription privileges for psychologists. *American Psychologist*, 51, 207–212.

- Fagan, T. J., Ax, R. K., Resnick, R. J., Liss, M., Johnson, R. T., & Forbes, M. R. (2004). Attitudes among interns and directors of training: Who wants to prescribe, who doesn't, and why. *Professional Psychology: Research and Practice*, 35, 345–356.
- Fox, R. E. (2003). Early efforts by psychologists to obtain prescriptive authority. In M. T. Sammons, R. F. Levant, & R. U. Paige (Eds.), *Prescriptive authority for psychologists: A history and guide* (pp. 33–47). Washington, DC: American Psychological Association.
- Fox, R. E., DeLeon, P. H., Newman, R., Sammons, M. T., Dunivin, D. L., & Baker, D. C. (2009). Prescriptive authority and psychology: A status report. *American Psychologist*, 64, 257–268.
- Gutierrez, P. M., & Silk, K. R. (1998). Prescription privileges for psychologists: A review of the psychological literature. *Professional Psychology: Research and Practice*, 29, 213–222.
- Hayes, S. C., & Heiby, E. (1996). Psychology's drug problem: Do we need a fix or should we just say no? *American Psychologist*, 51, 198.
- Heiby, E. M. (2010). Concerns about substandard training for prescription privileges for psychologists. *Journal of Clinical Psychology*, 66, 104–111.
- Julien, R. M., Advokat, C. D., & Comaty, J. E. (2011). *A primer of drug action: A comprehensive guide to the actions, uses and side effect of psychoactive drugs*. New York, NY: Worth Publishers.
- Kaplin, D., & Dacunto, K. (2014). Exploring the benefits and drawbacks of granting prescriptive authority to psychologists. *Archives of Medical Psychology*, 5, 12–30.
- Lenz, E. R., Mundinger, M. O., Kane, R. L., Hopkins, S. C., & Lin, S. X. (2004). Primary care outcomes in patients treated by nurse practitioners or physicians: Two-year follow-up. *Medical Care Research and Review*, 61, 332–251.
- LeVine, E., Wiggins, J., & Masse, E. (2011). Prescribing psychologists in private practice: The dream and the reality of the experiences of prescribing psychologists. *AMP: Archives of Medical Psychology*, 2, 1–14.
- Linda, W. P., & McGrath, R. (2016). The current status of prescribing psychologists: Practice patterns and medical professional evaluations. *Professional Psychology: Research and Practice*, 48, 38–45.
- Mantell, O., Ortiz, S. O., & Planthara, P. M. (2004). Prescribing? A commentary on the effect of prescription authority on psychological practice. *Professional Psychology: Research and Practice*, 35, 164–169.
- Mark, T. L., Levit, K. R., & Buck, J. A. (2009). Psychotropic drug prescriptions by medical specialty. *Psychiatric Services*, 60, 1167.
- McGrath, R. E. (2004). Saving our psychosocial souls. *American Psychologist*, 59, 644–646.
- McGrath, R. E. (2010). Prescriptive authority for psychologists. *Annual Review of Clinical Psychology*, 6, 21–47.
- Moore, B. A., & McGrath, R. E. (2007). How prescriptive authority for psychologists would help service members in Iraq. *Professional Psychology: Research and Practice*, 38, 191–195.
- Muse, M., & McGrath, R. E. (2010). Training comparisons among three professions prescribing psychoactive medications: Psychiatric nurse practitioners, physicians, and pharmacologically trained psychologists. *Journal of Clinical Psychology*, 66, 96–103.
- Newman, R., Phelps, R., Sammons, M. T., Dunivin, D. L., & Cullen, E. A. (2000). Evaluation of the psychopharmacology demonstration project: A retrospective analysis. *Professional Psychology: Research and Practice*, 31, 598–603.
- Olfson, M., Marcus, S. C., & Pincus, H. A. (1999). Trends in office-based psychiatric practice. *The American Journal of Psychiatry*, 156(3), 451–457.
- Rae, W. A., Jensen-Doss, A., Bowden, R., Mendoza, M., & Banda, T. (2008). Prescription privileges for psychologists: Opinions of pediatric psychologists and pediatricians. *Journal of Pediatric Psychology*, 33, 176–184.
- Rao, N. R. (2003). Recent trends in psychiatry residency workforce with special reference to international medical graduates. *Academic Psychiatry*, 27, 269–276.
- Resnick, R. J., Ax, R. K., Fagan, T. J., & Nussbaum, D. (2012). Predoctoral prescriptive authority curricula: A training option. *Journal of Clinical Psychology*, 68, 246–262.

- Robiner, W. N., Bearman, D. L., Berman, M., Grove, W. M., Colón, E., Mareck, S., & Armstrong, J. (2002). Prescriptive authority for psychologists: A looming health hazard? *Clinical Psychology: Science and Practice*, 9, 231–248.
- Robiner, W. N., Tumlin, T. R., & Tompkins, T. L. (2013). Psychologists and medications in the era of interprofessional care: Collaboration is less problematic and costly than prescribing. *Clinical Psychology: Science and Practice*, 20, 489–507.
- Sammons, M. T., & Brown, A. B. (1997). The department of defense psychopharmacology demonstration project: An evolving program for postdoctoral education in psychology. *Professional Psychology: Research and Practice*, 28, 107–112.
- Shearer, D. S., Harmon, S. C., Seavey, B. M., & Tiu, A. Y. (2012). The primary care prescribing psychologist model: Medical provider rating of the safety and utility of prescribing psychology in a primary care setting. *Journal of Clinical Psychology in Medical Settings*, 14, 420–429.
- Smyer, M. A., Balster, R. L., Egli, D., Johnson, D. L., Kilbey, M. M., Leith, N. J., & Puente, A. E. (1993). Summary of the report of the ad hoc task force on psychopharmacology of the American Psychological Association. *Professional Psychology: Research and Practice*, 24, 394–403.
- Stahl, S. M., & Moore, B. A. (2013). *Anxiety disorders: A guide for integrating psychopharmacology and psychotherapy*. London, England: Routledge.
- Thomas, K. C., Ellis, A. R., Konrad, T. R., Holzer, C. E., & Morrissey, J. P. (2009). County-level estimates of mental health professional shortage in the United States. *Psychiatric Services*, 60, 1323–1328.
- VandenBos, G. R., & Williams, S. (2000). Is psychologists' involvement in the prescribing of psychotropic medication really a new activity? *Professional Psychology: Research and Practice*, 31, 615–618.
- Vento, C. (2014). Report from the trenches: Survey of New Mexico prescribing psychologists' outpatient practice characteristics and impact on mental health care disparities in calendar 2013. *Archives of Medical Psychology*, 5, 30–34.
- Walters, G. D. (2001). A meta-analysis of opinion data on the prescription privilege debate. *Canadian Psychology/Psychologie Canadienne*, 42, 119–125.

Suggested Reading

- Ball, C. R., Kratochwill, T. R., Johnston, H. F., & Fruehling, J. J. (2009). Limited prescription privileges for psychologists: Review and implications for the practice of psychology in the schools. *Psychology in the Schools*, 46, 836–845.
- Greenberg, R. P. (2010). Prescriptive authority in the face of research revelations. *American Psychologist*, 65, 136–137.
- Julien, R. M. (2011). Psychopharmacology training in clinical psychology: A renewed call for action. *Journal of Clinical Psychology*, 67, 446–449.
- McGrath, R. E., & Muse, M. (2010). Room for a new standard? Response to comments by Heiby. *Journal of Clinical Psychology*, 66, 112–115.
- McGrath, R. E., & Sammons, M. (2011). Prescribing and primary care psychology: Complementary paths for professional psychology. *Professional Psychology: Research and Practice*, 42, 113–120.
- Rinaldi, A. P. (2013). Prescriptive authority and counseling psychology: Implications for practitioners. *The Counseling Psychologist*, 41, 1213–1228.

Health-Related Quality of Life

Amy E. Ustjanauskas^{1,2} and Vanessa L. Malcarne^{1,2,3}

¹*San Diego State University, UC San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA*

²*UC San Diego Moores Cancer Center, San Diego, CA, USA*

³*Department of Psychology, San Diego State University, San Diego, CA, USA*

Overview

Quality of life (QoL) is a broad construct that encompasses multiple dimensions of a person's current status. The construct has been defined in numerous ways. Most definitions of QoL focus on people's perceptions of physical, psychological, and social well-being. However, definitions of QoL are wide ranging. Some definitions additionally include an individual's perceptions of all aspects of life including occupation, housing, neighborhood, culture, values, and spirituality. Other definitions assert that QoL is composed of both objective and subjective components; that is, in addition to self-perceptions of one's current status, QoL also includes objective components that can be measured using biological, social, or behavioral indicators of one's status.

While several definitions of QoL exist, a widely accepted definition of QoL has been delineated by the World Health Organization (WHO). The WHO defines QoL as "an individual's perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns" (WHOQOL Group, 1997, p. 1). The WHO's definition of QoL encompasses people's "physical health, psychological state, personal beliefs, social relationships, and their relationship to salient features of their environment" (WHOQOL Group, 1997, p. 1), as well as spirituality and religion.

Health-related quality of life (HRQoL) is a sub-construct of QoL. Like QoL, HRQoL is a multidimensional construct that has been defined in several ways. HRQoL has been generally conceptualized as the domains of QoL specifically related to health; that is, it includes the impact of health, disease, and treatment on QoL. Similar to QoL, the construct of HRQoL is also composed of both subjective and objective components. HRQoL includes individuals' perceptions with regard to physical, psychological, social, and other domains of health, as well

as individuals' actual functioning and objective ratings in the context of these domains. HRQoL has also been defined with regard to values assigned to particular health states, for example, as "the value assigned to duration of life as modified by the impairments, functional states, perceptions, and social opportunities that are influenced by disease, injury, [and] treatment" (Patrick & Erickson, 1993, p. 22).

The construct of HRQoL arose in order to complement traditional indices of health used in the medical field, namely, mortality and morbidity. While research on QoL began in the 1950s, interest in HRQoL increased in the 1970s. With greater prevalence of chronic versus acute disease and reductions in disease-related mortality due to advancements in medical technologies, an increasing number of individuals began living with chronic disease for extended periods of time. HRQoL arose as an important indicator of health in these contexts, in which outcomes such as mortality fail to capture the health status of an individual coping with a long-term chronic disease and its treatment.

Measurement

HRQoL has typically been assessed through self-report measures because of the need to capture people's perceptions of their health-related status. Many instruments are completed in paper-and-pencil or computer-administered versions; measures can also be administered via in-person interview or telephone or even by proxy (e.g., caregiver) if the individual is unable to complete the measure. HRQoL measures can be generic and/or specific. Generic measures can be used with any adult population, regardless of diagnosed disease, treatment received, or demographic background. In contrast, specific measures are tailored to a particular disease, condition, treatment setting, symptom, or population.

Generic Self-Report Measures of HRQoL

Generic HRQoL measures allow for assessment across groups with varying disease types, disease severity, and demographic characteristics. Generic measures are broad in nature and assess attributes of QoL that are applicable to diverse groups and diseases. Generic measures are commonly used in cost-effectiveness studies comparing outcomes across various groups or interventions. While such measures allow for cross-group comparison, these instruments may not capture important aspects of HRQoL specific to a particular disease or group.

Examples of commonly used generic measures of HRQoL include the Medical Outcomes Study 36-Item Short-Form Health Survey (SF-36) and shortened versions of the instrument (RAND Corporation, 2017; Ware & Sherbourne, 1992), the World Health Organization Quality of Life (WHOQOL) assessments (WHOQOL-100 and WHOQOL-BREF) (WHOQOL Group, 1997, 1998), Health and Activity Limitation Index (HALex) (Erickson, 1998), EuroQol-5 Dimension (EQ-5D) (Brooks, Rabin, & de Charro, 2003), Health Utilities Index Mark 2 (HUI2) and Health Utilities Index Mark 3 (HUI3) (Horsman, Furlong, Feeny, & Torrance, 2003), Quality of Well-Being Scale Self-Administered (QWB-SA) (Seiber, Groessl, David, Ganiats, & Kaplan, 2008), and Patient-Reported Outcomes Measurement Information System Global Health Scale (PROMIS Global Health) (Hays, Bjorner, Revicki, Spritzer, & Cella, 2009). Additionally, quality-adjusted life years (QALYs) can be calculated from several generic measures to allow for broad comparisons across studies (Kaplan & Frosch, 2005).

The SF-36 is a 36-item measure of health status that is one of the most widely used generic measures of HRQoL worldwide (Ware & Sherbourne, 1992). The instrument assesses eight

domains for which scores are calculated, namely, “(a) limitations in physical activities because of health problems; (b) limitations in social activities because of physical or emotional problems; (c) limitations in usual role activities because of physical health problems; (d) bodily pain; (e) general mental health (psychological distress and well-being); (f) limitations in usual role activities because of emotional problems; (g) vitality (energy and fatigue); and (h) general health perceptions,” and additionally contains a single item measuring perceived change in health (Ware & Sherbourne, 1992, p. 473). Shortened versions of the SF-36 include Short Form-6 Dimension (SF-6D), 12-Item Short Form Survey (SF-12), and 20-Item Short Form Survey (SF-20) (RAND Corporation, 2017).

The WHOQOL-100 and the WHOQOL-BREF are two widely used measures of QoL developed by the WHOQOL Group (WHOQOL Group, 1997, 1998). The WHOQOL-100 and WHOQOL-BREF are included in this section on HRQoL assessment tools because the instruments were designed for use and are commonly used in health contexts (WHOQOL Group, 1997). Notably, because most assessment instruments have been developed for use in one specific cultural setting, the WHOQOL Group designed the WHOQOL-100 and the WHOQOL-BREF as assessment tools that could be used internationally in health contexts both within and across various cultures. The WHOQOL-100, a 100-item measure, was developed in fifteen centers simultaneously around the world (WHOQOL Group, 1997, 1998). The WHOQOL-100 contains items that assess overall QoL and health as well as 24 facets of QoL, which can be grouped into 4 domains: physical, psychological, social relationships, and environment (WHOQOL Group, 1998). The instrument generates scores for overall QoL and health, facets of QoL, as well as for each of the four domains (WHOQOL Group, 1997). The WHOQOL-BREF, a 26-item short form of the WHOQOL-100, was developed thereafter and contains items measuring both overall QoL and the same facets of QoL assessed by the WHOQOL-100 (WHOQOL Group, 1997). The WHOQOL-100 and WHOQOL-BREF are available in more than 20 different languages and are used to capture health both within and across cultures (WHOQOL Group, 1997).

The HALex is a measure of health that takes into account two components: (a) perceived health and (b) perceived activity limitation, that is, an individual’s perception of their ability to perform age-relevant social roles (Erickson, 1998). The HALex yields a total score representing HRQoL that is a composite of the aforementioned components (Erickson, 1998).

The EQ-5D is a measure of perceived health status that is used with individuals with a variety of health conditions (Brooks et al., 2003). The EQ-5D is composed of five domains: mobility, self-care, usual activities, pain/discomfort, and anxiety/depression. The instrument yields five one-digit numbers expressing how problematic each domain is for the respondent. The five one-digit numbers are combined into a single five-digit number representing a single summary index of health. Additionally, the measure has a visual analog scale, which provides a measure of perceived general health status (Brooks et al., 2003).

The HUI2 and HUI3 are multidimensional measures of perceived health status (Horsman et al., 2003). Both instruments yield information about overall health as well as functioning pertaining to specific health-state attributes. HUI2 provides information about seven health-state attributes (sensation, mobility, emotion, cognition, self-care, pain, and fertility), whereas the HUI3 provides information about eight health-state attributes (vision, hearing, speech, ambulation, dexterity, emotion, cognition, and pain). Both the HUI2 and HUI3 are composed of two systems. One system, the “generic comprehensive health status classification system,” provides written descriptive ratings of a person’s ability or disability with regards to each health-state attribute (Horsman et al., 2003, p. 2). The other system, the “generic HRQoL utility scoring system,” yields scores representing level of morbidity for

each health-state attribute as well as a score representing overall health (Horsman et al., 2003, p. 2). HUI2 and HUI3 differ with regard to the health-state attributes each include as well as the fact that HUI3 descriptive ratings are more detailed than those of HUI2 (Horsman et al., 2003).

The QWB-SA is a measure of perceived health status commonly used in cost-effectiveness research (Seiber et al., 2008). The instrument is composed of five parts that assess (a) chronic symptoms, (b) acute physical symptoms and mental health symptoms, (c) mobility, (d) physical activity, and (e) social activity. The QWB-SA yields a single index measuring wellness.

PROMIS Global Health is a measure of perceived overall health (Hays et al., 2009). The measure was created through the National Institutes of Health (NIH) Patient-Reported Outcomes Measurement Information System (PROMIS) Roadmap initiative, which focuses on the development of a computerized system measuring patient-reported outcomes. PROMIS Global Health contains ten items and yields summary scores for physical health and mental health. Items from PROMIS Global Health can also be examined separately to assess perceptions of general health, social roles, physical function, pain, fatigue, and emotional problems (Hays et al., 2009).

QALYs are an additional index that can be used to summarize health outcomes (Kaplan & Frosch, 2005). QALYs summarize health status by taking both morbidity and mortality into account. QALYs describe “well years of life,” that is, they describe years of life with adjustments for HRQoL (Kaplan & Frosch, 2005). QALYs apply weights to years of life lived to describe the value of HRQoL each year. QALYs range from 0.0 to 1.0, representing death and optimal health, respectively. QALYs are advantageous as they can be computed from several commonly used measures of HRQoL including the SF-6D, HALex, EQ-5D, HUI2, HUI3, and QWB-SA and then used to make broad comparisons across various studies or interventions.

Specific Self-Report Measures of HRQoL

Specific HRQoL measures provide in-depth assessment of attributes relevant to groups of individuals with specific health conditions or challenges. These measures allow for detailed assessment of HRQoL in the context of a particular disease, condition, treatment, symptom, or population. Disease-specific measures often exhibit greater sensitivity to change than generic measures and are commonly used in studies assessing clinically significant changes in HRQoL within a given population or group. Specific measures, however, do not allow for comparisons across different groups or diseases and have limitations when used with individuals with multiple medical comorbidities.

An example of a disease-specific measure is the European Organisation for Research and Treatment of Cancer (EORTC) Core Quality of Life Questionnaire (QLQ-C30) (van Andel et al., 2008). The EORTC QLQ-C30 is a measure of HRQoL developed specifically for use in the context of cancer. Add-on modules to disease-specific measures are also available. For instance, the EORTC Quality of Life Questionnaire module for prostate cancer (QLQ-PR25) is an example of an add-on module to the EORTC QLQ-C30 that specifically evaluates HRQoL in the context of prostate cancer (van Andel et al., 2008). Specific measures of HRQoL also exist as stand-alone instruments. An example of a specific stand-alone instrument is the Expanded Prostate Cancer Index Composite for Clinical Practice (EPIC-CP) (Chang et al., 2011). This instrument assesses attributes of HRQoL specifically integral to prostate cancer including urinary incontinence and irritation and bowel, sexual, and vitality/hormonal health that would likely not be captured using generic measures.

Interventions

Interventions exist to improve HRQoL. Patient self-management is the main intervention used in this regard. Patient self-management is a multicomponent psychoeducational intervention designed to facilitate a patient's daily management of all aspects of a chronic illness including its symptoms, treatment, and physical and psychological sequelae (Jonkman, Schuurmans, Groenwold, Hoes, & Trappenburg, 2016), with the ultimate goal of improving or maintaining HRQoL. Self-management interventions emphasize the role of the patient in conjunction with a healthcare team in managing a chronic illness. Patient self-management can be implemented in both group and individual formats and often focuses on patient education as well as the acquisition of skills such as effective problem-solving and decision making, resource utilization, and action taking. Self-management interventions designed to improve HRQoL exist for a variety of chronic illnesses including arthritis, diabetes, asthma, hypertension, and chronic obstructive pulmonary disease.

In addition to self-management, several additional evidence-based interventions exist to improve or maintain the various domains of HRQoL. For instance, there are evidence-based pharmacological (e.g., pain medications) and behavioral (e.g., sleep hygiene) interventions available to address physical components of HRQoL. There are also evidence-based interventions available to address components of the psychological domain of HRQoL, such as cognitive behavioral therapy for anxiety and depression. Further, effective interventions exist to improve the social domain of HRQoL, including interventions to enhance and/or utilize social support and interventions involving or focused on caregivers. The multitude of evidence-based interventions that exist to improve patient physical, psychological, social, and other aspects of well-being can be used to promote HRQoL among individuals facing various health issues.

Cross-References

(See Also: Chronic Illness and Disability; Health)

Author Biographies

Amy E. Ustjanauskas, MS, is a doctoral student in the San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology. Amy's research is focused on helping patients and families cope with chronic illness, distress screening and coordination of psychosocial services, patient navigation, and health disparities.

Vanessa L. Malcarne, PhD, is co-director of the San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology, as well as professor in the Department of Psychology at San Diego State University, and is appointed faculty member at the University of California, San Diego Moores Cancer Center. Dr. Malcarne's research focuses on prevention and quality of life issues in serious illness, health disparities, and test construction and validation across cultural groups.

References

Brooks, R., Rabin, R., & de Charro, F. (Eds.) (2003). *The measurement and valuation of health status using EQ-5D: A European perspective*. Rotterdam: Springer-Science+Business Media.

- Chang, P., Szymanski, K. M., Dunn, R. L., Chipman, J. J., Litwin, M. S., Nguyen, P. L., ... Sanda, M. G. (2011). Expanded prostate cancer index composite for clinical practice: Development and validation of a practical health related quality of life instrument for use in the routine clinical care of patients with prostate cancer. *The Journal of Urology*, 186(3), 865–872. doi:10.1016/j.juro.2011.04.085
- Erickson, P. (1998). Evaluation of a population-based measure of quality of life: The health and activity limitation index (HALex). *Quality of Life Research*, 7(2), 101–114. doi:10.1023/a:1008897107977
- Hays, R. D., Bjorner, J. B., Revicki, D. A., Spritzer, K. L., & Cella, D. (2009). Development of physical and mental health summary scores from the patient-reported outcomes measurement information system (PROMIS) global items. *Quality of Life Research*, 18, 873–880. doi:10.1007/s11136-009-9496-9
- Horsman, J., Furlong, W., Feeny, D., & Torrance, G. (2003). The health utilities index (HUI®): Concepts, measurement properties and applications. *Health and Quality of Life Outcomes*, 1(54), 1–13. doi:10.1186/1477-7525-1-54
- Jonkman, N. H., Schuurmans, M. J., Groenwold, R. H. H., Hoes, A. W., & Trappenburg, J. C. A. (2016). Identifying components of self-management interventions that improve health-related quality of life in chronically ill patients: Systematic review and meta-regression analysis. *Patient Education and Counseling*, 99(7), 1087–1098. doi:10.1016/j.pec.2016.01.022
- Kaplan, R. M., & Frosch, D. L. (2005). Decision making in medicine and health care. *Annual Review of Clinical Psychology*, 1, 525–556. doi:10.1146/annurev.clinpsy.1.102803.144118
- Patrick, D. L., & Erickson, P. (1993). *Health status and health policy. Allocating resources to health care*. New York, NY: Oxford University Press.
- RAND Corporation. (2017). *RAND website*. Retrieved from <http://www.rand.org>.
- Seiber, W. J., Groessl, E. J., David, K. M., Ganiats, T. G., & Kaplan, R. M. (2008). *Quality of well being self-administered (QWB-SA) scale user's manual*. San Diego: Health Services Research Center University of California.
- van Andel, G., Bottomley, A., Fossa, S. D., Efficace, F., Coens, C., Guerif, S., ... Aaronson, N. K. (2008). An international field study of the EORTC QLQ-PR25: A questionnaire for assessing the health-related quality of life of patients with prostate cancer. *European Journal of Cancer*, 44(16), 2418–2424. doi:10.1016/j.ejca.2008.07.030
- Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Medical Care*, 30(6), 473–483. doi:10.1097/00005650-199206000-00002
- WHOQOL Group. (1997). Measuring quality of life. In *The World Health Organization Quality of Life Instruments (The WHOQOL-100 and the WHOQOL-BREF)*. Geneva: World Health Organization.
- WHOQOL Group. (1998). The World Health Organization Quality of Life Assessment (WHOQOL): Development and general psychometric properties. *Social Science & Medicine*, 46(12), 1569–1585. doi:10.1016/S0277-9536(98)00009-4

Suggested Reading

- Office of Disease Prevention and Health Promotion. Healthy people website. Retrieved from <https://www.healthypeople.gov>.

The Roles of Health Psychologists in Surgery

Lauren Decaporale-Ryan¹ and John D. Robinson²

¹*Departments of Psychiatry, Medicine, & Surgery, University of Rochester Medical Center, Rochester, NY, USA*

²*Department of Surgery and Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine/Hospital, Washington, DC, USA*

Clinical health psychologists today are uniquely prepared to impact healthcare systems with expertise and knowledge in the biopsychosocial framework, impacts of medical illness on psychological well-being, communication, and systems thinking. With such skill sets, psychologists have increasing opportunity to care for patients in medically integrated settings and to serve as educators to interprofessional learners. Though interprofessional roles have mainly evolved in primary care settings, they are vital in all medical specialties, as today's healthcare systems must pay increased attention to patient satisfaction and incorporation of patients' psychosocial needs. While surgical departments have long appreciated the importance of consultation for preoperative evaluations, there is increasing awareness among surgeons of the value of psychological consultation and intervention in other areas as well.

Surgeons receive a great deal of technical training but can significantly benefit from psychologists' knowledge and analytical approach to problem solving. Specifically, psychologists are well versed in subjects pertinent to surgical practice, including ethics, professionalism, and burnout. This chapter provides recommendations on how to orient oneself to surgical culture and explores the possible roles psychologists may find themselves in, or wish to create, in surgery departments. Special considerations are offered that will influence psychologists' work in surgical environments.

Surgical Culture: The Importance of Observation

Psychologists are trained in the art of behavioral observation with the awareness that observation allows understanding. It is imperative when entering any setting outside our normal purview that we apply these skills to best understand the culture. Knowledge of cross/multicultural issues enables clinical health psychologists to interact effectively in various environments and with different professional groups. When entering a world that is unknown and for which we are not trained, we must take time to become better informed about the environment and its people before offering recommendations. Though surgical settings are influenced by individual system factors, there are certain commonalities across surgery departments. These features deeply influence the collaborative work that occurs between psychologists and surgeons. For example, surgery departments do not function solely from nine to five. To effectively save lives, surgeons are on call 24/7 and operate at all hours of the day and sometimes through the night; this is a drastic difference in culture for a psychologist.

Action Oriented

The mindset of a surgeon must also be understood, as it is generally different even from other physicians (Katz, 1990; Page, 2011) and especially from that of a psychologist. While psychology emphasizes supporting others to identify a problem and develop a solution, surgeons traditionally are action oriented (Page, 2011), tending to see a problem and quickly fix it. Speed and decisiveness are critical skills in the operating room (OR); surgeons frequently face life-or-death situations, which require immediate action and could have drastic consequences if delayed. They are trained to be both confident and decisive (Katz, 1990). This approach is of course what is medically necessary but can be deleterious when managing things other than direct patient care. Surgeons, like all other physicians, should be encouraged to take time for reflection. Such introspection can reduce risk of surgeon burnout and improve the patient and team experience (Page, 2011).

In this context, psychologists have much to offer to surgical culture. Psychologists can encourage surgeons to methodically evaluate non-urgent situations so that they can more wholly define the problem, give more thorough explanations, and provide a more comprehensive plan of intervention. Application of their critical thinking skills and analytical approach can then result in more meaningful and long-lasting solutions for nonsurgical team members and surgical systems. Facilitation of such reflection and analysis also supports development of a healthy team environment that allows for information exchange and sharing of ideas outside of the OR.

Surgical Hierarchy

The surgeon is a part of a much larger team, which often reflects a defined and established hierarchy. Though other settings may have deemphasized hierarchy, it remains a part of surgical culture. Surgeons' leadership styles differ from other medical disciplines in authority and responsibility, which are necessary due to the action-oriented structure of surgery and the need to intervene quickly. It is important to understand that there is a reason this particular hierarchy exists and observe how and why it works and can be more efficient. One may also observe that this system is personality driven and suits the general personality of surgeons, which may be markedly different than internist, psychiatrists, and other physicians. The psychologist's knowledge of personality styles is invaluable. An understanding of the system will also allow the clinical health psychologist to apply his or her knowledge in a very effective

manner. It is important to be a member of the team with skills that become valued by its constituents. Being part of a team and recognized as a specialist with unique skills is important just as the various surgical specialists (plastic, ENT, CT, etc.) are each valued members of the surgical team with different skills to offer.

Creating an environment where such hierarchy is respected but also allows for shared communication can be challenging. One must respect others but challenge inappropriate behaviors, lack of professionalism, and poor communication when necessary. When hierarchy is appropriately managed, it promotes patient safety and supports education. Prentice (2007) has noted that hierarchy allows surgical faculty to evaluate both the technical and social readiness of learners in the OR and their preparedness to take on more tasks. Psychologists can help promote and teach about healthy hierarchical boundaries, ensuring that hierarchy does not endorse microaggressions (or outward aggressions) and instead values the importance of collegiality and collaboration.

Different Divisions, Different Needs

It is important for a psychologist working in a surgical environment to remember that their job is not to make the surgeon a psychiatrist, but to make them a better, more effective surgeon. As psychologists become acquainted with the surgery environment, they will also find that different surgical specialties have unique attributes and needs. The demands on a trauma surgeon are unlike those placed on a cardiothoracic surgeon. The skills (technically, cognitively, interpersonally) required of each setting are intricate and determined by the clinical operations, the teams, and the larger medical setting in which a surgery service is embedded. Often times, these details may result in apparent changes in individual and team behavior and communication.

Moreover, these distinctions influence the learning environment and how trainees engage with faculty surgeons. For example, surgical residents in training may find that they have greater autonomy and ability to run a team when the stress level is deemed more manageable; conversely, they may have to rely more heavily on faculty guidance or work more closely with mid-level providers if the residents' knowledge base and technical skill are not adequate enough for safe patient care. Recognizing these differences in settings allows psychologists to adapt and appropriately assess, consider, and respond to the needs of surgeons in these cultures effectively. How a psychologist might become integrated, give feedback, or teach in one surgical setting may not apply in the very next corridor. Psychologists should use their observational and assessment skills to determine the needs in that particular setting based on their assigned duties.

Surgeons Are Stressed, and So Are the Patients

Surgery goes beyond the scalpel and sutures and leaves scars, both seen and unseen (Balch, Shanafelt, Sloan, Satele, & Freischlag, 2011). Anton, Montero, Howley, Brown, and Stefanidis (2015) note that 40% of surgeons reported witnessing an intraoperative complication that was directly related to the surgeon's stress. Stress in the OR is just one forum that challenges the psychological well-being of surgeons; this clinician population must also deliver bad news and communicate about death and other forms of loss to patients and families.

Moreover, surgery to a patient is much more than a procedure. Surgery has significant implications for patients and their loved ones, as it creates personal, professional, and financial disruptions that are often unexpected (Petry, 2000). In this context, patients' anxiety can be heightened, changing how they interface with the medical team. Psychologists are well

prepared to recognize the signs and symptoms of anxiety (or other psychological distress) and can help surgeons' identify and respond to such affect so that patients are heard and understood. Meeting the needs of patients while also responding to the administrative and revenue obligations required by the hospital system is taxing. Psychology has the opportunity to offer consultation regarding coping strategies to enhance surgeons' effectiveness and support their emotional well-being as well.

Observations Made, Now What?

The psychologist must not only observe surgical culture but also become a part of the milieu. This is no easy task. It requires a clear role, tough skin, and a surgical champion who supports the psychologist's work. Surgery departments may not know the exact ways in which psychology can contribute. However, when surgeons identify and acknowledge a need for support of any kind, there are many opportunities for psychology to critically influence the environment. To do so most effectively, psychologists should strongly advocate to be directly embedded in the department. There are some relatively easy ways to initiate this type of working relationship, such as having an office on a surgical floor: be centrally located to the rest of the department so that faculty, staff, and learners have access to share information, exchange ideas, and ask questions. Additionally, based on the defined role and responsibilities, psychologists should find ways to become integrated into meetings, training curriculums, and team rounds. Such efforts will also help identify additional roles that psychologists might be interested in and well trained to support.

In order to have maximum effectiveness, it is valuable for the psychologist to be seen as a part of the surgical faculty, not as a visitor from another department with no ties to surgery. Therefore, a dual/joint faculty appointment with surgery is ideal. Once in the department, the psychologist should make attempts to not only understand the difference in culture but to also be seen as part of it; as a dedicated member of the team, the psychologist can make significant contributions.

The Potential Roles of Psychology in Surgery

Exploration of the culture and observations may reveal multiple possible roles for the psychologist who wishes to support surgical departments. Clinical health psychologists are well prepared to take on any number of tasks from preoperative patient evaluations to assessment and consultation regarding team effectiveness and individual clinician well-being to administrative and teaching roles. Robinson and Baker (2006) have discussed some of these roles and duties in a general medical/academic health center. Surgical faculty, staff, and learners will value psychologists' ability to listen and help them deal with concerns that impact the efficiency of the department.

Psychologists as Educators

Psychologists are uniquely positioned as experts in communication to support a breadth of training experiences in surgical education and for medical teams. While physicians have some exposure to communication techniques that support enhanced patient care and clinician efficiency, such methods are rarely reiterated throughout surgical residency. Psychologists can provide consultation regarding best practices at the bedside and also offer more knowledge of

how to implement techniques like teach-back and balancing open and close-ended questions. There is clear evidence that communication has direct impact on patient care experiences, influencing postoperative outcomes, malpractice lawsuits, and general patient satisfaction (Greenberg et al., 2007). In this context, psychologists are appropriately suited to provide education to surgical residents, faculty, and staff about how to best approach patient and family dialogue, thus enhancing the physician–patient relationship. Models of this vary from one institution to the next but can include didactic training, simulation exercises, or even one-to-one coaching.

Such training initiatives can also reveal the other learning needs of clinicians in surgery. For example, surgeons are expected to run teams but may have minimal training (aside from lived experience) about team culture, managing different personality styles, and pertaining to leadership. Additionally, each surgeon (like all of us) comes with their own strengths and shortcomings as a leader: some have an innate ability to take a systems' perspective, while others struggle to build a trusting relationship with even one team member. Psychologists, especially those with specialized training in medical integration and systems thinking, can offer important perspective that supports development of surgeons as professionals in nonoperative roles and as leaders of the medical field.

Presently, the Accreditation Council for Graduate Medical Education (ACGME) requires assessment of a range of competencies. These competencies not only reflect knowledge about patient and team communication but also reflect the need to train clinicians who carry themselves professionally and who can navigate complex medical ethics and understand the biopsychosocial needs of patients. Psychologists offer unique perspective on these subjects and have opportunity to create psychosocial curriculum including content in ethics and professionalism. Such training can be built into surgical education over the course of a resident's education.

Notably though, the psychologist who serves in this educational capacity must articulate and maintain clear boundaries. Specifically, the psychologist who serves as an educator (and therefore evaluator of knowledge and behavior) should not also serve as a clinician to residents or faculty members. In such instances, it is important to remind others of the role as an educator and support referral to an outside clinician (ideally someone who is familiar with the demands of medical settings).

Psychologists as Clinicians

Of course, psychologists often serve as direct clinical care providers. Psychologists are increasingly embedded in surgical departments or sought as consultants for preoperative assessments. In this context, psychologists can conduct psychosocial evaluations that help to identify risk factors that may influence surgical outcomes. Bariatric and transplant surgeries have both demonstrated the importance of psychological evaluation. Preoperative psychological evaluations are now required for metabolic and bariatric surgery centers to be accredited by the American College of Surgeons and the American Society for Metabolic and Bariatric Surgery (American College of Surgeons, American Society for Metabolic and Bariatric Surgery, 2014; Mechanick et al., 2013). Similarly, preoperative psychosocial evaluations for transplant surgery have been shown to positively contribute to the ultimate success of surgery and overall care (Dobbels, Verleden, Dupont, Vanhaecke, & De Geest, 2006).

Evaluations identify patients' available social supports, level of cognitive functioning, and capacity to implement and sustain behavioral change after surgery. As such, psychologists can speak to individuals' strengths and vulnerabilities and offer recommendations to enhance the success of surgical intervention and mitigate possible risk (Dobbels et al., 2006). Recommendations may include additional psychoeducation to support patients' full understanding of the surgery

or necessary behavior change afterward, pharmacological management of mood, and psychotherapy pre- and/or post-surgery to support adjustment to physiological changes. The psychoeducational group approach has been used by organ transplant programs for some time (Stewart, Kelly, Robinson, & Callender, 1995).

Psychologists can also provide clinical care in response to conditions such as needle phobias, distress in preparation of surgery, trauma related to past healthcare experiences, and so on. Such interventions are brief in nature and focused on improving patients' coping strategies, problem-solving abilities, and general management of anxiety in healthcare settings (Petry, 2000). When able, psychologists should offer these services in the healthcare setting, allowing for ease of access to care, collaboration with patients' treatment team, and an opportunity to observe patients' needs in this environment. It is important to remember that when patients are facing surgery, this can be one of the most stressful periods of time in their lives (Petry, 2000). Psychologists can support patients through this period and can also help surgeons better recognize and respond to patient affect.

When surgeons struggle with this work or do not have adequate time to explore the psychosocial needs of patients, clinical health psychologists may find that this results in increased referrals for therapeutic intervention. In these cases, the psychologist should definitely be aware of shared information with the clinical team and make these limits on boundaries of confidentiality clear to the patient. This work may frequently focus on providing psychoeducation on the mind-body relationship and the influence of stress on physiological health and about the manifestation of somatic symptoms. With a collaborative approach, patients' biopsychosocial needs are more readily identified, and psychologists can work with patients to reduce distress and physiological symptoms, including pain (Turk & Gatchel, 2013) and GI distress (Palsson & Whitehead, 2013).

Psychologists as Consultants

Psychologists can provide a range of additional consultation that reflects the unique range of skills of the discipline. Similar to what is outlined above, clinical health psychologists embedded in healthcare settings, including surgical departments, can provide consultation regarding patients. Such consultation might be pertaining to acute management of anxiety, in support of facilitating effective family meetings, or if there is awareness that psychiatric symptoms are hindering establishment of safe discharge planning. Such roles are more frequently and formally met by psychiatric services. The surgical clinical health psychologist may be asked to provide formal consultations or more informal "curbside" consultation.

Additional roles of consultation that directly support surgical colleagues are easily identifiable. Surgeons face many professional stressors, including tragic and unanticipated losses (Shanafelt et al., 2009). Though surgeons have the opportunity to learn from such events through morbidity and mortality conferences, this venue focuses on clinical skill and how management of patient care could have been navigated differently. Others have advocated that surgeons should also have the opportunity to explore their emotional reactions following adverse events to support their learning, growth, and healing (Plews-Ogan et al., 2016). Psychologists can offer space to process the emotional aspects of patient death and more broadly as it relates to the demands of the job. Such consultation can occur on a one-to-one basis or in group venues that allow for exploration of affect, normalizing grief, and establishment of coping strategies to minimize distress.

It is not uncommon that the professional stress that surgeons face creates interpersonal struggles outside of work and psychological impairment. Surgeons, like physicians across many

other disciplines, face high rates of burnout, depression, and increased substance abuse (Oreskovich et al., 2014; Spickard, Gabbe, & Christensen, 2002). Moreover, they may struggle to share their experiences with family and friends who cannot fully appreciate the nature of their work with significant impact on their family system. Home life experiences and conflict between work and home balance influence rates of burnout among surgeons (Dyrbye, Shanafelt, Blach, Satele, & Fresichlag, 2011). Psychologists can promote a culture that supports individual wellness and/or may provide direct care to surgeons. It is important to note that psychologists who serve as educators and administrators should not provide this direct clinical care to individuals but can support establishment of referrals to others who have sophisticated knowledge of medical culture. However, those embedded in surgery departments can help establish a curriculum that promotes wellness, training surgeons about techniques that support mindfulness and reduced distress.

Special Considerations

The OR and Other Elements of the Clinical Environment

Physicians' exposure to the OR occurs early in their medical school training and continues for surgeons through residency and often fellowship training. Surgeons are required to participate in a defined number of major operative cases with a designated role during the course of their residency training as defined by ACGME. This means they are well equipped to navigate cutting, suturing, knotting, and the like. The capacity to engage in this work requires extraordinary technical skill, a balance between detachment and appropriate intimacy with patients (Katz, 1990), and critical thinking and confidence to respond to changes in medical status quickly and through crisis. To be most effective at this work, surgeons also must know their team members well and have an ability to communicate instructions, needs, and plans. Not everyone can do this work: it requires a sharp mind, a high tolerance to work under pressure, and the capacity to separate from the person behind the surgical drapes.

Psychologists may find themselves in this setting, observing the team and providing direct consultation about the surgeon's engagement with others. It is strongly recommended that the clinical health psychologist in a department of surgery observe at least one major surgical procedure in order to get a different understanding of the surgeon's world and the stressors involved. This also assists the psychologist in being seen as a member of the team who also has an understanding of the demands of the service and the skills needed by the team.

Psychologists doing this work should take time to understand the OR: know the setting, the expectations for team participation (including one's self), and the expected course of the procedure. Knowing how to remain out of the way to maintain a sterile environment is crucial, and yet, determining how to be able to see all aspects of the room is important if you are to observe or provide feedback regarding the team's dynamic. Knowing one's own limits is also critical to the process: the OR is not for everyone, and you may encounter things you are not accustomed to seeing.

Time

Surgery happens at all hours of the day. Though psychologists frequently provide emergency consultation and are accessible much of the time, the demands placed on a surgeon are quite different. Specifically, when considering that most surgeons have already begun operating

before 8 am, this means that education efforts, clinical collaboration, and so on must occur before that time. For psychologists, this means moving away from the expectations of a “typical” 9 am to 5 pm day. Surgeons will make themselves available on nights, weekends, holidays, and, of course, in the very early morning. To be effective, psychologists must be willing to be flexible with their time.

And Remember, You Are Not a Surgeon

It goes without saying that psychologists receive very different training than surgeons. To actively work in a surgical environment requires that psychologists not only modify their schedule but take time to best understand the experience and culture of surgery departments and the subsequent lifestyle of surgeons. This requires, in many ways, becoming a part of the surgical world. Despite this, maintaining identity as a psychologist is crucial. The job is not to be a surgeon, to act like a surgeon, or even to think like a surgeon. Psychologist’s role can be to understand how surgeons think and support them to improve efficiency, to develop a broader perspective about patient care, and, in some instances, to help them slow down.

Psychologists must be confident in their role identity and competence. As a nonsurgical clinician, psychologists may have to prove their knowledge, skill, and value. Worth will be demonstrated in the ability to positively influence patient and team interactions, to foster clinician efficiency, and to improve culture. Enter with awareness that often there will be opinions about social sciences (e.g., “this stuff is fluff, it can’t possibly matter”) and remember that this judgment is not personal (though it may feel that way). Assertive communication skills, ability to set appropriate boundaries, and grit will all support the psychologist to feel comfortable and confident in the surgical setting.

In addition to the above, clinical health psychologists in medical settings should consider seeking board certification, which is a standard medical practice. Nearly all surgeons, and most physicians, in academic health centers and large nonacademic hospitals are board certified by an organization such as the American Board of Surgery (ABS), the American Board of Psychiatry and Neurology (ABPN), etc. If psychologists seek to be seen as peers in this environment, it is important that they have a recognized credential equivalent to medical board certification. The American Board of Professional Psychology (ABPP) offers this examination based and nationally recognized board certification. Specialty certification is available in areas such as clinical psychology, clinical health psychology, clinical child and adolescent psychology, geropsychology, clinical neuropsychology, and others.

Conclusions

In sum, opportunities abound for psychologists interested in working in a surgical environment. Such work comes in many rewarding forms, allowing psychologists to express creativity, generate innovative ideas, and engage in a challenging and ever-changing landscape. Surgical environments may have a clearly defined need and identified role for psychologists entering the setting. Despite this, surgeons will look to psychologists to be the expert of their own trade to help guide program implementation and sustainability whether for education or clinical care. Clinical health psychologists must take the time necessary to build relationships with surgical colleagues and become part of the milieu to create effective change that supports patients, families, staff, trainees, and surgeons alike.

Author Biographies

Lauren DeCaporale-Ryan, PhD, is an assistant professor in psychiatry, medicine, and surgery at the University of Rochester Medical Center. She is the assistant program director of General Surgery in Wellness, the associate program director of the Primary Care Family Psychology Fellowship, and the director of the Adult Psychology Internship. She completed her doctorate in clinical psychology at the University of Missouri–St. Louis in 2011 and went on to a fellowship in Primary Care Family Psychology with specialty focus in geriatrics and internal medicine at URMC. She provides communication coaching to residents, faculty, and staff clinicians, aiming to improve both bedside manner and team operations. She is the current recipient of the George W. Merck Dean's Teaching Fellowship and is subsequently implementing and evaluating a curriculum for surgical residents to develop leadership skills.

A graduate of the University of Texas at Austin, the University of Massachusetts at Amherst, and Harvard University, John D. Robinson, EdD, MPH, ABPP, is currently professor emeritus in both the Department of Surgery and the Department of Psychiatry and Behavioral Sciences at Howard University College of Medicine. He served as chief of behavioral medicine for both the organ transplant program and the bariatric program at Howard with a clinical appointment as professor of psychiatry at Georgetown University School of Medicine. Dr. Robinson is a former Navy psychologist and former president of both the American Board of Clinical Psychology and American Board of Clinical Health Psychology (ABPP). His specialty focus is working with patients who require surgical intervention, graduate medical education, and cultural competence training. He has over 40 years of experience as a mentor to medical students, medical/surgical residents, and psychology graduate students and interns.

References

- American College of Surgeons, American Society for Metabolic and Bariatric Surgery. (2014). *Resources for optimal care of the metabolic and bariatric surgery patient 2014*. Chicago: American College of Surgeons. Retrieved from <http://www.mbsaqip.org/docs/Resources%20for%20Optimal%20Care%20of%20the%20MBS%20Patient.pdf>
- Anton, N. E., Montero, P. N., Howley, L. D., Brown, C., & Stefanidis, D. (2015). What stress coping strategies are surgeons relying upon during surgery? *The American Journal of Surgery*, 210, 846–851.
- Balch, C. M., Shanafelt, T. D., Sloan, J. A., Satele, D. V., & Freischlag, J. A. (2011). Distress and career satisfaction among 14 surgical specialties, comparing academic and private practice settings. *Annals of Surgery*, 254(4), 558–568.
- Dobbels, F., Verleden, G., Dupont, L., Vanhaecke, J., & De Geest, S. (2006). To transplant or not? The importance of psychosocial and behavioural factors before lung transplantation. *Chronic Respiratory Disease*, 3(1), 39–47.
- Dyrbye, L. N., Shanafelt, T. D., Balch, C. M., Satele, D., Sloan, J., & Freischlag, J. (2011). Relationship between work-home conflicts and burnout among American surgeons: A comparison by sex. *Archives of Surgery*, 146(2), 211–217.
- Greenberg, C. C., Regenbogen, S. E., Studdert, D. M., Lipsitz, S. R., Rogers, S. O., Zinner, M. J., & Gawande, A. A. (2007). Patterns of communication breakdowns resulting in injury to surgical patients. *Surgeons*, 204(4), 533–540.
- Katz, P. (1990). *The Scalpel's edge*. Boston, MA: Allyn & Bacon.
- Mechanick, J. I., Youdim, A., Jones, D. B., Garvey, W. T., Hurley, D. L., McMahon, M., ... Brethauer, S. (2013). Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical

- support of the bariatric surgery patient—2013 update: Cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. *Obesity*, 21(S1), S1–S27.
- Oreskovich, M. R., Shanafelt, T., Dyrbye, L. N., Tan, L., Sotile, W., Satele, D., ... Boone, S. (2014). The prevalence of substance use disorders in American physicians. *The American Journal on Addictions*, 24(1), 30–38.
- Page, D. W. (2011). Are surgeons capable of introspection? *Thoracic Surgery Clinics*, 21(3), 431–439.
- Palsson, O. S., & Whitehead, W. E. (2013). Psychological treatments in functional gastrointestinal disorders: A primer for the gastroenterologist. *Clinical Gastroenterology & Hepatology*, 11(3), 208–216.
- Petry, J. J. (2000). Surgery and complementary therapies: A review. *Alternative Therapies in Health & Medicine*, 6(5), 64–74.
- Plews-Ogan, M., May, N., Owens, J., Ardelt, M., Shapiro, J., & Bell, S. K. (2016). Wisdom in medicine: What helps physicians after a medical error? *Academic Medicine*, 91(2), 233–241.
- Prentice, R. (2007). Drilling surgeons: The social lessons of embodied surgical learning. *Science, Technology & Human Values*, 32(5), 534–553.
- Robinson, J. D., & Baker, J. (2006). Psychological consultation and services in a general medical hospital. *Professional Psychology: Research and Practice*, 37(3), 264–267.
- Shanafelt, T. D., West, C. P., Sloan, J. A., Novotny, P. J., Poland, G. A., Menaker, R., ... Dyrbye, L. N. (2009). Career fit and burnout among academic faculty. *Archives of Internal Medicine*, 169(10), 990–995.
- Spickard, A., Jr., Gabbe, S.G., & Christensen, J.F. (2002). Mid-career burnout in generalist and specialist physicians. *Journal of the American Medical Association*, 288(12), 1447–1450.
- Stewart, A., Kelly, B., Robinson, J. D., & Callender, C.O. (1995). The Howard University Hospital transplant & dialysis group: Twenty years and going strong. *International Journal of Group Psychotherapy*, 45(4).
- Turk, D. C., & Gatchel, R. J. (Eds.) (2013). *Psychological approaches to pain management: A practitioner's handbook*. New York, NY: Guilford Publications.

Suggested Reading

- Groopman, J. (2007). *How doctors think*. New York, NY: Houghton Mifflin.
- Hong, B., and Robiner, W. (2016). Psychologists in Academic Health Centers and Medical Centers: Being visible, relevant, and integral. *Journal of Clinical Psychology in Medical Settings* 23(1), 11–20. doi: 10.1007/s10880-016-9450-2
- Ofri, D. (2013). *What doctors feel: How emotions affect the practice of medicine*. Boston, MA: Beacon Press.
- Orri, M., Revah-Levy, A., & Farges, O. (2015). Surgeons' Emotional experience of their everyday practice—A qualitative study. *PLoS One*, 10(11), e0143763.
- Pories, S., Jain, S., & Harper, G. (2012). *The soul of a doctor: Harvard medical students face life and death*. New York, NY: Algonquin Books.

The Bidirectional Relationship Between Sleep and Health

Elizabeth M. Stewart¹, Shane Landry^{1,2},
Bradley A. Edwards^{1,2}, and Sean P. A. Drummond¹

¹*Monash Institute of Cognitive and Clinical Neurosciences, School of Psychological Sciences,
Monash University, Melbourne, VIC, Australia*

²*Sleep and Circadian Medicine Laboratory, Department of Physiology,
School of Biomedical Sciences, Monash University, Melbourne, VIC, Australia*

Introduction to Sleep

Sleep is “a reversible behavioural state of perceptual disengagement from and unresponsiveness to the environment” (Carskadon & Dement, 2011). Sleep is composed of two distinct states: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is further composed of three stages: N1, N2, and N3. Each successive stage represents a deeper stage of sleep. Sleep oscillates among the stages, with NREM stages followed by REM stages. This oscillation takes about 80–100 min and is repeated 3–4 times each night. The exact timing and composition of sleep episodes are complex and affected by a number of factors. A full explanation is beyond the scope of this chapter, and the interested reader is referred to several excellent sources for more information (Borbély, 1982; Carskadon & Dement, 2011; McCarley, 2007; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).

Sleep is essential for health and well-being. A recent expert consensus conference convened by the American Academy of Sleep Medicine and Sleep Research Society determined, based on an extensive literature review, that adults should regularly obtain at least 7 hr of sleep per night to promote optimal health in a variety of key areas. Less than 7 hr of sleep per night increases likelihood of obesity, diabetes, cardiovascular disease, depression, and mortality, among other negative consequences (Watson et al., 2015).

Inadequate nightly sleep has major consequences for daytime functioning, resulting in reduced alertness, poor attention and concentration, daytime sleepiness, and increased risk of

accidents, as well as compromising immune, cardiovascular, and metabolic systems (Besedovsky, Lange, & Born, 2012; Ferrie et al., 2007; Knutson, Spiegel, Penev, & Van Cauter, 2007). Cognitive performance is impaired in domains including executive function, processing speed, working memory, visual learning, memory, decision making, and judgment (Czisch et al., 2012; Diekelmann & Born, 2010; Drummond, Anderson, Straus, Vogel, & Perez, 2012; Fischer, Diekelmann, & Born, 2011; Killgore, Balkin, & Wesensten, 2006; Lim & Dinges, 2008; Wolfson & Carskadon, 1998). These deficits cannot always be compensated for by use of stimulants (Killgore, Grugle, & Balkin, 2012).

Assessing Sleep

Sleep can be assessed in a variety of ways, including objectively and subjectively, retrospectively and prospectively. Naturally, some methods are more labor intensive than others. A brief overview of the major assessment tools is presented here, from most to least labor intensive and costly.

Polysomnography (PSG)

Polysomnography (PSG) is the gold standard of objective sleep measurement. It uses electroencephalography (EEG) electrodes attached to the scalp to measure electrical brain activity, from which sleep and wake states can be distinguished. Other information about the body is measured concurrently: eye and leg movements, muscle tone, heart rate, oxygen levels in the blood, and breathing patterns (Berry et al., 2012). This provides highly accurate and clinically relevant information for diagnosis of many sleep disorders, and can be conducted either in lab or at home, using fewer numbers of electrodes. For further information, refer to reviews by Geyer, Talathi, and Carney (2009) and Vaughn and Giallanza (2008).

Multiple Sleep Latency Test (MSLT)

The Multiple Sleep Latency Test (MSLT) is an objective measure of daytime sleepiness. Standard MSLT protocols exist and are widely used in both clinical and research settings (Arand et al., 2005). Individuals have four to five designated sleep opportunities throughout the day after a typical night's sleep while connected to PSG. Each nap opportunity lasts 20 min and occurs every 2 hr. Level of sleepiness due to sleep deprivation can be assessed, as well as some sleep disorders (Carskadon & Dement, 1982). For further information, refer to Carskadon et al. (1987) and Littner et al. (2005).

Actigraphy

Sleep can be profiled objectively without PSG. Actigraphy uses a wristwatch-like device that includes an accelerometer, and often a light sensor, to measure movement as a proxy for sleep (Sadeh, 2011). This is less invasive and has greater ecological validity than an in laboratory PSG as the device can be worn at home, for many nights in a row. Practice guidelines suggest it be used in combination with a sleep diary (Kawada, 2013; Sadeh, 2011). Information collected between actigraphy and PSG is different. No brain activity or sleep architecture is recorded with actigraphy. Instead, movement allows the determination of sleep and wake. For further information, refer to Sadeh and Acebo (2002) and Ancoli-Israel et al. (2015).

Sleep Diary

The most detailed way to assess subjective sleep is with a daily sleep diary. While many investigators and clinicians use a diary of their own design, there is a consensus diary recommending the minimum information one should obtain (Carney et al., 2012). Typical measures collected with a sleep diary include bedtime and wake time, length of time to fall asleep, number and length of nocturnal awakenings, and time out of bed. Naps, caffeine and alcohol intake, and a variety of other variables are also commonly obtained. When compared with PSG, healthy individuals tend to overestimate their sleep, while people with insomnia underestimate their sleep (Bixler, Kales, Leo, & Slye, 1973). Nonetheless, it is highly useful, particularly in diagnosis and treatment of sleep disorders such as insomnia (Morin & Espie, 2003). Indeed, sleep diaries form one of the key underpinnings of cognitive behavioral therapy for insomnia (CBTI), discussed later in this chapter. For more information about use of sleep diaries, refer to Carney et al. (2012).

Self-Report Questionnaires

Validated questionnaires are widely utilized in both healthy populations and those with sleep, psychiatric, and medical disorders. Some of the most common include the Pittsburgh Sleep Quality Index (global sleep quality) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), the Epworth Sleepiness Scale (trait sleepiness) (Johns, 1991), Karolinska Sleepiness Scale (state sleepiness) (Åkerstedt & Gillberg, 1990), the Insomnia Severity Index (insomnia symptoms) (Morin, Belleville, Belanger, & Ivers, 2011), the STOP-BANG (sleep apnea risk) (Chung et al., 2008), and the Horne–Ostberg Morningness-Eveningness Questionnaire (chronotype) (Horne & Ostberg, 1976).

The Bidirectional Relationship Between Sleep and Health

There is a bidirectional relationship between sleep and health. Sleep problems increase the risk of developing various physical and mental health conditions, and in turn, many disorders have characteristic sleep sequelae.

Poor sleep comes in many forms. Within this chapter, three major categories of poor sleep will be discussed: (a) inappropriate sleep quantity, (b) insomnia, and (c) sleep-disordered breathing (SDB), in particular obstructive sleep apnea (OSA). All three of these categories have direct links with physical and mental health and carry huge economic burdens (Deloitte Access Economics, 2011; National Institutes of Health, 2005). Targeting sleep as a health behavior has downstream effects for other health domains. By treating the sleep problems, many comorbid health problems improve too. The following sections provide an overview of the relationship between sleep and health in each of these three areas, greater detail on one specific aspect of health within each, and an overview of how treating sleep can improve health.

Inappropriate Sleep Quantity

Like many things in health, sleep works on the principle of optimization, as opposed to the principle that more is better. Epidemiological studies show there is an inverted U-shaped curve describing the relationship between sleep and health (Kojima et al., 2000; Kripke,

Garfinkel, Wingard, Klauber, & Marler, 2002; Kripke, Simons, Garfinkel, & Hammond, 1979; Patel et al., 2004). This relationship was first observed by Hammond (1964), where the lowest risk of mortality was observed in individuals sleeping 7 hr per night. This has been consistently replicated in the literature since, with most studies showing 7–7.5 hr being optimal for health outcomes (Cai et al., 2015; Ferrie et al., 2007; Hublin, Partinen, Koskenvuo, & Kaprio, 2007; Kripke et al., 2002; Patel et al., 2004). As sleep duration becomes progressively further away from this in either direction, risk of poor health outcomes, including mortality, increases. Interestingly, though, the specific negative health outcomes of short versus long sleep duration often differ.

Sleep Duration and Health

The most obvious consequence of short sleep duration is daytime sleepiness. However, short sleep also has more dangerous health effects. Sleep loss causes characteristic neurological (EEG) changes, alterations in autonomic functions including cardiovascular function, respiration rates, biochemical and metabolic changes, and altered gene expression, due to stress placed on the body by prolonged wakefulness with insufficient recovery sleep between wake periods (Aho et al., 2013; Banks & Dinges, 2007; Buxton et al., 2012). These underlying physiological processes then lead to poor health outcomes. Bidirectional relationships exist between short sleep (<6 hr) and weight gain/obesity, impaired glucose tolerance, type 2 diabetes mellitus (T2DM), hypertension, cardiovascular disease, stroke, anxiety symptoms, depression, impaired immunity, increased pain, and increased risk of accidents and mortality (Dew et al., 2003; Ferrie et al., 2007; Karimi, Hedner, Häbel, Nerman, & Grote, 2015; Prather, Janicki-Deverts, Hall, & Cohen, 2015; Sivertsen et al., 2015; Watson et al., 2015; Zhang & Chan, 2014). Additionally, animal studies have shown that complete sleep loss kills animals in approximately the same amount of time as total food deprivation (Rechtschaffen & Bergmann, 1995).

Long sleep is also associated with increased mortality, perhaps to an even greater extent than short sleep. In an epidemiological study by Patel, Malhotra, Gottlieb, White, and Hu (2006), long sleepers had increased likelihood of depression, lack of physical exercise, and lower socioeconomic status, all of which are independently associated with poor health. Long sleepers were also more likely to have a diagnosis of medical and psychiatric disorders including diabetes, obesity, hypothyroidism, seizures, multiple sclerosis, stroke, cardiovascular disease, hypertension, rheumatoid arthritis, osteoarthritis, herniated disc, asthma, chronic obstructive pulmonary disease, cancer, inflammatory bowel disease, anxiety disorders, depression, restless legs syndrome, and snoring (Ohayon, 2004; Patel et al., 2006).

The reasons long sleep is associated with increased mortality are less clear than the well-established links between short sleep/sleep deprivation and health. Grandner and Drummond (2007) identified six main contributors to the link between long sleep and poor health: sleep fragmentation, fatigue, altered immune function, circadian abnormalities, lack of physiological challenge, depression, or underlying disease processes (e.g., OSA, heart disease, or failing health). In addition, reports of sleep duration may actually represent total time in bed, rather than truly time asleep (Ferrie et al., 2007). Long time in bed often reflects increased sleep fragmentation, which in its own right has significant associations with poor health (Bennett, Barbour, Langford, Stradling, & Davies, 1999; Dew et al., 2003). In essence, long sleep may be problematic because it is a proxy for general poor or declining health, rather than mechanistically responsible for poor health outcomes.

Based on the large sleep and health literature, sleeping 7–7.5 hr per night is thought to be optimal for health, and as cited above, the current recommendation is that adults sleep a

minimum 7 hr per night. Worryingly, fewer hours of sleep and greater levels of sleep disturbance have become widespread in industrialized societies, largely due to trading off sleep to fit in more work and leisure activities (Bartlett, Marshall, Williams, & Grunstein, 2008; Ferrie et al., 2007). Such voluntary restricted sleep duration is a common occurrence in modern society. In Australia, a 2010 community study found 18.4% of adults slept less than 6.5 hr per night and 11.7% reported chronic sleepiness (Bartlett et al., 2008). This prevalence is even higher in the United States: 30% of all workers and 44% of night shift workers reported sleeping less than 6 hr per night (Luckhaupt, 2012). Some populations are more prone to obtaining insufficient sleep and/or experiencing sleep disruption than others, including shift workers, physicians, truck drivers, parents, and teenagers. This increases risk of many health problems and also directly impacts daytime functioning the day after a night of short sleep.

IMPAIRED GLUCOSE TOLERANCE

One important example of a negative health consequence of inappropriate sleep duration is impaired glucose tolerance. The importance of sleep to glucose metabolism, and the influence of sleep on development of T2DM, is well documented. Epidemiology studies indicate both short and long sleepers are at significantly increased risk of developing T2DM (Cespedes et al., 2016). A meta-analysis by Shan et al. (2015) showed the risk of T2DM with suboptimal sleep length, finding in 482,502 participants with follow-up between 2.5 and 16 years that the lowest risk of T2DM was seen in those sleeping 7–8 hr per night. For each hour regularly slept below this, T2DM risk significantly increased by 9%, and for each hour above 8 hr, risk increased by 14%.

The process by which short sleep¹ leads to glucose abnormalities is well documented. Short sleep leads to decreased glucose tolerance and insulin sensitivity and reduced glucose disposal throughout the body (Buxton et al., 2010; Donga et al., 2010; Schmid et al., 2011; Spiegel, Leproult, & Van Cauter, 1999). In a seminal study, glucose tolerance was 40% lower after only four nights of short sleep, relative to long sleep, leading to a 30–40% decrease in glucose effectiveness in the body (Spiegel et al., 1999). During short sleep, sympathetic nerve activity increases, resulting in reduced β -cell responsiveness and reduced pancreatic insulin secretion (Buxton et al., 2012). At the same time, the sleep-deprived brain increases its glucose uptake, increasing circulating glucose levels (Herzog et al., 2013). Short sleep duration also leads to food cravings and increased calorie intake: sleep-restricted individuals increase their daily energy intake by up to 20% (Nedeltsheva et al., 2009; St-Onge et al., 2011). Therefore, short sleep duration contributes to insulin resistance and T2DM mechanistically through both reduced insulin production and increased glucose levels. Recent prospective research indicates some of these negative effects on metabolism and glucose tolerance may be due to inappropriate timing of food consumption throughout the day, rather than due to short sleep per se (Brianc on-Marjollet et al., 2015; Gale et al., 2011; Sherman et al., 2012). Nonetheless, inappropriate timing of eating in these cases is still driven by the short sleep opportunity.

In addition to increasing risk of T2DM directly through changes in insulin and glucose, short sleep duration increases T2DM risk through multiple indirect pathways. For example, epidemiological and experimental studies show an association between short-duration sleep, high ghrelin, low leptin, and increased obesity risk, which is itself a risk factor for T2DM (Schmid, Hallschmid, Jauch-Chara, Born, & Schultes, 2008; Spiegel et al., 2004; Taheri, Lin, Austin, Young, & Mignot, 2004). Therefore, a combination of several endocrine abnormalities leads to the development of T2DM and obesity in short sleepers.

The reciprocal relationship between T2DM and sleep also exists. Individuals with T2DM are at increased risk of restless legs syndrome and OSA, which have debilitating effects on sleep

and health (Lopes et al., 2005; Resnick et al., 2003; Sanders & Givelber, 2003). The association with OSA is thought to be due to metabolic abnormalities and not due to other common risk factors such as weight (Peppard & Young, 2004; Schmid, Hallschmid, & Schultes, 2015). Additionally, T2DM can result in peripheral neuropathy. This damage to nerves in the feet and legs is another cause of sleep disruption, due to the numbness, tingling, or burning pain (McDermott, Toelle, Rowbotham, Schaefer, & Dukes, 2006; Rosenstock, Tuchman, Lamoreaux, & Sharma, 2004; Vinik, Emir, Cheung, & Whalen, 2013). Thus T2DM can also contribute to sleep disturbances, which may, in turn, worsen the T2DM via the mechanisms described above.

Treatment Effects on Health

Despite the major health consequences of sleep loss and overly long sleep, treatment and management of inappropriate sleep duration in “an otherwise healthy” individual is rarely addressed by clinicians. There are no formal treatment guidelines for managing sleep loss. The most popular professional advice is to take a short nap (Veasey, Rosen, Barzansky, Rosen, & Owens, 2002) and to sleep longer on a regular basis. The former is not particularly efficacious for health, and the latter may be perceived as impractical advice. Furthermore, catching up on sleep during the weekends is not sufficient to return functioning to baseline levels (Klerman & Dijk, 2005; Szymczak, Jasinska, Pawlak, & Zwierzykowska, 1993).

While there are no evidence-based guidelines for managing sleep duration in healthy populations, health improvements seen with improved sleep duration have been investigated in two ways: longitudinal epidemiological studies and experimental studies that systematically restrict the sleep of individuals in a laboratory setting and then observe over a period of recovery. A recent longitudinal epidemiological study investigated how increasing sleep duration to the healthy range improved mortality risk (Ferrie et al., 2007). When regular sleep length increased from 5–6 to 7–8 hr over the 3-year follow-up period, there were decreases in risk of both cardiovascular and non-cardiovascular disease-related mortality. This has not been consistently replicated in other epidemiological studies though (Dew et al., 2003; Hublin et al., 2007), suggesting the relationships observed by between sleep duration and mortality may be mediated by other specific health improvements, which were not explicitly reported.

IMPAIRED GLUCOSE TOLERANCE

In the experimental literature cited above, studies indicate a return to sufficient sleep duration after short sleep improves glucose regulation. This has direct clinical implications for preventing T2DM. Clinically, T2DM has stages of progression. Impaired glucose regulation is an intermediate stage between normal glucose tolerance and T2DM. This altered metabolic state may be a stage where appropriate sleep duration can improve regulation and protect against T2DM development (Ip & Mokhlesi, 2007).

The protective mechanism of adequate sleep has been consistently demonstrated. Experimental periods of partial or total sleep deprivation in healthy human and animal models have shown consistent decreases in glucose metabolism and insulin response that reverse after recovery sleep. Sleep restricted to 4 hr per night for between four and seven nights resulted in a 16–29% reduction in insulin sensitivity, without a similar magnitude of insulin release to compensate. After five to seven nights of recovery sleep, levels of plasma glucose and insulin responses returned to baseline levels (Barf, Meerlo, & Scheurink, 2010; Broussard, Ehrmann, Van Cauter, Tasali, & Brady, 2012; Buxton et al., 2010; Spiegel et al., 1999). However, the extent of sleep required to reverse metabolic effects of chronic short sleep, and whether it is possible to reduce these to baseline levels, is still under investigation.

Of course, while these short-term experimental studies are promising, it may also be the case that T2DM risk can reach a threshold where adequate sleep no longer helps protect against development. One epidemiological study found that once T2DM risk developed, fixing sleep was not adequate to completely reverse risk. In a longitudinal study of 59,031 women, those who slept <6 hr per night in 1986 were found to be at higher risk of developing T2DM than those who sleep 7–8 hr per night, as assessed by validated diabetes symptom questionnaires. When assessed again in 2000, those who had increased their sleep habits to 7–8 hr per night were still at the same elevated risk of developing T2DM as those who were still restricting their sleep to <6 hr (Cespedes et al., 2016). In this case, the women may have passed the threshold of sleep's usefulness as a protective factor. However, the improvements seen in short-term studies indicate sleeping appropriate duration may lower risk for T2DM before its full development.

Insomnia

The current Diagnostic and Statistical Manual (DSM-5) defines insomnia disorder as a combination of dissatisfaction with sleep quality or quantity, characterized by (a) difficulty initiating sleep, maintaining sleep, or early-morning awakening with inability to return back to sleep and (b) a negative impairment in some aspect of daytime functioning, which the individual relates back to poor sleep (i.e., they believe the poor sleep causes or exacerbates the daytime impairment). The insomnia occurs on at least three nights per week for a minimum of 3 months, occurs despite adequate opportunity for sleep, and is not fully explained by another sleep–wake disorder or other mental or medical disorder, nor is it attributable to physiological effects of a substance (American Psychiatric Association [APA], 2013). It is important to note the concept of “secondary insomnia” is no longer considered valid. A diagnosis of insomnia disorder can occur in the context of any other physical or mental health condition and is considered a comorbid diagnosis, not a secondary diagnosis. This is because once insomnia becomes chronic (i.e., ≥ 3 months in duration), it typically needs specific and independent treatment and does not respond adequately to treating the “other” diagnosis.

Insomnia is one of the most common sleep disorders and is among the most prevalent of all mental health disorders, affecting 10–15% of adults worldwide (Bartlett et al., 2008; Ford & Kamerow, 1989; Morin, LeBlanc, et al., 2011; National Institutes of Health, 2005; Ohayon & Bader, 2010). Insomnia commonly presents with a range of psychiatric and medical comorbidities. As such, insomnia represents a huge health burden. In an epidemiological study of almost 15,000 individuals aged 15–100 years in the United Kingdom, Germany, Italy, and Portugal, only 2% of individuals meeting criteria for insomnia disorders had “pure” insomnia, without any current or past psychiatric history (Ohayon & Roth, 2003). In the literature, comorbid insomnia accounts for up to 90% of insomnia, leading to increased healthcare burden (Ford & Kamerow, 1989; Klink, Quan, Kaltenborn, & Lebowitz, 1992). A 2010 report estimated that in Australia, insomnia and attributable conditions cost the economy A\$10.9 billion (Deloitte Access Economics, 2011). This is minor in comparison with other places around the world. In the province of Québec alone, the total annual cost of insomnia and associated health concerns was estimated at C\$6.6 billion (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009).

Insomnia is characterized by chronicity. Approximately 80% of individuals with insomnia experience it for at least 1 year, and 40% for 5 years (Bixler, Kales, Soldatos, Kales, & Healey, 1979; Hohagen et al., 1993; Morin, LeBlanc, Daley, Gregoire, & Merette, 2006). Despite this, it is vastly undertreated. One estimate suggests only 13% of individuals who complain of insomnia symptoms seek professional help (Morin et al., 2006).

Insomnia and Health

Given the high rates of comorbidity and the chronic nature of the disorder, chronic insomnia is linked with substantial decreases in quality of life, including by measures of absenteeism, accidents, and decreased vitality and social functioning. Insomnia plays key roles in the course of many psychological problems, contributing to their development or relapse, and exacerbating symptoms. These disorders include anxiety, depression, bipolar disorder, posttraumatic stress disorder (PTSD), adjustment disorder, panic disorder, somatoform disorder, schizophrenia, neurocognitive disorders, eating disorders, and substance use disorders, and physical health problems including arthritis, cancer, hypertension, chronic pain, cardiovascular disease, and T2DM (APA, 2013; Buysse et al., 1994; Foley et al., 1995; Ohayon, Caulet, & Lemoine, 1998). In particular, depression is highly comorbid with insomnia. Depression itself carries a large economic burden, estimated to cost the Australian economy A\$12.6 billion per year and the US economy \$210.5 billion per year (Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015; LaMontagne, Sanderson, & Cocker, 2010). The two disorders have a bidirectional and complex relationship.

DEPRESSION

About three-quarters of all depressed patients complain of insomnia symptoms, and 41% report sufficient symptoms to warrant an insomnia diagnosis (Staner, 2010; Stewart et al., 2006). Until recently, when the two disorders were comorbid, insomnia was viewed as a symptom of depression and so was not considered a focus of treatment. More recent research indicates a more complex bidirectional relationship. While insomnia typically has its origins before depression develops, high levels of depressive symptoms are classified as a premorbid psychological vulnerability to insomnia (Ford & Kamerow, 1989; LeBlanc et al., 2009).

The Nord-Trøndelag Health Study (HUNT) is a large Norwegian population-based prospective study investigating this bidirectional relationship. A sample of 24,715 individuals completed questionnaires assessing whether they had depression and insomnia on 2 occasions, approximately 10 years apart. Participants with insomnia when first assessed were 6.2 times as likely to develop depression by follow-up assessment as those without insomnia. Equally, those with depression at first were 6.7 times more likely to develop insomnia by their follow-up assessment than those without depression (Sivertsen et al., 2012).

Insomnia impacts not only the development but also the course of depression once developed. It can perpetuate depression, exacerbate depression, and increase risk of its recurrence. In a multisite randomized control trial (RCT) of elderly depressed patients, participants were randomized to receive either specialist depression treatment or treatment as usual. After 3 months of therapy, those with persistent insomnia were 3.5 times more likely to remain depressed at 12 months, compared with those without insomnia, showing how insomnia perpetuates depression (Pigeon et al., 2008). Similarly, in a 9-week RCT where depressed participants were treated with either 3 mg fluoxetine or placebo, participants with higher levels of insomnia experienced significantly greater intensity of suicidal ideation, indicating insomnia exacerbates depressive symptoms (McCall et al., 2010). After an RCT treating depression with interpersonal psychotherapy and/or pharmacotherapy, residual sleep disturbance was the strongest independent predictor of depression recurrence (Perlis, Giles, Buysse, Tu, & Kupfer, 1997).

Treatment Effects on Health

CBTI is currently the gold standard insomnia intervention. It is highly effective and has been extensively researched: it has a 70–80% success rate in efficacy studies for chronic insomnia

(Irwin, Cole, & Nicassio, 2006; Morin, Culbert, & Schwartz, 1994). Efficacy remains even when insomnia is comorbid with a range of other medical and psychiatric disorders (Geiger-Brown et al., 2015; Haynes et al., 2016; Mundt et al., 2016; Talbot et al., 2014; Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015; Ye et al., 2015). Research consistently shows that CBTI is, at minimum, equally as effective as pharmacological treatment and has significantly longer duration of benefits after treatment ceases.

CBTI is a multimodal therapy, typically provided over a four- to eight-session course of treatment (Perlis, Benson-Jungquist, Smith, & Posner, 2005). It has two main foci: altering behaviors to improve sleep patterns (behavioral therapy) and altering the unhelpful and/or inaccurate thoughts, beliefs, and attitudes people hold regarding sleep (cognitive therapy). The ultimate goals of CBTI are to (a) consolidate sleep (behavioral), (b) break associations between sleep and anxiety and/or arousal (behavioral), and (c) to develop a more realistic picture of sleep and sleeping habits (cognitive). It is effective for reducing insomnia symptoms under many conditions, including when delivered in group settings, over the phone or internet, and in brief 1–2 session treatments, though different patient populations benefit most from different conditions (Cape, Leibowitz, Whittington, Espie, & Pilling, 2015; Ellis, Cushing, & Germain, 2015; Ho, Chung, Yeung, Ng, & Cheng, 2014; Norell-Clarke, Jansson-Fröjmark, Tillfors, Holländare, & Engström, 2015; Ye et al., 2015; Zachariae, Lyby, Ritterband, & O'Toole, 2016).

While experiencing insomnia, individuals report lower quality of life and many negative health outcomes (APA, 2013; Buysse et al., 1994; Foley et al., 1995; Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2010; Ohayon et al., 1998). Every health domain thus far studied improves significantly when insomnia is treated with CBTI (Manber et al., 2011; McCall et al., 2010). The direct impact on depression is discussed in detail.

DEPRESSION

While the relationship between insomnia and depression is bidirectional, when it comes to improving health, the effect is unidirectional: improving insomnia alleviates depression symptoms. An RCT of CBTI for comorbid insomnia and depression by Ashworth et al. (2015) found that depression scores dropped substantially more in the CBTI versus self-help control group. At 3-month follow-up, 61.1% of the CBTI group were in remission from both their insomnia and depression, compared with only 5.6% of the control group. These results are consistent within the broader literature: when patients are treated with CBTI, whether individually or in group format, both insomnia and depression symptoms improve, and remission rates for depression are higher in CBTI than in control groups (Bei, Ong, Rajaratnam, & Manber, 2015; Manber et al., 2011; Manber et al., 2008; Norell-Clarke et al., 2015; Trockel, Manber, Chang, Thurston, & Taylor, 2011).

Importantly, these therapeutic effects are not bidirectional. When depression is treated with CBT or with antidepressants, while depressive symptoms improve, sleep problems are not consistently alleviated (Carney, Segal, Edinger, & Krystal, 2007). In essence, once insomnia develops, it must be treated in its own right. This is also true for other mental health disorders, including PTSD, bipolar disorder, and substance use disorders (Belleville, Guay, & Marchand, 2011; Brower, Krentzman, & Robinson, 2011; St-Amand, Provencher, Bélanger, & Morin, 2013; Zayfert & De Viva, 2004), indicating the importance for treating insomnia symptoms independently and early. These findings also underscore the reason “secondary insomnia” is no longer considered a valid diagnosis. Even when another health condition is the precipitating event initially triggering the insomnia, once chronic, the insomnia has developed into its own disorder.

Sleep-Disordered Breathing

SDB refers to a range of sleep disorders characterized by insufficient or abnormal ventilation during sleep. Importantly, disordered breathing is specific to the sleep state, with respiration during wake being normal. The most common form of SDB is sleep apnea whereby the individual suffers from repetitive episodes of reductions (hypopnea) or cessation (apnea) in airflow during sleep. Sleep apnea is typically divided into two main forms: (a) OSA, where apneas/hypopneas occur due to complete or partial collapse of the upper airway, or (b) central sleep apnea (CSA), where apneas/hypopneas occur due to a reduction in brainstem respiratory motor output (i.e., no effort is made to breathe). These repeated respiratory events are associated with reductions in blood oxygen saturation (hypoxemia), rises in blood carbon dioxide (hypercapnia), and surges in sympathetic activation. Unfortunately, regular breathing usually only resumes upon arousal from sleep. Individuals with a SDB diagnosis stop breathing at least 5 times an hour, with 30 or more events per hour considered severe. The cumulative impact of repeated respiratory events (i.e., reduction/cessation of breathing followed by an awakening) is significant sleep fragmentation and reductions in both deep stage N3 sleep and REM sleep (Lamphere et al., 1989; Quan et al., 2011; Waldhorn et al., 1990).

Of the two types of SDB, OSA is by far the most common, with prevalence rates as high as 10% in adults between the ages of 30 and 70 (Peppard et al., 2013). There are a number of factors that put people at higher risk for developing OSA. The most common risk factor is obesity, specifically central obesity. In both sexes, OSA is more prevalent with increasing age (Young et al., 1993), reaching very high rates in patients over 65 years (Ancoli-Israel, Klauber, Kripke, Parker, & Cobarrubias, 1989). Males appear to have a higher rate of sleep apnea than women, with a ratio of 2:1–10:1, depending on the study (Strohl & Redline, 1996). In addition, any condition resulting in a smaller airway can predispose toward the development of OSA. Finally, several studies demonstrated a higher risk of OSA with a positive family history, and risk increases with the number of affected relatives (Redline et al., 1995). There are also several environmental factors increasing the risk of developing OSA. For instance, use of alcohol or sedative medication can contribute to development OSA through their relaxant effect on the upper airway muscles (Issa & Sullivan, 1982). A longitudinal epidemiological study showed that smokers are at an increased risk for developing OSA with current smokers at greater risk than nonsmokers and heavy smokers having the greatest risk (Wetter, Young, Bidwell, Badr, & Palta, 1994). Cessation of smoking eliminated the increased risk.

By comparison, CSA is much less prevalent; however it is commonly found in certain populations such as in preterm and term infants, in patients with heart failure (~30%) (Sin et al., 1999), and in most adults when ascending to high altitude (Burgess, Johnson, & Edwards, 2004) and can also be a side effect of opioids. Given that this review will largely focus on OSA, a more detailed description of CSA can be found in a recent review by Edwards, Sands, and Berger (2013).

Sleep-Disordered Breathing and Health

One of the most commonly reported symptoms of SDB is excessive daytime sleepiness (AASM, 2014), and this may explain the increased risk of motor vehicles accidents seen in this population (Terán-Santos, Jiménez-Gómez, & Cordero-Guevara, 1999; Young, Blustein, Finn, & Palta, 1997). However, the relationship between crash risk and measures of sleep apnea severity and subjective sleepiness have not always been consistently demonstrated (Ellen et al., 2006).

Sleep apnea has also been associated with a range of cognitive impairments, most particularly in attention (Ayalon, Ancoli-Israel, & Drummond, 2010; Bedard, Montplaisir, Malo, Richer, & Rouleau, 1993; Bedard, Montplaisir, Richer, Rouleau, & Malo, 1991; Canessa et al., 2011), executive function (Canessa et al., 2011; Naëgelé et al., 1998; Saunamäki, Himanen, Polo, & Jehkonen, 2009), and memory (Canessa et al., 2011; Daurat, Foret, Bret-Dibat, Fureix, & Tiberge, 2008; Salorio, White, Piccirillo, Duntley, & Uhles, 2002). A number of recent systematic reviews and meta-analyses have confirmed the consistency of these findings (Beebe, Groesz, Wells, Nichols, & McGee, 2003; Olaithe & Bucks, 2013; Saunamäki & Jehkonen, 2007; Wallace & Bucks, 2013). These impairments in cognition are also consistent with imaging data showing significant cerebral gray matter loss in OSA patients (Yaouhi et al., 2009). Nonetheless, cognitive deficits are often not as extreme as one might expect given the severity of sleep disruption and hypoxia, as well as gray matter abnormalities experienced, suggesting that some compensatory mechanisms may be at work in these individuals (Ayalon, Ancoli-Israel, Klemfuss, Shalauta, & Drummond, 2006; Castronovo et al., 2009). Based on a range of converging evidence from animal and human studies, it would appear that sleep fragmentation, intermittent hypoxia, or both play causal roles in the pathogenesis of these cognitive deficits (Beebe & Gozal, 2002; Knoepke & Aloia, 2009; Verstraeten, 2007).

CARDIOVASCULAR AND METABOLIC HEALTH

Population-based studies have demonstrated SDB to be associated with metabolic abnormalities including glucose intolerance and insulin resistance, with risk increasing with increasing hypoxemic and SDB severity indices (Punjabi et al., 2004). Furthermore SDB has been shown to be an independent risk factor for diabetes (Botros et al., 2009; Reichmuth, Austin, Skatrud, & Young, 2005; Wang, Bi, Zhang, & Pan, 2013).

Hypertension is extremely prevalent in SDB populations. Numerous data from animal models and cross-sectional studies have demonstrated significant associations between SDB and blood pressure (Brooks, Horner, Kozar, Render-Teixeira, & Phillipson, 1997; Fletcher, DeBehnke, Lovoi, & Gorin, 1985; Nieto et al., 2000; Stoohs et al., 1996). Prospective longitudinal studies have demonstrated patients with SDB have increased risk of developing hypertension, with increasing severity of SDB associated with increasing risk (O'Connor et al., 2009; Peppard, Young, Palta, & Skatrud, 2000). Although SDB has been shown to be an independent risk factor for hypertension, comorbid obesity has previously been shown to mediate some of this dose-response relationship (Nieto et al., 2000; O'Connor et al., 2009).

SDB has also been associated with a range of other cardiovascular morbidities including, but not limited to, atherosclerosis, atrial fibrillation, stroke, and heart failure (Gottlieb et al., 2010; Hoffstein & Mateika, 1994; Hung, Whitford, Parsons, & Hillman, 1990; Neilan et al., 2013; Redline et al., 2010; Shahar et al., 2001; Somers, Dyken, Clary, & Abboud, 1995; Wessendorf et al., 2000; Yaggi et al., 2005). Importantly, longitudinal studies have shown that untreated OSA patients are at increased risk of both fatal and nonfatal cardiovascular events compared with controls (Marin, Carrizo, Vicente, & Agusti, 2005), as well as an increased risk of all-cause mortality (Punjabi et al., 2009).

Treatment Effects on Health

The current gold standard treatment for OSA in adults is continuous positive airway pressure (CPAP) therapy, which works by producing a positive pressure in a patient's airway in order to "splint" the airway open such that obstruction is no longer possible. CPAP is exceedingly efficacious in reducing OSA severity and improving hypoxemic and sleep quality parameters

(Issa & Sullivan, 1986; Kakkar & Berry, 2007; Sullivan, Issa, Berthon-Jones, & Eves, 1981). However clinical effectiveness is mainly limited by its tolerability. Long-term usage studies suggest approximately half of patients continue to use therapy after 3 months. Approximately 30% of patients fail to even initiate treatment (Wolkove, Baltzan, Kamel, Dabrusin, & Palayew, 2008). The second major treatment modality for OSA is mandibular advancement splints (MAS) (also referred to as oral appliances). These devices reposition the tongue and protrude the mandible forward in order to tighten several soft tissue structures and so increase the overall size of the upper airway (Chan *et al.*, 2010). A variety of surgical procedures are also utilized for OSA treatment; however these are typically undertaken as a last resort option, often when patients are unable to adhere to these noninvasive treatments. The treatment success of these surgical procedures varies considerably in the literature (Caples *et al.*, 2010) although is generally considered to be quite low. Part of this variability is likely due to differences in the way treatment successes have been defined in such studies as well as differences in the way OSA severity/burden was assessed.

Although there is a high degree of variability between studies, meta-analyses show that treatment with CPAP can significantly reduce subjective sleepiness complaints and, to a lesser degree, reduce objective sleepiness parameters (Patel, White, Malhotra, Stanchina, & Ayas, 2003). These effects are typically stronger in patients with more severe SDB and those with a greater degree of baseline sleepiness. A reduction in motor vehicle crash risk as well as improved driving simulator performance has also been demonstrated after successful CPAP treatment (Antonopoulos, Sergentanis, Daskalopoulou, & Petridou, 2011; Tregear, Reston, Schoelles, & Phillips, 2010). While a number of studies have demonstrated the success of CPAP in normalizing various aspects of neurocognitive function (Borak, Cieřlicki, Koziej, Matuszewski, & Zieliński, 1996; Canessa *et al.*, 2011; Engleman, Martin, Deary, & Douglas, 1994), others have demonstrated enduring impairments in some cognitive domains after treatment (Bedard *et al.*, 1993; Ferini Strambi *et al.*, 2003; Kotterba *et al.*, 1998). Available meta-analyses suggest that deficits in attention are most effectively resolved by CPAP treatment (Kylstra, Aaronson, Hofman, & Schmand, 2013), whereas improvements in executive functions and memory are less consistently reported (Aloia, Arnedt, Davis, Riggs, & Byrd, 2004; Sánchez, Martínez, Miró, Bardwell, & Buena-Casal, 2009).

CARDIOVASCULAR AND METABOLIC HEALTH

In general there is a relative paucity of RCT examining the effect of treatment on the metabolic disturbances associated with OSA. As yet, meta-analytic evidence does not suggest that glucose metabolism or measures of insulin resistance improve with CPAP usage (Hecht, Möhler, & Meyer, 2011). Furthermore, a recent systematic review suggests that with the exception of a reduced sympathetic activity, there is little evidence that treatment of OSA is associated with an improvement metabolic and inflammatory indexes (Jullian-Desayes *et al.*, 2015).

In terms of the effect of treatment on cardiovascular sequelae, two meta-analyses have demonstrated that CPAP usage significantly reduces blood pressure. However, the effect size is small in comparison with antihypertensive medications (Fava *et al.*, 2014; Montesi, Edwards, Malhotra, & Bakker, 2012). Despite this, successful treatment with CPAP is associated with a reduced risk of fatal and nonfatal cardiovascular events (Doherty, Kiely, Swan, & McNicholas, 2005; Marin *et al.*, 2005). While the majority of the treatment data currently available applies to the use of CPAP, treatment data from patients using a MAS device generally demonstrates improvement in health outcomes equivalent to those observed with CPAP (Bratton, Gaisl, Wons, & Kohler, 2015; Phillips *et al.*, 2013).

Summary

This chapter has emphasized the critical importance of achieving healthy sleep for a diverse set of physical and mental health morbidities. The mechanisms linking sleep and health are often complex and bidirectional in nature; however treatment outcomes tend to be unidirectional. Insomnia and OSA can be highly effectively treated using psychological and medical treatments. These treatments then contribute to subsequent improvements in health problems. However, the unidirectionality of these improvements means that chronic sleep problems require direct treatment for improvement. This reflects the special role that sleep plays in health and the attention that needs to be paid to healthy sleep for overall physical and mental health.

Note

- 1 Experimentally induced short sleep duration is typically called “sleep restriction” and much of the literature cited here uses that term. For consistency, we will always use the term “short sleep” even when referring to experimentally induced short sleep.

References

- AASM. (2014). *International classification of sleep disorders* (3rd ed.). Darien, IL: Author.
- Aho, V., Ollila, H. M., Rantanen, V., Kronholm, E., Surakka, I., van Leeuwen, W. M. A., ... Porkka-Heiskanen, T. (2013). Partial sleep restriction activates immune response-related gene expression pathways: Experimental and epidemiological studies in humans. *PLoS One*, 8(10). doi:10.1371/journal.pone.0077184
- Åkerstedt, T., & Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience*, 52, 29–37.
- Aloia, M., Arnedt, J. T., Davis, J., Riggs, R., & Byrd, D. (2004). Neuropsychological sequelae of obstructive sleep apnea-hypopnea syndrome: A critical review. *Journal of the International Neuropsychological Society*, 10(5), 772–785.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorder* (5th ed.). Washington, DC: Author.
- Ancoli-Israel, S., Klauber, M. R., Kripke, D. F., Parker, L., & Cobarrubias, M. (1989). Sleep apnea in female patients in a nursing home. Increased risk of mortality. *Chest*, 96(5), 1054–1058.
- Ancoli-Israel, S., Martin, J. L., Blackwell, T., Buenaver, L., Liu, L., Meltzer, L. J., ... Taylor, D. J. (2015). The SBSM guide to actigraphy monitoring: Clinical and research applications. *Behavioral Sleep Medicine*, 13, S4–S38. doi:10.1080/15402002.2015.1046356
- Antonopoulos, C. N., Sargentanis, T. N., Daskalopoulou, S. S., & Petridou, E. T. (2011). Nasal continuous positive airway pressure (nCPAP) treatment for obstructive sleep apnea, road traffic accidents and driving simulator performance: A meta-analysis. *Sleep Medicine Reviews*, 15(5), 301–310. doi:10.1016/j.smrv.2010.10.002
- Arand, D., Bonnet, M., Hurwitz, T., Mitler, M., Rosa, R., & Sangal, R. B. (2005). The clinical use of the MSLT and MWT. *Sleep*, 28(1), 123–144.
- Ashworth, D. K., Sletten, T. L., Junge, M., Simpson, K., Clarke, D., Cunningham, D., & Rajaratnam, S. M. W. (2015). A randomized controlled trial of cognitive behavioral therapy for insomnia: An effective treatment for comorbid insomnia and depression. *Journal of Counseling Psychology*, 62(2), 115–123. doi:10.1037/cou0000059
- Ayalon, L., Ancoli-Israel, S., & Drummond, S. P. A. (2010). Obstructive sleep apnea and age: A double insult to brain function? *American Journal of Respiratory and Critical Care Medicine*, 182(3), 413–419.

- Ayalon, L., Ancoli-Israel, S., Klemfuss, Z., Shalauta, M. D., & Drummond, S. P. (2006). Increased brain activation during verbal learning in obstructive sleep apnea. *NeuroImage*, 31(4), 1817–1825. doi:10.1016/j.neuroimage.2006.02.042
- Banks, S., & Dinges, D. F. (2007). Behavioral and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine*, 3(5), 519–528.
- Barf, R. P., Meerlo, P., & Scheurink, A. J. W. (2010). Chronic sleep disturbance impairs glucose homeostasis in rats. *International Journal of Endocrinology*, 2010. doi:10.1155/2010/819414
- Bartlett, D. J., Marshall, N. S., Williams, A., & Grunstein, R. R. (2008). Sleep health New South Wales: Chronic sleep restriction and daytime sleepiness. *Internal Medicine Journal*, 38(1), 24–31. doi:10.1111/j.1445-5994.2007.01395.x
- Bedard, M. A., Montplaisir, J., Malo, J., Richer, F., & Rouleau, I. (1993). Persistent neuropsychological deficits and vigilance impairment in sleep apnea syndrome after treatment with continuous positive airways pressure (CPAP). *Journal of Clinical and Experimental Neuropsychology*, 15(2), 330–341.
- Bedard, M. A., Montplaisir, J., Richer, F., Rouleau, I., & Malo, J. (1991). Obstructive sleep apnea syndrome: Pathogenesis of neuropsychological deficits. *Journal of Clinical and Experimental Neuropsychology*, 13(6), 950–964.
- Beebe, D. W., & Gozal, D. (2002). Obstructive sleep apnea and the prefrontal cortex: Towards a comprehensive model linking nocturnal upper airway obstruction to daytime cognitive and behavioral deficits. *Journal of Sleep Research*, 11(1), 1–16.
- Beebe, D. W., Groesz, L., Wells, C., Nichols, A., & McGee, K. (2003). The neuropsychological effects of obstructive sleep apnea: A meta-analysis of norm-referenced and case-controlled data. *Sleep*, 26(3), 298–307.
- Bei, B., Ong, J. C., Rajaratnam, S. M. W., & Manber, R. (2015). Chronotype and improved sleep efficiency independently predict depressive symptom reduction after group cognitive behavioral therapy for insomnia. *Journal of Clinical Sleep Medicine*, 11(9), 1021–1027. doi:10.5664/jcsm.5018
- Belleville, G., Guay, S., & Marchand, A. (2011). Persistence of sleep disturbances following cognitive-behavior therapy for posttraumatic stress disorder. *Journal of Psychosomatic Research*, 70(4), 318–327. doi:10.1016/j.jpsychores.2010.09.022
- Bennett, L. S., Barbour, C., Langford, B., Stradling, J. R., & Davies, R. J. O. (1999). Health status in obstructive sleep apnea: Relationship with sleep fragmentation and daytime sleepiness, and effects of continuous positive airway pressure treatment. *American Journal of Respiratory and Critical Care Medicine*, 159(6), 1884–1890. doi:10.1164/ajrccm.159.6.9808107
- Berry, R. B., Brooks, R., Gamaldo, C. E., Harding, S., Marcus, C., & Vaughn, B. (2012). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications*. Darien, IL: American Academy of Sleep Medicine.
- Besedovsky, L., Lange, T., & Born, J. (2012). Sleep and immune function. *Pflügers Archiv European Journal of Physiology*, 463(1), 121–137. doi:10.1007/s00424-011-1044-0
- Bixler, E. O., Kales, A., Leo, L., & Slye, T. (1973). A comparison of subjective estimates and objective sleep laboratory findings in insomniac patients. *Sleep Research*, 2, 143.
- Bixler, E. O., Kales, A., Soldatos, C. R., Kales, J. D., & Healey, S. (1979). Prevalence of sleep disorders in the Los Angeles metropolitan area. *American Journal of Psychiatry*, 136, 1257–1262. doi:10.1176/ajp.136.10.1257
- Borak, J., Cieśllicki, J. K., Koziej, M., Matuszewski, A., & Zieliński, J. (1996). Effects of CPAP treatment on psychological status in patients with severe obstructive sleep apnoea. *Journal of Sleep Research*, 5(2), 123–127.
- Borbély, A. A. (1982). A two process model of sleep regulation. *Human Neurobiology*, 1(3), 195–204.
- Botros, N., Concato, J., Mohsenin, V., Selim, B., Doctor, K., & Yaggi, H. K. (2009). Obstructive sleep apnea as a risk factor for type 2 diabetes. *American Journal of Medicine*, 122(12), 1122–1127. doi:10.1016/j.amjmed.2009.04.026
- Bratton, D. J., Gaisl, T., Wons, A. M., & Kohler, M. (2015). CPAP vs mandibular advancement devices and blood pressure in patients with obstructive sleep apnea a systematic review and meta-analysis.

- JAMA: The Journal of the American Medical Association*, 314(21), 2280–2293. doi:10.1001/jama.2015.16303
- Briançon-Marjollet, A., Weiszenstein, M., Henri, M., Thomas, A., Godin-Ribuot, D., & Polak, J. (2015). The impact of sleep disorders on glucose metabolism: Endocrine and molecular mechanisms. *Diabetology and Metabolic Syndrome*, 7(1). doi:10.1186/s13098-015-0018-3
- Brooks, D., Horner, R. L., Kozar, L. F., Render-Teixeira, C. L., & Phillipson, E. A. (1997). Obstructive sleep apnea as a cause of systemic hypertension. Evidence from a canine model. *Journal of Clinical Investigation*, 99(1), 106–109.
- Broussard, J. L., Ehrmann, D. A., Van Cauter, E., Tasali, E., & Brady, M. J. (2012). Impaired insulin signaling in human adipocytes after experimental sleep restriction: A randomized, crossover study. *Annals of Internal Medicine*, 157(8), 549–557. doi:10.7326/0003-4819-157-8-201210160-00005
- Brower, K. J., Krentzman, A., & Robinson, E. A. R. (2011). Persistent insomnia, abstinence, and moderate drinking in alcohol-dependent individuals. *American Journal on Addictions*, 20(5), 435–440. doi:10.1111/j.1521-0391.2011.00152.x
- Burgess, K. R., Johnson, P. L., & Edwards, N. (2004). Central and obstructive sleep apnoea during ascent to high altitude. *Respirology*, 9(2), 222–229. doi:10.1111/j.1440-1843.2004.00576.x
- Buxton, O. M., Cain, S. W., O'Connor, S. P., Porter, J. H., Duffy, J. F., Wang, W., ... Shea, S. A. (2012). Adverse metabolic consequences in humans of prolonged sleep restriction combined with circadian disruption. *Science Translational Medicine*, 4(129), 129ra43. doi:10.1126/scitranslmed.3003200
- Buxton, O. M., Pavlova, M., Reid, E. W., Wang, W., Simonson, D. C., & Adler, G. K. (2010). Sleep restriction for 1 week reduces insulin sensitivity in healthy men. *Diabetes*, 59(9), 2126–2133. doi:10.2337/db09-0699
- Buyse, D. J., Reynolds, C. F., 3rd, Kupfer, D. J., Thorpy, M. J., Bixler, E., Manfredi, R., ... Mesiano, D. (1994). Clinical diagnoses in 216 insomnia patients using the International Classification of Sleep Disorders (ICSD), DSM-IV and ICD-10 categories: A report from the APA/NIMH DSM-IV field trial. *Sleep*, 17(7), 630–637.
- Buyse, D. J., Reynolds, C. F., 3rd, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193–213. doi:10.1016/0165-1781(89)90047-4
- Cai, H., Shu, X. O., Xiang, Y. B., Yang, G., Li, H., Ji, B. T., ... Zheng, W. (2015). Sleep duration and mortality: A prospective study of 113,138 middle-aged and elderly Chinese men and women. *Sleep*, 38(4), 529–536. doi:10.5665/sleep.4564
- Canessa, N., Castronovo, V., Cappa, S. F., Aloia, M. S., Marelli, S., Falini, A., ... Ferini-Strambi, L. (2011). Obstructive sleep apnea: Brain structural changes and neurocognitive function before and after treatment. *American Journal of Respiratory and Critical Care Medicine*, 183(10), 1419–1426.
- Cape, J., Leibowitz, J., Whittington, C., Espie, C. A., & Pilling, S. (2015). Group cognitive behavioural treatment for insomnia in primary care: A randomized controlled trial. *Psychological Medicine*, 1–11. doi:10.1017/S0033291715002561
- Caples, S. M., Rowley, J. A., Prinsell, J. R., Pallanch, J. F., Elamin, M. B., Katz, S. G., ... Tracy, S. L. (2010). Surgical modifications of the upper airway for obstructive sleep apnea in adults: A systematic review and meta-analysis. *Sleep*, 33(10), 1396–1407.
- Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The consensus sleep diary: Standardizing prospective sleep self-monitoring. *Sleep*, 35(2), 287–302. doi:10.5665/sleep.1642
- Carney, C. E., Segal, Z. V., Edinger, J. D., & Krystal, A. D. (2007). A comparison of rates of residual insomnia symptoms following pharmacotherapy or cognitive-behavioral therapy for major depressive disorder. *Journal of Clinical Psychiatry*, 68(2), 254–260.
- Carskadon, M. A., & Dement, W. C. (1982). The multiple sleep latency test: What does it measure? *Sleep*, 5(Suppl. 2), S67–S72.
- Carskadon, M. A., & Dement, W. C. (2011). Chapter 2—Normal human sleep: An overview. In M. H. K. R. C. Dement (Ed.), *Principles and practice of sleep medicine* (5th ed., pp. 16–26). Philadelphia, PA: W.B. Saunders.

- Carskadon, M. A., Dement, W. C., Mitler, M. M., Roth, T., Westbrook, P. R., & Keenan, S. (1987). Guidelines for the multiple sleep latency test (MSLT): A standard measure of sleepiness. *Sleep*, 9(4), 519–524.
- Castronovo, V., Canessa, N., Strambi, L. F., Aloia, M. S., Consonni, M., Marelli, S., ... Cappa, S. F. (2009). Brain activation changes before and after PAP treatment in obstructive sleep apnea. *Sleep*, 32(9), 1161–1172.
- Cespedes, E. M., Bhupathiraju, S. N., Li, Y., Rosner, B., Redline, S., & Hu, F. B. (2016). Long-term changes in sleep duration, energy balance and risk of type 2 diabetes. *Diabetologia*, 59(1), 101–109. doi:10.1007/s00125-015-3775-5
- Chan, A. S. L., Sutherland, K., Schwab, R. J., Zeng, B., Petocz, P., Lee, R. W. W., ... Cistulli, P. A. (2010). The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. *Thorax*, 65(8), 726–732. doi:10.1136/thx.2009.131094
- Chung, F., Yegneswaran, B., Liao, P., Chung, S. A., Vairavanathan, S., Islam, S., ... Shapiro, C. M. (2008). STOP questionnaire: A tool to screen patients for obstructive sleep apnea. *Anesthesiology*, 108(5), 812–821. doi:10.1097/ALN.0b013e31816d83e4
- Czisch, M., Wehrle, R., Harsay, H. A., Wetter, T. C., Holsboer, F., Sämann, P. G., & Drummond, S. P. A. (2012). On the need of objective vigilance monitoring: Effects of sleep loss on target detection and task-negative activity using combined EEG/fMRI. *Frontiers in Neurology*, APR. doi:10.3389/fneur.2012.00067
- Daley, M., Morin, C. M., LeBlanc, M., Gregoire, J. P., & Savard, J. (2009). The economic burden of insomnia: Direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep*, 32(1), 55–64.
- Daurat, A., Foret, J., Bret-Dibat, J. L., Fureix, C., & Tiberge, M. (2008). Spatial and temporal memories are affected by sleep fragmentation in obstructive sleep apnea syndrome. *Journal of Clinical and Experimental Neuropsychology*, 30(1), 91–101.
- Deloitte Access Economics. (2011). Re-awakening Australia: Economic cost of sleep disorders in Australia: Sleep Health Foundation. Retrieved from <https://www.sleephealthfoundation.org.au/pdfs/news/Reawakening%20Australia.pdf>
- Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., Houck, P. R., ... Reynolds, C. F., 3rd. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine*, 65(1), 63–73.
- Dickelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews Neuroscience*, 11(2), 114–126. doi:10.1038/nrn2762
- Doherty, L. S., Kiely, J. L., Swan, V., & McNicholas, W. T. (2005). Long-term effects of nasal continuous positive airway pressure therapy on cardiovascular outcomes in sleep apnea syndrome. *Chest*, 127(6), 2076–2084. doi:10.1378/chest.127.6.2076
- Donga, E., Van Dijk, M., Van Dijk, J. G., Biermasz, N. R., Lammers, G. J., Van Kralingen, K. W., ... Romijn, J. A. (2010). A single night of partial sleep deprivation induces insulin resistance in multiple metabolic pathways in healthy subjects. *Journal of Clinical Endocrinology and Metabolism*, 95(6), 2963–2968. doi:10.1210/jc.2009-2430
- Drummond, S. P., Anderson, D. E., Straus, L. D., Vogel, E. K., & Perez, V. B. (2012). The effects of two types of sleep deprivation on visual working memory capacity and filtering efficiency. *PLoS One*, 7(4), e35653. doi:10.1371/journal.pone.0035653
- Edwards, B. A., Sands, S. A., & Berger, P. J. (2013). Postnatal maturation of breathing stability and loop gain: The role of carotid chemoreceptor development. *Respiratory Physiology & Neurobiology*, 185(1), 144–155. doi:10.1016/j.resp.2012.06.003
- Ellen, R. L. B., Marshall, S. C., Palayew, M., Molnar, F. J., Wilson, K. G., & Man-Son-Hing, M. (2006). Systematic review of motor vehicle crash risk in persons with sleep apnea. *Journal of Clinical Sleep Medicine*, 2(2), 193–200.
- Ellis, J. G., Cushing, T., & Germain, A. (2015). Treating acute insomnia: A randomized controlled trial of a “single-shot” of cognitive behavioral therapy for insomnia. *Sleep*, 38(6), 971–978. doi:10.5665/sleep.4752

- Engleman, H. M., Martin, S. E., Deary, I. J., & Douglas, N. J. (1994). Effect of continuous positive airway pressure treatment on daytime function in sleep apnoea/hypopnoea syndrome. *Lancet*, 343(8897), 572–575.
- Fava, C., Dorigoni, S., Dalle Vedove, F., Danese, E., Montagnana, M., Guidi, G. C., ... Minuz, P. (2014). Effect of CPAP on blood pressure in patients with OSA/hypopnea: A systematic review and meta-analysis. *Chest*, 145(4), 762–771. doi:10.1378/chest.13-1115
- Ferini Strambi, L., Baietto, C., Di Gioia, M. R., Castaldi, P., Castronovo, C., Zucconi, M., & Cappa, S. F. (2003). Cognitive dysfunction in patients with obstructive sleep apnea (OSA): Partial reversibility after continuous positive airway pressure (CPAP). *Brain Research Bulletin*, 61(1), 87–92.
- Ferrie, J. E., Shipley, M. J., Cappuccio, F. P., Brunner, E., Miller, M. A., Kumari, M., & Marmot, M. G. (2007). A prospective study of change in sleep duration: Associations with mortality in the Whitehall II cohort. *Sleep*, 30(12), 1659–1666.
- Fischer, S., Diekelmann, S., & Born, J. (2011). Sleep's role in the processing of unwanted memories. *Journal of Sleep Research*, 20(2), 267–274. doi:10.1111/j.1365-2869.2010.00881.x
- Fletcher, E. C., DeBehnke, R. D., Lovo, M. S., & Gorin, A. B. (1985). Undiagnosed sleep apnea in patients with essential hypertension. *Annals of Internal Medicine*, 103(2), 190–195.
- Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1995). Sleep complaints among elderly persons: An epidemiologic study of three communities. *Sleep*, 18(6), 425–432.
- Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders: An opportunity for prevention? *JAMA: The Journal of the American Medical Association*, 262(11), 1479–1484. doi:10.1001/jama.1989.03430110069030
- Fortier-Brochu, É., Beaulieu-Bonneau, S., Ivers, H., & Morin, C. M. (2010). Relations between sleep, fatigue, and health-related quality of life in individuals with insomnia. *Journal of Psychosomatic Research*, 69(5), 475–483. doi:10.1016/j.jpsychores.2010.05.005
- Gale, J. E., Cox, H. I., Qian, J., Block, G. D., Colwell, C. S., & Matveyenko, A. V. (2011). Disruption of circadian rhythms accelerates development of diabetes through pancreatic beta-cell loss and dysfunction. *Journal of Biological Rhythms*, 26(5), 423–433.
- Geiger-Brown, J. M., Rogers, V. E., Liu, W., Ludeman, E. M., Downton, K. D., & Diaz-Abad, M. (2015). Cognitive behavioral therapy in persons with comorbid insomnia: A meta-analysis. *Sleep Medicine Reviews*, 23, 54–67. doi:10.1016/j.smrv.2014.11.007
- Geyer, J. D., Talathi, S., & Carney, P. R. (2009). Introduction to sleep and polysomnography. In *Reading EEGs: A practical approach* (pp. 258–280). Philadelphia, PA: Lippincott Williams & Wilkins.
- Gottlieb, D. J., Yenokyan, G., Newman, A. B., O'Connor, G. T., Punjabi, N. M., Quan, S. F., ... Shahar, E. (2010). Prospective study of obstructive sleep apnea and incident coronary heart disease and heart failure: The sleep heart health study. *Circulation*, 122(4), 352–360. doi:10.1161/CIRCULATIONAHA.109.901801
- Grandner, M. A., & Drummond, S. P. (2007). Who are the long sleepers? Towards an understanding of the mortality relationship. *Sleep Medicine Reviews*, 11(5), 341–360. doi:10.1016/j.smrv.2007.03.010
- Greenberg, P. E., Fournier, A. A., Sisitsky, T., Pike, C. T., & Kessler, R. C. (2015). The economic burden of adults with major depressive disorder in the United States (2005 and 2010). *Journal of Clinical Psychiatry*, 76(2), 155–162. doi:10.4088/JCP.14m09298
- Hammond, E. C. (1964). Some preliminary findings on physical complaints from a prospective study of 1,064,004 men and women. *American Journal of Public Health and the Nation's Health*, 54, 11–23.
- Haynes, P. L., Kelly, M., Warner, L., Quan, S. F., Krakow, B., & Bootzin, R. R. (2016). Cognitive Behavioral Social Rhythm Group Therapy for Veterans with posttraumatic stress disorder, depression, and sleep disturbance: Results from an open trial. *Journal of Affective Disorders*, 192, 234–243. doi:10.1016/j.jad.2015.12.012
- Hecht, L., Möhler, R., & Meyer, G. (2011). Effects of CPAP-respiration on markers of glucose metabolism in patients with obstructive sleep apnoea syndrome: A systematic review and meta-analysis. *German Medical Science: GMS e-Journal*, 9. Doc20. https://doi.org/10.3205/000143

- Herzog, N., Jauch-Chara, K., Hyzy, F., Richter, A., Friedrich, A., Benedict, C., & Oltmanns, K. M. (2013). Selective slow wave sleep but not rapid eye movement sleep suppression impairs morning glucose tolerance in healthy men. *Psychoneuroendocrinology*, 38(10), 2075–2082. doi:10.1016/j.psyneuen.2013.03.018
- Ho, F. Y. Y., Chung, K. F., Yeung, W. F., Ng, T. H. Y., & Cheng, S. K. W. (2014). Weekly brief phone support in self-help cognitive behavioral therapy for insomnia disorder: Relevance to adherence and efficacy. *Behaviour Research and Therapy*, 63, 147–156. doi:10.1016/j.brat.2014.10.002
- Hoffstein, V., & Mateika, S. (1994). Cardiac arrhythmias, snoring, and sleep apnea. *Chest*, 106(2), 466–471.
- Hohagen, F., Rink, K., Kappler, C., Schramm, E., Riemann, D., Weyerer, S., & Berger, M. (1993). Prevalence and treatment of insomnia in general practice. *European Archives of Psychiatry and Clinical Neuroscience*, 242, 329–336.
- Horne, J. A., & Ostberg, O. (1976). A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *International Journal of Chronobiology*, 4, 97–110.
- Hublin, C., Partinen, M., Koskenvuo, M., & Kaprio, J. (2007). Sleep and mortality: A population-based 22-year follow-up study. *Sleep*, 30(10), 1245–1253.
- Hung, J., Whitford, E. G., Parsons, R. W., & Hillman, D. R. (1990). Association of sleep apnoea with myocardial infarction in men. *Lancet*, 336(8710), 261–264.
- Ip, M., & Mokhlesi, B. (2007). Sleep and glucose intolerance/diabetes mellitus. *Sleep Medicine Clinics*, 2(1), 19–29. doi:10.1016/j.jsmc.2006.12.002
- Irwin, M. R., Cole, J. C., & Nicassio, P. M. (2006). Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. *Health Psychology*, 25(1), 3–14. doi:10.1037/0278-6133.25.1.3
- Issa, F. G., & Sullivan, C. E. (1982). Alcohol, snoring and sleep apnoea. *Journal of Neurology, Neurosurgery, and Psychiatry*, 45(4), 353–359.
- Issa, F. G., & Sullivan, C. E. (1986). The immediate effects of nasal continuous positive airway pressure treatment on sleep pattern in patients with obstructive sleep apnea syndrome. *Electroencephalography and Clinical Neurophysiology*, 63(1), 10–17. [http://dx.doi.org/10.1016/0013-4694\(86\)90056-8](http://dx.doi.org/10.1016/0013-4694(86)90056-8)
- Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep*, 14(6), 540–545.
- Jullian-Desayes, I., Joyeux-Faure, M., Tamisier, R., Launois, S., Borel, A. L., Levy, P., & Pepin, J. L. (2015). Impact of obstructive sleep apnea treatment by continuous positive airway pressure on cardiometabolic biomarkers: A systematic review from sham CPAP randomized controlled trials. *Sleep Medicine Reviews*, 21, 23–38. doi:10.1016/j.smr.2014.07.004
- Kakkar, R. K., & Berry, R. B. (2007). Positive airway pressure treatment for obstructive sleep apnea. *Chest*, 132(3), 1057–1072.
- Karimi, M., Hedner, J., Häbel, H., Nerman, O., & Grote, L. (2015). Sleep apnea related risk of motor vehicle accidents is reduced by continuous positive airway pressure: Swedish traffic accident registry data. *Sleep*, 38(3), 341–349. doi:10.5665/sleep.4486
- Kawada, T. (2013). Sleep parameters from actigraphy and sleep diary: Is the agreement important for sleep study? *Sleep Medicine*, 14(3), 298–299. doi:10.1016/j.sleep.2012.09.016
- Killgore, W. D., Balkin, T. J., & Wesensten, N. J. (2006). Impaired decision making following 49 h of sleep deprivation. *Journal of Sleep Research*, 15(1), 7–13.
- Killgore, W. D., Grugle, N. L., & Balkin, T. J. (2012). Gambling when sleep deprived: Don't bet on stimulants. *Chronobiology International*, 29(1), 43–54. doi:10.3109/07420528.2011.635230
- Klerman, E. B., & Dijk, D. J. (2005). Interindividual variation in sleep duration and its association with sleep debt in young adults. *Sleep*, 28(10), 1253–1259.
- Klink, M. E., Quan, S. F., Kaltenborn, W. T., & Lebowitz, M. D. (1992). Risk factors associated with complaints of insomnia in a general adult population: Influence of previous complaints of insomnia. *Archives of Internal Medicine*, 152(8), 1634–1637. doi:10.1001/archinte.1992.00400200070012
- Knoepke, C., & Aloia, M. (2009). Proposed mechanisms of cognitive dysfunction in obstructive sleep apnea. *Primary Psychiatry*, 16(10), 51–56.

- Knutson, K. L., Spiegel, K., Penev, P., & Van Cauter, E. (2007). The metabolic consequences of sleep deprivation. *Sleep Medicine Reviews*, 11(3), 163–178. <http://dx.doi.org/10.1016/j.smrv.2007.01.002>
- Kojima, M., Wakai, K., Kawamura, T., Tamakoshi, A., Aoki, R., Lin, Y., ... Ohno, Y. (2000). Sleep patterns and total mortality: A 12-year follow-up study in Japan. *Journal of Epidemiology / Japan Epidemiological Association*, 10(2), 87–93.
- Kotterba, S., Rasche, K., Widdig, W., Duscha, C., Blombach, S., Schultze-Werninghaus, G., & Malin, J. P. (1998). Neuropsychological investigations and event-related potentials in obstructive sleep apnea syndrome before and during CPAP-therapy. *Journal of the Neurological Sciences*, 159(1), 45–50.
- Kripke, D. F., Garfinkel, L., Wingard, D. L., Klauber, M. R., & Marler, M. R. (2002). Mortality associated with sleep duration and insomnia. *Archives of General Psychiatry*, 59(2), 131–136.
- Kripke, D. F., Simons, R. N., Garfinkel, L., & Hammond, E. (1979). Short and long sleep and sleeping pills: Is increased mortality associated? *Archives of General Psychiatry*, 36(1), 103–116. doi:10.1001/archpsyc.1979.01780010109014
- Kylstra, W. A., Aaronson, J. A., Hofman, W. F., & Schmand, B. A. (2013). Neuropsychological functioning after CPAP treatment in obstructive sleep apnea: A meta-analysis. *Sleep Medicine Reviews*, 17(5), 341–347.
- LaMontagne, A., Sanderson, K., & Cocker, F. (2010). *Estimating the economic benefits of eliminating job strain as a risk factor for depression*. Australia: VicHealth.
- Lamphere, J., Roehrs, T., Wittig, R., Zorick, F., Conway, W. A., & Roth, T. (1989). Recovery of alertness after CPAP in apnea. *Chest*, 96(6), 1364–1367.
- LeBlanc, M., Mérette, C., Savard, J., Ivers, H., Baillargeon, L., & Morin, C. M. (2009). Incidence and risk factors of insomnia in a population-based sample. *Sleep*, 32(8), 1027–1037.
- Lim, J., & Dinges, D. F. (2008). Sleep deprivation and vigilant attention. *Annals of the New York Academy of Sciences*, 1129, 305–322. doi:10.1196/annals.1417.002
- Littner, M. R., Kushida, C., Wise, M., Davila, D. G., Morgenthaler, T., Lee-Chiong, T., ... Kramer, M. (2005). Practice parameters for clinical use of the multiple sleep latency test and the maintenance of wakefulness test. *Sleep*, 28(1), 113–121.
- Lopes, L. A., Lins, C. D. M. M., Adeodato, V. G., Quental, D. P., De Bruin, P. F. C., Montenegro, R. M., Jr., & De Bruin, V. M. S. (2005). Restless legs syndrome and quality of sleep in type 2 diabetes. *Diabetes Care*, 28(11), 2633–2636. doi:10.2337/diacare.28.11.2633
- Luckhaupt, S. E. (2012). Short sleep duration among workers—United States, 2010. *Morbidity and Mortality Weekly Report*, 61(16), 281–285.
- Manber, R., Bernert, R. A., Suh, S., Nowakowski, S., Siebern, A. T., & Ong, J. C. (2011). CBT for insomnia in patients with high and low depressive symptom severity: Adherence and clinical outcomes. *Journal of Clinical Sleep Medicine*, 7(6), 645–652. doi:10.5664/jcsm.1472
- Manber, R., Edinger, J. D., Gress, J. L., San Pedro-Salcedo, M. G., Kuo, T. F., & Kalista, T. (2008). Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep*, 31(4), 489–495.
- Marin, J. M., Carrizo, S. J., Vicente, E., & Agustí, A. G. N. (2005). Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: An observational study. *Lancet*, 365(9464), 1046–1053. doi:10.1016/s0140-6736(05)71141-7
- McCall, W. V., Blocker, J. N., D'Agostino, R., Kimball, J., Boggs, N., Lasater, B., & Rosenquist, P. B. (2010). Insomnia severity is an indicator of suicidal ideation during a depression clinical trial. *Sleep Medicine*, 11(9), 822–827. doi:10.1016/j.sleep.2010.04.004
- McCarley, R. W. (2007). Neurobiology of REM and NREM sleep. *Sleep Medicine*, 8(4), 302–330. doi:10.1016/j.sleep.2007.03.005
- McDermott, A. M., Toelle, T. R., Rowbotham, D. J., Schaefer, C. P., & Dukes, E. M. (2006). The burden of neuropathic pain: Results from a cross-sectional survey. *European Journal of Pain*, 10(2), 127–135. doi:10.1016/j.ejpain.2005.01.014
- Montesi, S. B., Edwards, B. A., Malhotra, A., & Bakker, J. P. (2012). Effect of continuous positive airway pressure treatment on blood pressure: A systematic review and meta-analysis of randomized controlled trials. *Journal of Clinical Sleep Medicine*, 8(5), 587–596. doi:10.5664/jcsm.2170

- Morin, C. M., Belleville, G., Belanger, L., & Ivers, H. (2011). The Insomnia Severity Index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, 34(5), 601–608.
- Morin, C. M., Culbert, J. P., & Schwartz, S. M. (1994). Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. *The American Journal of Psychiatry*, 151(8), 1172–1180.
- Morin, C. M., & Espie, C. A. (2003). *Insomnia: A clinical guide to assessment and treatment*. New York, NY: Kluwer Academic/Plenum Publishers.
- Morin, C. M., LeBlanc, M., Bélanger, L., Ivers, H., Mérette, C., & Savard, J. (2011). Prevalence of insomnia and its treatment in Canada. *Canadian Journal of Psychiatry*, 56(9), 540–548.
- Morin, C. M., LeBlanc, M., Daley, M., Gregoire, J. P., & Merette, C. (2006). Epidemiology of insomnia: Prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. *Sleep Medicine*, 7(2), 123–130. doi:10.1016/j.sleep.2005.08.008
- Mundt, J. M., Crew, E. C., Krietsch, K., Roth, A. J., Vathauer, K., Robinson, M. E., ... McCrae, C. S. (2016). Measuring treatment outcomes in comorbid insomnia and fibromyalgia: Concordance of subjective and objective assessments. *Journal of Clinical Sleep Medicine*, 12(2), 215–223. doi:10.5664/jcsn.5488
- Naëgelé, B., Pepin, J. L., Levy, P., Bonnet, C., Pellat, J., & Feuerstein, C. (1998). Cognitive executive dysfunction in patients with obstructive sleep apnea syndrome (OSAS) after CPAP treatment. *Sleep*, 21(4), 392–397.
- National Institutes of Health. (2005). National Institutes of Health State of the Science Conference statement on manifestations and management of chronic insomnia in adults, June 13–15, 2005. *Sleep*, 28(9), 1049.
- Nedeltcheva, A. V., Kilkus, J. M., Imperial, J., Kasza, K., Schoeller, D. A., & Penev, P. D. (2009). Sleep curtailment is accompanied by increased intake of calories from snacks. *American Journal of Clinical Nutrition*, 89(1), 126–133. doi:10.3945/ajcn.2008.26574
- Neilan, T. G., Farhad, H., Dodson, J. A., Shah, R. V., Abbasi, S. A., Bakker, J. P., ... Kwong, R. Y. (2013). Effect of sleep apnea and continuous positive airway pressure on cardiac structure and recurrence of atrial fibrillation. *Journal of the American Heart Association*, 2(6), e000421. doi:10.1161/JAHA.113.000421
- Nieto, F., Young, T. B., Lind, B. K., Shahar, E., Samet, J. M., Redline, S., ... Pickering, T. G. (2000). Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. *JAMA: The Journal of the American Medical Association*, 283, 1829–1836.
- Norell-Clarke, A., Jansson-Fröjmark, M., Tillfors, M., Holländare, F., & Engström, I. (2015). Group cognitive behavioural therapy for insomnia: Effects on sleep and depressive symptomatology in a sample with comorbidity. *Behaviour Research and Therapy*, 74, 80–93. doi:10.1016/j.brat.2015.09.005
- O'Connor, G. T., Caffo, B., Newman, A. B., Quan, S. F., Rapoport, D. M., Redline, S., ... Shahar, E. (2009). Prospective study of sleep-disordered breathing and hypertension: The sleep heart health study. *American Journal of Respiratory and Critical Care Medicine*, 179(12), 1159–1164. doi:10.1164/rccm.200712-1809OC
- Ohayon, M. M. (2004). Interactions between sleep normative data and sociocultural characteristics in the elderly. *Journal of Psychosomatic Research*, 56(5), 479–486. doi:10.1016/j.psychores.2004.04.365
- Ohayon, M. M., & Bader, G. (2010). Prevalence and correlates of insomnia in the Swedish population aged 19–75 years. *Sleep Medicine*, 11(10), 980–986. doi:10.1016/j.sleep.2010.07.012
- Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep*, 27(7), 1255–1273.
- Ohayon, M. M., Caulet, M., & Lemoine, P. (1998). Comorbidity of mental and insomnia disorders in the general population. *Comprehensive Psychiatry*, 39(4), 185–197. doi:10.1016/S0010-440X(98)90059-1
- Ohayon, M. M., & Roth, T. (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research*, 37(1), 9–15.
- Olaith, M., & Bucks, R. S. (2013). Executive dysfunction in OSA before and after treatment: A meta-analysis. *Sleep*, 36(9), 1297–1305. doi:10.5665/sleep.2950

- Patel, S. R., Ayas, N. T., Malhotra, M. R., White, D. P., Schernhammer, E. S., Speizer, F. E., ... Hu, F. B. (2004). A prospective study of sleep duration and mortality risk in women. *Sleep*, 27(3), 440–444.
- Patel, S. R., Malhotra, A., Gottlieb, D. J., White, D. P., & Hu, F. B. (2006). Correlates of long sleep duration. *Sleep*, 29(7), 881–889.
- Patel, S. R., White, D. P., Malhotra, A., Stanchina, M. L., & Ayas, N. T. (2003). Continuous positive airway pressure therapy for treating sleepiness in a diverse population with obstructive sleep apnea results of a meta-analysis. *Archives of Internal Medicine*, 163(5), 565–571. doi:10.1001/archinte.163.5.565
- Peppard, P. E., & Young, T. (2004). Exercise and sleep-disordered breathing: An association independent of body habitus. *Sleep*, 27(3), 480–484.
- Peppard, P. E., Young, T., Barnett, J. H., Palta, M., Hagen, E. W., & Hla, K. M. (2013). Increased prevalence of sleep-disordered breathing in adults. *American Journal of Epidemiology*, 177(9), 1006–1014.
- Peppard, P. E., Young, T., Palta, M., & Skatrud, J. (2000). Prospective study of the association between sleep-disordered breathing and hypertension. *New England Journal of Medicine*, 342(19), 1378–1384. doi:10.1056/nejm200005113421901
- Perlis, M. L., Benson-Jungquist, C., Smith, M. T., & Posner, D. A. (2005). *Cognitive behavioral treatment of insomnia: A session-by-session guide*. New York: Springer.
- Perlis, M. L., Giles, D. E., Buysse, D. J., Tu, X., & Kupfer, D. J. (1997). Self-reported sleep disturbance as a prodromal symptom in recurrent depression. *Journal of Affective Disorders*, 42(2–3), 209–212.
- Phillips, C. L., Grunstein, R. R., Darendeliler, M. A., Mihailidou, A. S., Srinivasan, V. K., Yee, B. J., ... Cistulli, P. A. (2013). Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea: A randomized controlled trial. *American Journal of Respiratory and Critical Care Medicine*, 187(8), 879–887. doi:10.1164/rccm.201212-2223OC
- Pigeon, W. R., Hegel, M., Unützer, J., Fan, M. Y., Sateia, M. J., Lyness, J. M., ... Perlis, M. L. (2008). Is insomnia a perpetuating factor for late-life depression in the IMPACT cohort? *Sleep*, 31(4), 481–488.
- Prather, A. A., Janicki-Deverts, D., Hall, M. H., & Cohen, S. (2015). Behaviorally assessed sleep and susceptibility to the common cold. *Sleep*, 38(9), 1353–1359. doi:10.5665/sleep.4968
- Punjabi, N. M., Caffo, B. S., Goodwin, J. L., Gottlieb, D. J., Newman, A. B., O'Connor, G. T., ... Samet, J. M. (2009). Sleep-disordered breathing and mortality: A prospective cohort study. *PLoS Medicine*, 6(8). doi:10.1371/journal.pmed.1000132
- Punjabi, N. M., Shahar, E., Redline, S., Gottlieb, D. J., Givelber, R., & Resnick, H. E. (2004). Sleep-disordered breathing, glucose intolerance, and insulin resistance: The sleep heart health study. *American Journal of Epidemiology*, 160(6), 521–530. doi:10.1093/aje/kwh261
- Quan, S. F., Chan, C., Dement, W. C., Gevins, A., Goodwin, J. L., Gottlieb, D. J., ... Kushida, C. A. (2011). The association between obstructive sleep apnea and neurocognitive performance—The Apnea Positive Pressure Long-term Efficacy Study (APPLES). *Sleep*, 34(3), 303–314B.
- Rechtschaffen, A., & Bergmann, B. M. (1995). Sleep deprivation in the rat by the disk-over-water method. *Behavioural Brain Research*, 69(1–2), 55–63.
- Redline, S., Tishler, P. V., Tosteson, T. D., Williamson, J., Kump, K., Browner, I., ... Krejci, P. (1995). The familial aggregation of obstructive sleep apnea. *American Journal of Respiratory and Critical Care Medicine*, 151(3 I), 682–687.
- Redline, S., Yenokyan, G., Gottlieb, D. J., Shahar, E., O'Connor, G. T., Resnick, H. E., ... Punjabi, N. M. (2010). Obstructive sleep apnea-hypopnea and incident stroke: The sleep heart health study. *American Journal of Respiratory and Critical Care Medicine*, 182(2), 269–277. doi:10.1164/rccm.200911-1746OC
- Reichmuth, K. J., Austin, D., Skatrud, J. B., & Young, T. (2005). Association of sleep apnea and type II diabetes. *American Journal of Respiratory and Critical Care Medicine*, 172(12), 1590–1595. doi:10.1164/rccm.200504-637OC
- Resnick, H. E., Redline, S., Shahar, E., Gilpin, A., Newman, A., Walter, R., ... Punjabi, N. M. (2003). Diabetes and sleep disturbances: Findings from the Sleep Heart Health Study. *Diabetes Care*, 26(3), 702–709. doi:10.2337/diacare.26.3.702

- Rosenstock, J., Tuchman, M., Lamoreaux, L., & Sharma, U. (2004). Pregabalin for the treatment of painful diabetic peripheral neuropathy: A double-blind, placebo-controlled trial. *Pain*, 110(3), 628–638. doi:10.1016/j.pain.2004.05.001
- Sadeh, A. (2011). The role and validity of actigraphy in sleep medicine: An update. *Sleep Medicine Reviews*, 15(4), 259–267. doi:10.1016/j.smrv.2010.10.001
- Sadeh, A., & Acebo, C. (2002). The role of actigraphy in sleep medicine. *Sleep Medicine Reviews*, 6(2), 113–124.
- Salorio, C. F., White, D. A., Piccirillo, J., Duntley, S. P., & Uhles, M. L. (2002). Learning, memory, and executive control in individuals with obstructive sleep apnea syndrome. *Journal of Clinical and Experimental Neuropsychology*, 24(1), 93–100. doi:10.1076/jcen.24.1.93.973
- Sánchez, A. I., Martínez, P., Miró, E., Bardwell, W. A., & Bucla-Casal, G. (2009). CPAP and behavioral therapies in patients with obstructive sleep apnea: Effects on daytime sleepiness, mood, and cognitive function. *Sleep Medicine Reviews*, 13(3), 223–233. doi:10.1016/j.smrv.2008.07.002
- Sanders, M. H., & Givelber, R. (2003). Sleep disordered breathing may not be an independent risk factor for diabetes, but diabetes may contribute to the occurrence of periodic breathing in sleep. *Sleep Medicine*, 4(4), 349–350. doi:10.1016/S1389-9457(03)00118-7
- Saunamäki, T., Himanen, S. L., Polo, O., & Jehkonen, M. (2009). Executive dysfunction in patients with obstructive sleep apnea syndrome. *European Neurology*, 62(4), 237–242. doi:10.1159/000232156
- Saunamäki, T., & Jehkonen, M. (2007). A review of executive functions in obstructive sleep apnea syndrome. *Acta Neurologica Scandinavica*, 115(1), 1–11. doi:10.1111/j.1600-0404.2006.00744.x
- Schmid, S. M., Hallschmid, M., Jauch-Chara, K., Born, J., & Schultes, B. (2008). A single night of sleep deprivation increases ghrelin levels and feelings of hunger in normal-weight healthy men. *Journal of Sleep Research*, 17(3), 331–334. doi:10.1111/j.1365-2869.2008.00662.x
- Schmid, S. M., Hallschmid, M., Jauch-Chara, K., Wilms, B., Lehnert, H., Born, J., & Schultes, B. (2011). Disturbed glucoregulatory response to food intake after moderate sleep restriction. *Sleep*, 34(3), 371–377.
- Schmid, S. M., Hallschmid, M., & Schultes, B. (2015). The metabolic burden of sleep loss. *The Lancet Diabetes and Endocrinology*, 3(1), 52–62. doi:10.1016/S2213-8587(14)70012-9
- Shahar, E., Whitney, C. W., Redline, S., Lee, E. T., Newman, A. B., Nieto, F. J., ... Samet, J. M. (2001). Sleep-disordered breathing and cardiovascular disease: Cross-sectional results of the Sleep Heart Health Study. *American Journal of Respiratory and Critical Care Medicine*, 163(1), 19–25.
- Shan, Z., Ma, H., Xie, M., Yan, P., Guo, Y., Bao, W., ... Liu, L. (2015). Sleep duration and risk of type 2 diabetes: A meta-analysis of prospective studies. *Diabetes Care*, 38(3), 529–537. doi:10.2337/dc14-2073
- Sherman, H., Genzer, Y., Cohen, R., Chapnik, N., Madar, Z., & Froy, O. (2012). Timed high-fat diet resets circadian metabolism and prevents obesity. *FASEB Journal*, 26(8), 3493–3502. doi:10.1096/fj.12-208868
- Sin, D. D., Fitzgerald, F., Parker, J. D., Newton, G., Floras, J. S., & Bradley, T. D. (1999). Risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. *American Journal of Respiratory and Critical Care Medicine*, 160(4), 1101–1106.
- Sivertsen, B., Lallukka, T., Petrie, K. J., Steingrimsdottir, O. A., Stubhaug, A., & Nielsen, C. S. (2015). Sleep and pain sensitivity in adults. *Pain*, 156(8), 1433–1439. doi:10.1097/j.pain.0000000000000131
- Sivertsen, B., Salo, P., Mykletun, A., Hysing, M., Pallesen, S., Krokstad, S., ... Øverland, S. (2012). The bidirectional association between depression and insomnia: The HUNT study. *Psychosomatic Medicine*, 74(7), 758–765. doi:10.1097/PSY.0b013e3182648619
- Somers, V. K., Dyken, M. E., Clary, M. P., & Abboud, F. M. (1995). Sympathetic neural mechanisms in obstructive sleep apnea. *Journal of Clinical Investigation*, 96(4), 1897–1904.
- Spiegel, K., Leproult, R., L'Hermite-Balériaux, M., Copinschi, G., Penev, P. D., & Van Cauter, E. (2004). Leptin levels are dependent on sleep duration: Relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. *Journal of Clinical Endocrinology and Metabolism*, 89(11), 5762–5771. doi:10.1210/jc.2004-1003

- Spiegel, K., Leproult, R., & Van Cauter, E. (1999). Impact of sleep debt on metabolic and endocrine function. *Lancet*, 354(9188), 1435–1439.
- St-Amand, J., Provencher, M. D., Bélanger, L., & Morin, C. M. (2013). Sleep disturbances in bipolar disorder during remission. *Journal of Affective Disorders*, 146(1), 112–119. doi:10.1016/j.jad.2012.05.057
- Staner, L. (2010). Comorbidity of insomnia and depression. *Sleep Medicine Reviews*, 14(1), 35–46. doi:10.1016/j.smrv.2009.09.003
- Stewart, R., Besset, A., Bebbington, P., Brugha, T., Lindesay, J., Jenkins, R., ... Meltzer, H. (2006). Insomnia comorbidity and impact and hypnotic use by age group in a national survey population aged 16 to 74 years. *Sleep*, 29(11), 1391–1397.
- St-Onge, M. P., Roberts, A. L., Chen, J., Kelleman, M., O'Keeffe, M., RoyChoudhury, A., & Jones, P. J. H. (2011). Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. *American Journal of Clinical Nutrition*, 94(2), 410–416. doi:10.3945/ajcn.111.013904
- Stoohs, R. A., Gingold, J., Cohrs, S., Harter, R., Finlayson, E., & Guilleminault, C. (1996). Sleep-disordered breathing and systemic hypertension in the older male. *Journal of the American Geriatrics Society*, 44(11), 1295–1300.
- Strohl, K. P., & Redline, S. (1996). Recognition of obstructive sleep apnea. *American Journal of Respiratory and Critical Care Medicine*, 154(2), 279–289.
- Sullivan, C. E., Issa, F. G., Berthon-Jones, M., & Eves, L. (1981). Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. *Lancet*, 1(8225), 862–865.
- Szymczak, J. T., Jasinska, M., Pawlak, E., & Zwierzykowska, M. (1993). Annual and weekly changes in the sleep-wake rhythm of school children. *Sleep*, 16(5), 433–435.
- Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine*, 1(3), e62. doi:10.1371/journal.pmed.0010062
- Talbot, L. S., Maguen, S., Metzler, T. J., Schmitz, M., McCaslin, S. E., Richards, A., ... Neylan, T. C. (2014). Cognitive behavioral therapy for insomnia in posttraumatic stress disorder: A randomized controlled trial. *Sleep*, 37(2), 327–341. doi:10.5665/sleep.3408
- Terán-Santos, J., Jiménez-Gómez, A., & Cordero-Guevara, J. (1999). The association between sleep apnea and the risk of traffic accidents. *New England Journal of Medicine*, 340(11), 847–851. doi:10.1056/nejm199903183401104
- Trauer, J. M., Qian, M. Y., Doyle, J. S., Rajaratnam, S. M. W., & Cunnington, D. (2015). Cognitive behavioral therapy for chronic insomnia: A systematic review and meta-analysis. *Annals of Internal Medicine*, 163(3), 191–204. doi:10.7326/M14-2841
- Tregear, S., Reston, J., Schoelles, K., & Phillips, B. (2010). Continuous positive airway pressure reduces risk of motor vehicle crash among drivers with obstructive sleep apnea: Systematic review and meta-analysis. *Sleep*, 33(10), 1373–1380.
- Trockel, M., Manber, R., Chang, V., Thurston, A., & Taylor, C. B. (2011). An e-mail delivered CBT for sleep-health program for college students: Effects on sleep quality and depression symptoms. *Journal of Clinical Sleep Medicine*, 7(3), 276–281. doi:10.5664/JCSM.1072
- Vaughn, B. V., & Giallanza, P. (2008). Technical review of polysomnography. *Chest*, 134(6), 1310–1319. doi:10.1378/chest.08.0812
- Veasey, S., Rosen, R., Barzansky, B., Rosen, I., & Owens, J. (2002). Sleep loss and fatigue in residency training: A reappraisal. *Journal of the American Medical Association*, 288(9), 1116–1124.
- Verstraeten, E. (2007). Neurocognitive effects of obstructive sleep apnea syndrome. *Current Neurology and Neuroscience Reports*, 7(2), 161–166.
- Vinik, A., Emir, B., Cheung, R., & Whalen, E. (2013). Relationship between pain relief and improvements in patient function/quality of life in patients with painful diabetic peripheral neuropathy or postherpetic neuralgia treated with pregabalin. *Clinical Therapeutics*, 35(5), 612–623. doi:10.1016/j.clinthera.2013.03.008

- Waldhorn, R. E., Herrick, T. W., Nguyen, M. C., O'Donnell, A. E., Soderro, J., & Potolicchio, S. J. (1990). Long-term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnea. *Chest*, 97(1), 33–38.
- Wallace, A., & Bucks, R. S. (2013). Memory and obstructive sleep apnea: A meta analysis. *Sleep*, 36(2), 203–220.
- Wang, X., Bi, Y., Zhang, Q., & Pan, F. (2013). Obstructive sleep apnoea and the risk of type 2 diabetes: A meta-analysis of prospective cohort studies. *Respirology*, 18(1), 140–146. doi:10.1111/j.1440-1843.2012.02267.x
- Watson, N. F., Badr, M. S., Belenky, G., Bliwise, D. L., Buxton, O. M., Buysse, D., ... Heald, J. L. (2015). Recommended amount of sleep for a healthy adult: A joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society. *Sleep*, 38(6), 843–844. doi:10.5665/sleep.4716
- Wessendorf, T. E., Thilmann, A. F., Wang, Y., Schriber, A., Konietzko, N., & Teschler, H. (2000). Fibrinogen levels and obstructive sleep apnea in ischemic stroke. *American Journal of Respiratory and Critical Care Medicine*, 162(6), 2039–2042.
- Wetter, D. W., Young, T. B., Bidwell, T. R., Badr, M. S., & Palta, M. (1994). Smoking as a risk factor for sleep-disordered breathing. *Archives of Internal Medicine*, 154(19), 2219–2224.
- Wolfson, A. R., & Carskadon, M. A. (1998). Sleep schedules and daytime functioning in adolescents. *Child Development*, 69(4), 875–887.
- Wolkove, N., Baltzan, M., Kamel, H., Dabrusin, R., & Palayew, M. (2008). Long-term compliance with continuous positive airway pressure in patients with obstructive sleep apnea. *Canadian Respiratory Journal: Journal of the Canadian Thoracic Society*, 15(7), 365–369.
- Yaggi, H. K., Concato, J., Kernan, W. N., Lichtman, J. H., Brass, L. M., & Mohsenin, V. (2005). Obstructive sleep apnea as a risk factor for stroke and death. *The New England Journal of Medicine*, 353(19), 2034–2041. doi:10.1056/NEJMoa043104
- Yaouhi, K., Bertran, F., Clochon, P., Mzenge, F., Denise, P., Foret, J., ... Desgranges, B. (2009). A combined neuropsychological and brain imaging study of obstructive sleep apnea. *Journal of Sleep Research*, 18(1), 36–48.
- Ye, Y. Y., Zhang, Y. F., Chen, J., Liu, J., Li, X. J., Liu, Y. Z., ... Jiang, X. J. (2015). Internet-based cognitive behavioral therapy for insomnia (ICBT-i) improves comorbid anxiety and depression—A meta-analysis of randomized controlled trials. *PLoS One*, 10(11). doi:10.1371/journal.pone.0142258
- Young, T., Blustein, J., Finn, L., & Palta, M. (1997). Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. *Sleep*, 20(8), 608–613.
- Young, T., Palta, M., Dempsey, J., Skatrud, J., Weber, S., & Badr, S. (1993). The occurrence of sleep-disordered breathing among middle-aged adults. *The New England Journal of Medicine*, 328(17), 1230–1235.
- Zachariae, R., Lyby, M. S., Ritterband, L. M., & O'Toole, M. S. (2016). Efficacy of internet-delivered cognitive-behavioral therapy for insomnia—A systematic review and meta-analysis of randomized controlled trials. *Sleep Medicine Reviews*, 30, 1–10. doi:10.1016/j.smrv.2015.10.004
- Zayfert, C., & De Viva, J. C. (2004). Residual insomnia following cognitive behavioral therapy for PTSD. *Journal of Traumatic Stress*, 17(1), 69–73. doi:10.1023/B:JOTS.0000014679.31799.e7
- Zhang, T., & Chan, A. H. S. (2014). Sleepiness and the risk of road accidents for professional drivers: A systematic review and meta-analysis of retrospective studies. *Safety Science*, 70, 180–188. doi:10.1016/j.ssci.2014.05.022

Telepsychology

Shawna Wright and Eve-Lynn Nelson

*KU Center for Telemedicine & Telehealth, University of Kansas Medical Center,
University of Kansas, Kansas City, KS, USA*

Telepsychology is defined as the provision of psychological services using telecommunication technologies. The term *telepsychology* falls underneath the umbrella term *telemental health*, also called *telebehavioral health*. Telemental health refers to all the names and types of behavioral and mental health services, including discipline-specific applications such as telepsychology that are provided via synchronous telecommunication technologies. Telemental health specifically refers to mental health assessment, treatment, education, monitoring, and collaboration (Bashshur, Shannon, Bashshur, & Yellowlees, 2012). Secure videoconferencing allows psychologists to communicate with a client and observe nonverbal behavior in real time to approximate the relationship developed through in-person therapy. The visual component in videoconferencing establishes a social presence and promotes familiarity, connectedness, and comfort for discussing complex topics. This type of social presence cannot be established with the use of the telephone alone. The rationale for using televideo has remained consistent over the last two decades, with a focus on using telecommunication technology to bridge the gap between a high need for psychological services and limited or no access to a growing number of empirically supported and evidence-based interventions.

Telemental health saves telepsychologists the time and expense of travel and has been applied to both rural and urban settings. Additional benefits of this approach are found in connecting systems of care and enhancing care coordination. Supervised settings that include a telepresenter, such as clinics, hospitals, primary care practices, schools, daycare facilities, detention centers, and other settings, have been the most frequent sites of connection with clients and have been associated with reimbursement (Bashshur et al. 2012). Mobile devices are expanding the reach and consideration of expanding secure videoconferencing to unsupervised settings such as the home.

Telepsychology expansion is driven by increasing expectations for high-quality behavioral health services across geographies. Telepsychology is further advanced by the decreasing cost of secure videoconferencing options and increasing access to high-speed connectivity.

Telemental health is a relatively low-cost technology solution because behavioral health interventions predominantly rely on verbal communication and observation and there is minimal need for more expensive peripheral devices.

Healthcare reform has increased interest in innovative solutions to increase accessibility to behavioral health services due to challenges associated with the shortages of behavioral health specialists, a maldistribution of available specialists, a shrinking behavioral health specialist workforce, and instability in behavioral health funding. Rural and frontier communities are particularly affected by difficulties accessing behavioral health treatment because of shrinking populations, declining economies, limited transportation options, and increasing poverty (Myers & Turvey, 2012). The burdens of traveling for services are often amplified by the frequent standard of care for regular sessions sustained over a period of time.

Telepsychology services build on a long history of moving mental healthcare from the distant mental health clinic to the community in order to increase access to care, decrease stigma, and increase adherence to treatment planning. Telepsychology offers the opportunity to enhance treatment effectiveness and care coordination in naturalistic settings. The provision of care in local community settings has the advantages of gathering information from multiple informants/supporters about the broad range of contextual factors that influences a client's behaviors and mental health needs.

Telepsychology services initially focused on rural settings, but they are increasingly offered in urban/metropolitan locations to bridge access gaps. Mental health centers may provide infrastructure that facilitates the implementation of telepsychology services. For example, many schools are seeking to understand their students' mental health needs and are willing to use existing videoconferencing systems to access telemental health services. Most behavioral health diagnoses across the developmental spectrum have been evaluated through videoconferencing consistent with clients in usual outpatient practice. The authors first summarize key considerations in establishing a telepsychology clinic and then describe telepsychology best practices.

Establishing a Telepsychology Clinic

In order to maximize telepsychology clinic success, psychologists are encouraged to consider outreach best practices when establishing a clinic. Taking time to consider the unique needs of the clinic and to extend relationships with the target community/communities will assist in building a sustainable, high-quality telepractice.

Needs Assessment

Psychologists considering telepsychology services are encouraged to complete a needs assessment within the identified community to evaluate perceived interest and availability of such psychological services (Nelson & Duncan, 2015). This assessment should include discussions and meetings across key constituents, including consumers and their families, local mental health professionals, primary care providers, consumer advocates, and other community leaders likely to be affected by telepsychology initiation. Telepsychology works best when seen as complementing or adding to the community's local care.

The needs assessment should also identify the population the telepsychology service will serve. The choice of who will be seen depends on clients' preferences, developmental and diagnostic considerations, personnel and other resources at the distant site, and the

psychologist's comfort. Telepsychology may be especially suited for younger populations who are often already comfortable and accepting of the technology medium. However, there is no data suggesting client exclusion based on age. With telepsychology's extended reach comes the responsibility of the psychologist to follow all professional standards of cultural competence across ethnicities, languages, genders, religions, sexual preferences, geographies, and other competence areas, including an understanding of the impact of rural culture on the clinical presentation.

Technology Selection

Fortunately, televideo services have recently benefited from higher-quality technology paired with lower cost. Technological advances in personal computer-based videoconferencing systems have made inexpensive, user-friendly, reliable, secure videoconferencing more available and have promoted an increase in the use of clinical televideo.

The purpose of the psychological service should guide technology selection, which may or may not need more expensive or newest technology upgrades. When considering technologies and vendors, it is advisable to follow up with colleagues and resources such as the federally funded telehealth resource centers (see <http://www.telehealthresourcecenter.org>) in order to gain comprehensive information about the best fit. Psychological services require videoconferencing equipment, but do not generally require the more expensive peripherals associated with other specialties (e.g., digital otoscope, stethoscope). Similar to other equipment purchases, psychologists should communicate with technology vendors as well as with colleagues and peers who have used technology to deliver treatment. Regional telehealth resource centers may offer feedback and recommendations for matching the appropriate technology with the provider and service. While individual technology standards often facilitate interoperability with other sites, the use of consistent technologies (e.g., brand of equipment, level of equipment) across the televideo network may decrease difficulties with connections.

Successful implementation of videoconferencing services calls for the psychologist and distant site to assess the availability of (a) modern, well-functioning televideo equipment, (b) encrypted videoconferencing software, (c) secure clinical space for the equipment setup, and (d) consistent high-speed connectivity. For telepsychology consultations, serious consideration should be given to privacy and security. Most guidelines recommend use of point-to-point encrypted software, with transparent company information about transmission and encryption protocols. Some sites need to run an additional network line to support the additional bandwidth requirements associated with televideo services. It is important to address sustainability of maintaining and paying for equipment, software, and connectivity when establishing a telepsychology clinic. Initial and ongoing training in the technologies and access to technical assistance should be considered.

Televideo Clinic Room

Televideo brings together systems of care, linking the psychologist with schools, clinics, primary care offices, and other systems of care. Therefore, televideo sites should identify rooms large enough to accommodate many participants as well as strategies to manage the participants in and out of the televideo room. Lighting is also important to clearly see facial expressions and affective responses. The technology at the provider site may have some benefits not seen in the on-site setting, such as the ability to unobtrusively zoom in and note physical presentations such as tics, gait, or nonverbal gestures.

Telepsychology Protocols

Psychologists are encouraged to develop protocols for their telepsychology clinics and update protocols annually. Telepsychologists should consider maintaining protocols referral and scheduling, pre-consult preparation, televideo session, crisis intervention, and follow-up. Telepsychology protocols should identify who is expected to attend the appointment (client, family members, case managers, other community members) and who is expected to coordinate the appointment at the distant site (school nurse, clinic nurse, other clinic personnel, etc.). Telepsychology consults, other than in the home setting, most often include a telepsychology coordinator to assist during the consult. The coordinator at the client site facilitates telepsychology session and often serves as the telepsychology “champion.” Psychologists must carefully consider the risks and benefits related to inclusion coordinator for their specific clinical purpose. Psychologists often work with coordinators who provide assistance with the technology, support for the client, and immediate help in the event safety or other emergent concerns.

Telepsychology Coordinator

A telepsychology coordinator, sometimes called a telefacilitator, is an individual trained in assisting clients throughout their videoconferencing experience. The telepsychology coordinator is responsible for promoting the psychological service, scheduling the consultation, compiling intake packets, socializing the client and family to the televideo equipment, assisting during the consultation, and helping the family follow up on recommendations (Shore & Manson, 2005). The telepsychology coordinator also assists the psychologist in confirming that telepsychology encounters are private because the psychologist has less direct control of the distant environment. The telepsychology coordinator alerts the psychologist concerning those present in the secure televideo room as well as monitors the physical environment to ensure that others cannot overhear the communication occurring in the televideo room. While the telepsychology coordinator facilitates clinic implementation, his or her attendance in clinical encounters differs from most on-site clinics.

Ethical and Regulatory Considerations

Careful consideration and planning around regulatory and ethical issues may affect when telepsychology programs are able to launch. Comparable with on-site clinical settings, the core ethical concern to protect the client remains paramount for videoconferencing settings (Nelson & Duncan, 2015). In addition, professional guidelines addressing telepsychology are emerging to inform reasonable steps for telepsychology practice across clinical, administrative, and technical considerations, including child-specific guidelines. Telemental health guidelines are available through the American Telemedicine Association and the American Psychological Association, as well as a growing number of related mental health professional organizations.

There is limited state and national guidance concerning regulation of psychological services over televideo, with only a few state regulations specifically addressing televideo practice. Without guidance, a circular situation is created in which the lack of guidance potentially limits psychological practice and the lack of practice volume makes guidance a low priority. State psychology leaders (e.g., Ohio, others) encourage best practices within the framework of existing regulations. In general, the psychologist must be licensed in the state of the client’s clinic

location and across state, national, and international boundaries. Credentialing should also be considered based on the service sites.

At the national level, the Joint Commission on Accreditation of Healthcare Organizations has regulations applicable to telemedicine. Federal, state, and local laws, as well as institutional requirements, must also be reviewed related to the following: (a) involuntary commitment and reporting child maltreatment, (b) teleprovider credentialing and privileging requirements, and (c) malpractice insurance specific to telemedicine.

Interstate licensure is a complex issue; the telepsychologist is encouraged to review state licensure requirements related to the teleprovider location and the client location. Other regulatory/ethical requirements to consider include verifying the client identity and confirming the location of the client; this is often more easily accomplished in supervised than in unsupervised settings (e.g., the home). It is equally important for the teleprovider to inform the client of the teleprovider's location and credentials.

Other important factors to consider when initiating a telepsychology service include the client's privacy and compliance of the videoconferencing transmission with the Health Insurance Portability and Accountability Act. State law regarding confidentiality of behavioral health information should be reviewed, and when applicable, school-specific confidentiality requirements as outlined in the Family Educational Rights and Privacy Act Regulations. Informed consent for treatment may include an additional consent to receive telepsychology services. A review of the potential limitations and acknowledgment of the clients' right to refuse treatment over videoconferencing is part of informed consent as well as clarification of whether the sessions will be recorded and/or stored in any way. Best practices in documentation and use of the electronic health record should also be followed.

Sustainability

Psychologists should consider the sustainability of their telepsychology service over time. The initial costs associated with starting telepsychology include equipment and software costs, connectivity/line charges, installation costs, costs of remodeling or adding space, telemedicine training for personnel, and costs with adding staff to assist with telemedicine or with changing workflow to meet telemedicine responsibilities. Costs typically decrease over time as client volume increases. Psychologists have used various funding for initial televideo implementation, including institutional seed money, community and foundational support, state grants, federal funding, billing reimbursement, and contractual agreements.

Reimbursement is an important step toward sustaining telepsychology services. States are increasingly implementing parity legislation requiring televideo services be reimbursed as on-site benefits. A growing number of third-party insurers reimburse televideo services, including Medicare, the majority of state Medicaid policies, and many private insurers (Gros et al. 2013). In addition to provider billing, some sites may be eligible for Medicare/Medicaid's originating site fee. There is variation in coverage based on insurer, provider type, and location, and telepsychologists must carefully consider their own setting. In general, many, but not all, current procedural terminology (CPT) codes may be covered for televideo services, with the addition of a GT telemedicine modifier to the billing code, which indicates audio or video service provision. Billing reimbursement is an important step in televideo sustainability, but the same challenges seen in on-site billing for mental health services remain over televideo. Some telepsychologists may utilize other business models, such as contractual agreements with sites to cover both the psychologist's time and related costs (e.g., line charges, office management, etc.).

Telepsychology services to primary care settings are anticipated to increase with national and state initiatives around the client-centered medical home. The medical home model advances coordination and communication between primary care and psychologists. Telepsychology is a viable strategy to increase access to psychologists in primary care settings. As in other telepsychology settings spanning systems of care, team building and good communication skills remain crucial for a successful delivery of service.

New telepsychology models continue to emerge, including videoconferencing directly to the consumer in the home. A range of technologies are used for home-based services, and psychologists should consider the risks/benefits of the videoconferencing technology, including security and reliability. While home-based televideo services increase access, there is less control over the environment in this unsupervised setting (e.g., without a telepsychology coordinator). Psychologists providing service to the home should continue to consider key telepsychology components including (a) informed consent for telepsychology service; (b) protocols concerning the expected process, including key components discussed in other videoconferencing settings above (e.g., scheduling, follow-up, secure environment, backup plans in the event that the technology fails to the home, etc.); and (c) safety plans in the event the client reports suicidal, homicidal, or other safety concerns. Telepsychology services to the home are generally fee-for-service and not reimbursed by third-party payers. Mobile technologies (e.g., smartphones, tablets, etc.) with video capabilities expand accessibility further, but consideration must be given to the new benefits and risks for the client and their families.

Telepsychology in Practice

Telepsychologists

Due to the newness of the field, there are no established performance competencies specific to technology, but telehealth guidelines and overall professional ethics guidelines provide direction. Telepsychologists should consider practice with the technology as well as cultural competence with the population served by the telepsychology clinic. Ideally, psychologists observe other telemental health providers, seek out supervision specific to telehealth, and complete mock/practice sessions in order to build confidence working with the team and implementing the telepsychology protocols. The technology itself offers unique opportunities for telesupervision, with requirements varying across states and health professions.

Telepsychology Clients

Selection of clients to be seen over televideo is contingent upon client and guardian preferences, developmental and diagnostic considerations, personnel and other resources at the distant site, and the telepsychologist's level of televideo competence and comfort. Psychological services over televideo may be especially suited for youth who are often already comfortable and accepting of technological media. Some studies suggest that televideo offers unique advantages, including less self-consciousness and decreased confidentiality concerns, as the telepsychologist is outside of the local community.

Across telepsychology clinics, clients tend to present with the same concerns as seen in traditional clinic settings. No presentation or diagnostic category has been excluded from mental health services over televideo. However, the same careful consideration used in traditional clinics should be taken related to competence with the presenting concern as well

as access to necessary resources, particularly for clients with severe symptoms and impairments. Research has demonstrated that televideo is equivalent to face-to-face assessment and treatment of psychosis and that televideo is well tolerated and accepted by this population.

The Client–Provider Relationship and Televideo Etiquette

Psychologists should strive to replicate the same client engagement and rapport building strategies to the telemental health session (Grady et al., 2011). Shadowing existing teleproviders, seeking supervision for telepsychological intervention, and consulting with colleagues may foster the telepsychologist's comfort level associated with strong therapeutic relationship building. Strategies used in on-site visits translate to the telepsychology environment, such as noting a child wearing a shirt from a favorite team or taking time to talk about local events. Some telepsychologists may use more animated gestures or attend more closely to nonverbal cues such as facial expressions, although overly exaggerated/fast movements/hand gestures should be kept to a minimum to avoid pixilation. Verbal communications may be more deliberate to adjust for the slight auditory lag and to clearly indicate when the telepsychologist has finished speaking in order to facilitate reciprocity in communication, although this is decreasing as a less frequent concern due increasing availability of high-speed connectivity.

Families may be apt to quickly accommodate to the technology setting due to previous experience in using videoconferencing for social purposes, such as connecting with friends or family members. Telepsychologists should consider providing clients with reminders of the clinical nature of the televideo interaction as well as the additional security of the videoconferencing systems.

Technological Factors Affecting the Clinical Encounter

A high-quality video signal is imperative to the success of the telepsychology session, with recommendations generally advising a bandwidth of greater than 384 kB per second. A high-quality audio signal also assists with identifying nuances of an individual's verbal communication. The secure televideo room should ideally be away from clinic and street noise because the microphones are very sensitive, and extraneous sounds can interfere with the session. Toys can produce uncomfortable levels of noise, and foam blocks, books, markers, and papers may be an alternative, depending on the client population and developmental stage.

Video quality and camera placement are important to the caliber of the televideo session as they allow the telepsychologist to clearly observe facial expressions, affect, relatedness, crying, and other nonverbal reactions. Considerations for video quality include minimizing shadows, positioning lighting to emanate from behind the camera, selecting rooms without windows or adequate window cover to control lighting, staging professional backgrounds consistent with typical office settings, and avoidance of clothing with high contrast (e.g., stripes, busy patterns).

Technicians should support telepsychologists with strategies to maximize the quality of the videoconferencing encounter. Current videoconferencing speeds have decreased the pixilation/tiling associated with earlier videoconferencing rendering fewer technical difficulties over time. Consideration should be given to camera angle, monitor selection, and positioning in order to better facilitate communication. Telepsychologists are encouraged to check in with clients to make sure clients can see and hear telepsychologists as expected. Even though many televideo platforms have the ability to record sessions, telepsychologists should inform clients that sessions are not videotaped or archived in any way without client knowledge and consent.

Referral and Scheduling

As in other outreach clinics, telepsychologists should identify office personnel responsible for scheduling across the client sites (e.g., clinics, hospitals, schools, home, etc.). This person is responsible for coordinating the telepsychologist's schedule, the client site schedule, and room availability. As in the in-person setting, it is helpful to obtain the referral for the new client and determine if the client meets the insurance requirements consistent with the practice. Generally, the same paperwork as in the in-person setting is utilized (e.g., psychology intake form; registration form, including insurer information; consent form, including telemedicine-specific language; HIPAA-related Notice of Privacy Protection [NPP]; previous medical history and documentation; requested lab or other tests prior to the telemedicine encounter; any other information requested by the telemental health team specific to the consult). Best practices related to the transmission of health-related information should be followed, and the client's designated medical record is maintained by the telepsychologist.

Pre-session Preparation

The same on-site guidelines are followed for quiet, safe, handicap accessible, client-friendly space for televideo encounters. The secure televideo room must be large enough to accommodate all participants. The connection at the remote site should be tested for compatibility and entered into the directory for easy dialing. The technician may assist the telepsychologist with videoconferencing basics (e.g., that the microphone is not muted, that the camera is focused on the provider, that microphones are placed away from the monitor's speakers, and that the client is aware of any other individuals in the room). Backup plans should be in place in the event of technology failure, usually telephone follow-up. The televideo coordinator should complete training with the psychology team before initiating services. This includes shared expectations concerning support of the client related to the televideo encounters and assistance to the provider during the encounters. Safety protocols should be reviewed in the event the client presents with immediate suicidal concerns and needs local emergency services.

Televideo Session

The session approximates all key elements of the on-site visit at initial and follow-up sessions. The same attention to process and rapport building is given in the televideo appointments as in on-site appointments. The telepsychologist has full discretion to advise the client to be seen on-site. Overall, research has supported the feasibility and accuracy of psychological diagnostic interview and the diagnostic rating scales over televideo. Neuropsychological testing over televideo has been shown to be feasible as a broad indication of impairment, although some hands-on neuropsychological tests are more difficult to translate to the televideo setting. To date, there is very little clinical or research data concerning personality tests over televideo. Programmatic descriptions and a growing number of controlled trials directly comparing in-person with telepsychology services are encouraging, although more research is needed across presenting concerns and across the lifespan.

As in on-site clinics, the telepsychologist gathers information about the presenting concern and the relevant history. The telepsychologist observes the client throughout the session and notes behavior as well as physical presentation (e.g., gait, tics, affect). The zoom feature of the televideo camera can assist with this and allow unobtrusive close-up observation of such features. The telepsychologist completes the same documentation and billing forms as in on-site clinics and maintains the file/medical record.

Post-session

The telepsychologist and televideo coordinator collaborate to assist the client in understanding and following treatment recommendations and referral recommendations. The telepsychologist advises the client about follow-up appointments via televideo or on-site. Billing considerations are followed as described above. The same follow-up correspondence between telepsychologist and referral source is completed. The protocol for on-site visits is utilized for between-session needs, and information is shared with the client regarding designation of local emergency services in the event of crisis.

Conclusion

When best telepsychology practices are followed, telepsychology holds much promise to extend evidence-based treatments to clients and families and improve access to quality care for rural and other underserved clients/families. Evaluation across client, provider, and system metrics has been promising, although additional research is needed. Psychologists considering utilizing this technology should complete a needs assessment at both the community and clinic level in order to address the feasibility of acquiring and using the technology, understand the demand for the telepsychology service, and develop a business plan for sustaining services. Many successful telepsychology services benefit from strong relationships with communities and telehealth champions and are supported by a collaborative outreach team (i.e., psychologist, technical and scheduling support, telepsychology coordinators, etc.).

Psychologists who acquire this technology should use it in accordance with their professional organizations' ethical standards for best practice and develop a working protocol in order to minimize client risks. Initial and ongoing training and consultation with the telepsychologist's colleagues/community of practice is strongly encouraged. The unique risks and benefits of supervised and unsupervised/home telepsychology settings should be carefully considered and shared within the informed consent process. New technologies (e.g., smartphones, mobile tablets) offer exciting potential to extend reach with secure videoconferencing, as well as emerging potential to pair telepsychology services with other health technologies in order to best meet the needs of the individual client.

Author Biographies

Shawna Wright, associate director of KU's Center for Telemedicine and Telehealth, is a licensed psychologist. She obtained her graduate training in clinical psychology at Texas Tech University. Dr. Wright worked for over a decade in community mental health and has been a practicing telepsychologist since 2011.

Through her experience with providing treatment to rural and underserved populations, Dr. Wright is acutely aware of the impact of the shortage of medical and mental health providers, and she sees the promise of telehealth for improving access to care. She has interests in advancing telehealth policy and engaging medical and mental health professionals and students in telemedicine education. Dr. Wright works with KUMC's Project ECHO as project facilitator and behavioral health content expert to assist Kansas health/mental health providers with increasing access to up-to-date research outcomes, best practices, and standards of care to reduce health disparities.

Dr. Wright is a member of the Rural and Frontier Subcommittee of the Governor's Behavioral Health Services Planning Council, the Kansas State Epidemiological Outcomes Workgroup, and the Kansas Prescription Drug and Opioid Advisory Committee. She works with these groups to understand gaps in access to care and to identify ways telemedicine can be leveraged to increase health equity in Kansas.

Eve-Lynn Nelson is a research advisor and supervisor for health psychology students conducting clinical research and offers GRA positions at KUMC in the area of telemedicine.

Dr. Nelson also teaches graduate courses in health psychology and supervises the telemedicine practicum at KUMC.

References

- Bashshur, R., Shannon, G., Bashshur, N., & Yellowlees, P. (2012). The empirical evidence for telemedicine interventions in mental disorders. *Telemedicine and e-Health*, [Epub ahead of print].
- Grady, B., Myers, K. M., Nelson, E.-L., Belz, N., Bennett, L., Carnahan, L., ... Rowe, N. (2011). Evidence-based practice for telemental health. *Telemedicine and e-Health*, 17(2), 131–148.
- Gros, D., Morland, L., Greene, C., Aciero, R., Strachan, M., Egede, L., ... Frueh, B. C. (2013). Delivery of evidence-based psychotherapy via video telehealth. *Journal of Psychopathology and Behavioral Assessment*, 35(4), 506–521.
- Myers, K., & Turvey, C. (Eds.) (2012). *Telemental health: Clinical, technical and administrative foundation for evidence-based practice*. London: Elsevier.
- Nelson, E.-L., & Duncan, A. (2015). Cognitive-behavioral therapy using televideo. *Cognitive and Behavioral Practice*, 22(3), 269–280.
- Shore, J., & Manson, S. (2005). A developmental model for rural telepsychiatry. *Psychiatric Services*, 56(8), 976–980.

Suggested Reading

- Comer, J., & Barlow, D. (2014). The occasional case against broad dissemination and implementation: Retaining a role for specialty care in the delivery of psychological treatments. *American Psychologist*, 69(1), 1–18.
- Joint Task Force for the Development of Telepsychology Guidelines for Psychologists. (2013). Guidelines for the practice of telepsychology. *American Psychologist*, 68(9), 791–800. doi:10.1037/a0035001
- Kramer, G., & Luxton, D. (2016). Telemental health for children and adolescents: An overview of legal, regulatory, and risk management issues. *Journal of Child and Adolescent Psychopharmacology*, 26(3), 198–203.
- Lambert, D., Gale, J., Hartley, D., Croll, Z., & Hansen, A. (2015). Understanding the business case for telemental health in rural communities. *The Journal of Behavioral Health Services & Research*, 43(3), 366–379.
- Luxton, D. D., Nelson, E.-L., & Maheu, M. (2016). *A practitioner's guide to telemental health: How to conduct legal, ethical, and evidence-based telepractice*. Washington, DC: American Psychological Press.
- Nelson, E.-L., & Sharp, S. (2016). A review of pediatric telemental health. *Pediatric Clinics of North America*, 63(5), 913–931.
- Shore, J., Mishkind, M., Bernard, J., Doarn, C., Bell, I., Jr., Bhatla, R., ... Eppolito, A. (2014). A lexicon of assessment and outcome measures for telemental health. *Telemedicine and e-Health*, 20(3), 282–292.
- Substance Abuse and Mental Health Services Administration. (2015). *Using technology-based therapeutic tools in behavioral health services*. Treatment improvement protocol (TIP) Series 60. HHS Publication No. (SMA) 15-4924. Rockville, MD: Substance Abuse and Mental Health Services Administration.
- Turvey, C., Coleman, M., Dennison, O., Drude, K., Goldenson, M., Hirsch, P., ... Malik, T. S. (2013). ATA practice guidelines for video-based online mental health services. *Telemedicine and e-Health*, 19(9), 722–730.

Traditional Healing Practices and Healers

Robert M. Huff

*Department of Health Sciences, California State University, Northridge,
Morgan Hill, CA, USA*

Causes of illness, disease and death as well as how to prevent and/or treat these conditions have been and continue to be concerns that humankind has sought answers to since their earliest beginnings. Over the millennia, these questions have spawned a host of explanations for illness causality and treatment along with those who would become the carriers of this knowledge. *Traditional medicine* grew out of these early beginnings and continues to be utilized into the present all around the world. Traditional medicine is strongly influenced by the cultural/ethnic groups from which it has emerged with practices and practitioners often used as a first choice for care before or in tangent with Western medical modalities. Traditional medicine is sometimes preferred for three reasons: (a) The cost of treatment tends to be lower than Western biomedicine, (b) the healer can come to the patient, and (c) the healer and patient come from the same cultural milieu with similar worldviews. Traditional medicine can be defined as the health beliefs, practices, and techniques used by indigenous healers to treat and cure the sick. These healing approaches may include herbal and animal-derived products, various minerals, prayer, shamanic journeying and related supernatural modalities, and other forms of alternative medical practices that are passed down through the family/culture from generation to generation. The term traditional medicine is often used interchangeably with the terms *folk medicine*, *complimentary medicine*, or *alternative medicine* to describe these non-western approaches to healing the sick (World Health Organization, n.d.).

Kleinman (1980) in his seminal work, looked at how individual patients and their healers defined health, illness, and disease and calls these *explanatory models*. He notes that the explanatory models of both patients and their health practitioners are important to the understanding of potential or real problems in a health care encounter. For instance, many cultural groups view health and disease as imbalances within the individual and their environment with factors such as ones' emotions, social interactions, physical behaviors, and spiritual or

other metaphysical interactions contributing to these imbalances. Helman (2007) has identified four major categories of illness causality within a variety of cultural groups. These categories include: *the individual world*, *the natural world*, *the social world*, and *the supernatural world*. He notes that illness causality may be seen as emerging from one or more of these levels and in combinations of these. The individual level is concerned with personal behaviors (i.e., diet, exercise, tobacco use, etc.) over which the individual has some control. This also includes hereditary, social, economic and personality variables. The natural world is concerned with both animate and inanimate variables that can lead to illness, disease, and death. This level includes accidents, natural disasters, microbes, injuries from flora and fauna, toxins, weather, and other environmental factors. The social world encompasses interpersonal conflict such as bullying, gang violence, domestic abuse, war, and other similar causes. It should be noted that this level also is concerned with witchcraft, sorcery, evil eye, voodoo, and related practices. The supernatural world is concerned with a variety of entities including angry gods, ancestral spirits, soul loss, spirit possessions, and other metaphysical causes leading to illness and even death. At each of the four levels of disease causation, specialized healers may be necessary to help resolve the cause of a health problem. These may include a shaman, a root doctor, a curandero, spirit medium, herbalist, acupuncturist, or priest. Prayer, sweat lodge ceremonies, chanting, massage, a Western biomedicine practitioner, or combinations of any of these may also be utilized.

Archaeological evidence of *shamanic healing* practices date back thousands of years as seen in early burial practices, cave art, and related artifact materials. Injuries, illness, and death would have been common occurrences in these ancient human cultural groups and the need for someone to explain and treat these events fostered the rise of shamanic healing traditions that have persisted into modern times (Huff, 2002; Huff, Kline, & Peterson, 2015). A shaman is a man or woman capable of trance journeying into the spiritual dimension to seek knowledge regarding healing of the sick, communicating with the spirits of the dead, divining future events, and gaining the favor of the gods, spirit animals or other entities in the natural environment. They may be called upon to perform special ceremonies or rituals to keep a community safe, heal a community member, bring about a good harvest, or placate an angry god or spirit. They may be called to become a shaman as a result of a near death experience where they traveled to the spirit world and returned with new healing knowledge, songs, prayers, rituals, or other revelations given to them by teachers on the other side. A shaman may also be born into the shamanic role under certain circumstances such as being born under a veil (i.e., born with a portion of the amniotic tissue covering their face) or they may be trained if they seem to have special healing abilities (Eliade, 1964). Shamans may use rattles, drums, gongs, finger bells, dancing rituals, purification rites, or physical manipulation such as sucking (i.e., removing a stone, or other material from inside a person by sucking it out). Shamanism has seen resurgence in the United States, with training programs to prepare individuals to take on the role of a shaman in their personal lives.

In Latin American cultures a system known as *curanderismo* has evolved from a number of elements. These include spiritual, homeopathic and other indigenous and Western traditions, with specialized healers who use a variety of methods in their healing work. Medicinal plants are often used and include an extensive pharmacopeia of plants such as cat's claw and dragon's blood for arthritis; aloe vera and chamomile for asthma; camphor and kalallo bush for the common cold; anise, ginger, and mugwort for gastrointestinal problems; artichoke and wormwood for liver problems; and many others (DeStefano, 2001). Shops that sell herbals and other healing items can be found in Hispanic communities and are a ready source for medicines and healing advice. Other healers in the *curanderismo* system include spiritual healers

(i.e., curandero/curanderas who use ritual, prayer and other approaches); those who use massage and physical manipulation; midwives; and others including those who practice the dark arts such as witchcraft and sorcery. Hot and cold illnesses, thought to result from an imbalance between these two humors, are a significant component of Hispanic health beliefs and practices (Ortiz, Shields, Clauson, & Clay, 2007). Cold illnesses are treated with a hot remedy (i.e., upper respiratory infections are considered a cold illness and would be treated with steam baths, hot compresses, cinnamon, and herbal teas); sore throat, a hot condition, would be treated with a cold remedy (i.e., a half and half mixture of honey and lemon juice). The objective is to balance the illness with an opposite treatment-hot with cold and cold with hot. The use of traditional medicine approaches is a common practice among diverse Hispanic populations and should be explored by Western health care providers when treating this population group.

Traditional Southeast Asian groups believe that illness can arise from physical, metaphysical, or supernatural causes. Treatment for any of these health problems may require the services of a Buddhist monk, an herbalist, a shaman, a spirit medium, astrologer, acupuncturist, physician, or other specialists. Traditional Chinese medicine, which has evolved over thousands of years, is based on the concept of Tao (the way or path). It is concerned with harmony and balance between and within the individual and his or her environment. The forces of yin and yang (representing a duality of the universe) must be in balance. Yin is the female side (i.e., the passive or negative female force; the front and inside of the body) while yang is the active male side (the outside and back of the body) of the yin/yang duality. It is represented by light, heat, and fullness. Should this balance be disrupted, illness may be one of its' consequences (Helman, 2007; Kagawa-Singer & Kho, 2015; Spector, 2013). In traditional Chinese medicine one may be treated using acupuncture, acupressure, herbs, moxibustion, cupping, meditation, and may also require a shamanic practitioner. Western biomedical practitioners may also be used in tandem with one or more of the traditional approaches noted above. Traditional Hmong (many who have settled in the United States in recent years) are oriented to animistic beliefs. That is, a belief that spirits inhabit rocks, rivers, ponds, trees, etc. and may, if angered, cause illness and even death. Soul loss, a belief that the soul can leave the body on its own or by being taken by an evil spirit is part of their belief system. Shamanic practitioners may be called in by the family should someone in the household become ill. The shaman may use a number of healing approaches including bells, rattles, herbs, and shamanic journeying to find a lost soul. Western biomedical approaches may be resisted because they are not understood as reported by Fadiman (1997) in her seminal study. Other Southeast Asian population groups vary in their use of traditional healers and beliefs in illness causation. These beliefs range from the natural world to supernatural causes and herbal treatments, prayer, magic, charms, medicinal potions, and Western biomedicine may be used for healing purposes.

American Indian health beliefs and practices revolve around the concept that health is strongly linked to spirituality and the interconnectedness of all things. As with a number of other cultural groups, traditional American Indian medicine is holistic in nature and concerned with maintaining one's balance in the mental, physical, emotional and spiritual domains of life (Hodge & Hodge, 2015). Illness results from an imbalance in any of these domains brought on by natural or supernatural forces. Traditional healers/medicine people may be called upon to perform healing ceremonies that may involve prayers, chanting, purification activities including smudging and sweat lodge ceremonies, herbal medicines, physical manipulations, and diagnosing emotional and spiritual disharmony. The medicine person (male or female) may also function as an arbitrator, psychiatrist, diviner, and spiritual advisor. Western biomedicine is also an accepted modality and hospitals and health centers can be found on

reservations and in urban areas targeted specifically to American Indian and Alaska Native populations. In recent years there has been a trend in integrating traditional healers with Western biomedicine practice. That is, traditional healers work in tandem with Western health care practitioners in hospital settings to perform traditional healing ceremonies for patients while also receiving Western medical treatment modalities (Rhoades, 2009).

Black Americans for the most part share a greater belief in the Western biomedical model though traditional healing practices continue to a greater or lesser degree depending on regional differences, education, socioeconomic status, and related factors (Hopp & Herring, 2015; Mitchem, 2007). Immigrants from other areas of the globe are more likely to hold on to traditional health beliefs and practices that are associated with the cultural milieu from which they have come. For Black Americans, traditional folk medicine practices and practitioners were often their only recourse when formal health care services were not available to them. As with other groups discussed previously, the concepts of harmony and balance in ones' life are believed to help promote good health. Disharmony brings illness and poor health. Congress and Lyons (1992) note that Blacks tend to separate illness into two dimensions: *natural illnesses* that can result from stress, environmental factors, dietary habits, lack of moderation in daily activities, and as punishments from God for sins and other transgressions; and *unnatural illnesses*, which can be brought on by evil forces including bad spirits, witchcraft, spells, and magic. Traditional healers include spiritualists who may use ritual, prayer, magic, and other related activities, and herbalists (root doctors) who use botanicals (i.e., roots, herbs and teas) and may use magic also to bring about healing. Practitioners, who can be male or female, come to their calling by being apprenticed to a folk healer in the community or by being born with a veil (portion of the amniotic sac) over their faces. *Hoodoo* and *Voodoo* are two terms often used when describing healing traditions among Black Americans. Haskins (1990) describes hoodoo (or conjure) as the use of simple medicinal treatments as well as magical practices to bring about healing cures or to ward off or bring about evil. Voodoo is concerned with casting spells or hexes to bring evil and even death to the target of these processes. He notes that these terms are often used interchangeably and that the lines are frequently blurred between them. Folk healers don't necessarily advertise their practices, as they are typically already known in their communities. Spirituality, prayer, and the Church are also important themes in Black communities and many churches have health ministries where health promotion, health screening, counseling, and other health-related services are offered.

Euro-American folk medicine arrived with the first settlers into the New World. They brought with them health beliefs and practices reflecting the cultural areas they immigrated from. Doctors were not always available nor were they particularly well trained. These new colonists utilized what medicines they had from the areas they had come from but also medicines that were shared by the Indians living in close proximity to them. New plants and herbs found their way into the pharmacopeia of these early and later arrivals as they spread throughout the many geographical areas of what ultimately became the United States of America (U.S.) (Cavender, 2003). As these new groups spread across the American landscape, folk medicine practices began to change in the various geographical settings where people settled. Jarvis (1996), in his seminal writings about folk medicine in Vermont, discusses the impact of environment on the health and energy of those living there. He notes that Vermont folk medicine is focused on the entire body in an effort to prevent disease and other conditions of the body. Proper diet is a major key to good health as well as the impact of weather variability. Botanicals are a mainstay of Vermont folk medicine, as well as minerals, honey, and apple cider vinegar. For example, apple cider vinegar is used to treat dizziness, overweight, sore throat, impetigo, and a host of other medical conditions. Many Americans use a teaspoon of apple

cider vinegar and honey as a daily tonic. Honey and honeycomb, alone or in combination with lemon juice or apple cider vinegar, is considered useful for a number of conditions including muscle cramps, burns, hay fever, sinusitis, bed-wetting and other health problems.

Cavender (2003) discusses the use of folk medicine healers and practices in Southern Appalachia from early settlement to the modern era. Many folk medicine practices were brought with these early settlers while botanicals in Southern Appalachia and healing practices learned from the Indians were added to this repertoire. Traditional healers included herb doctors; bloodstoppers; burn doctors; midwives; faith healers; prayer, and reading the Bible as psychological coping mechanisms; magic, and witchcraft. A rich pharmacopeia of medicinal plants utilized by healers in this region included such botanicals as apple cider vinegar for burns, colds, and cuts, black snakeroot for colds and cough and to cleans the blood, pine bark and sap for coughs, worms, and cuts, and sage leaf for nervousness and fever. In today's world, many of the old folk medicine practices are giving way to Western biomedicine. However, folk medicine practices may still be used in conjunction with formal healthcare modalities. Cavender (2003) also notes that a number of other alternative medicine practices including acupressure, Reiki, aromatherapy, massage, vitamin therapy and other modalities have also taken root in Southern Appalachia.

In any discussion of traditional healing practices and how these impact and may be integrated with modern biomedicine, the concept of *cultural competency* should be considered. That is, the need for the health care practitioner to become more knowledgeable about the cultural groups they may be treating. This involves not only looking into the culturally held beliefs and practices of the patient/culture(s) being treated but also ones' own biases and stereotypes and how these may affect the patient-provider encounter. Though the practitioner may not agree with their patient's health beliefs and practices, these must be respected just as the practitioner would expect this from their patient(s). This would seem to be particularly important when dealing with mental health problems that many people, no matter where they are from, are reluctant to talk about. Exploring the patient's use of traditional healing practices in a nonjudgmental manner can help illuminate a great deal about the patient that is being treated. Kleinman and Benson (2006) suggest that the practitioner seek to develop a "mini-ethnography" by questioning the patient as a way to determine their explanatory model for their illness. This would also include what they may have done to treat their problem. Huff, Kline, and Dobbins (2015) present a cultural assessment framework that in whole or part can help the health care provider develop the questions they might ask to help create this mini-ethnography.

Cross-references

Illness representations; Prejudice and stereotyping in health care; Spirituality and health; Assessment in health care settings; Complimentary and alternative medicine; African American health; Asian Health; Latino health; Native American health.

Author Biography

Robert M. Huff, MPH, PhD is an Emeritus Professor of Public Health Education at California State University, Northridge. His research interests blend his training in anthropology, public health, and confluent education to focus on multicultural health promotion and disease prevention programming in a variety of health care settings.

References

- Cavender, A. (2003). *Folk medicine in Southern Appalachia*. Chapel Hill and London: The University of North Carolina Press.
- Congress, E. P., & Lyons, B. P. (1992). Cultural differences in health beliefs: Implications for social work practice in health care settings. *Social Work in Health Care*, 17(3), 81–96.
- DeStefano, A. M. (2001). *Latino folk medicine*. New York, NY: Ballantine Books.
- Eliade, M. (1964). *Shamanism: Archaic techniques of ecstasy*. Princeton, NJ: Princeton University Press.
- Fadiman, A. (1997). *The spirit catches you and you fall down*. New York, NY: Farrar, Straus & Giroux.
- Haskins, J. (1990). *Voodoo and hoodoo: The craft as revealed by traditional practitioners*. New York, NY: Scarborough House.
- Helman, C. G. (2007). *Culture, health and illness* (5th ed.). London: Hodder Arnold.
- Hodge, F. S., & Hodge, C. E. (2015). Health and disease of American Indian and Alaska native populations. In R. M. Huff, M. V. Kline, & D. V. Peterson (Eds.), *Health promotion in multicultural populations: A handbook for practitioners and students* (3rd ed., pp. 270–291). Thousand Oaks, CA: Sage Publications, Inc.
- Hopp, J. W., & Herring, R. P. (2015). Promoting health among black Americans. In R. M. Huff, M. V. Kline, & D. V. Peterson (Eds.), *Health promotion in multicultural populations: A handbook for practitioners and students* (3rd ed., pp. 238–269). Thousand Oaks, CA: Sage Publications, Inc.
- Huff, R. M. (2002). Shamanic healing. In *The encyclopedia of public health* (Vol. 2). Farmington Hills, MI: The Gale Group Inc.
- Huff, R. M., Kline, M. V., & Dobbins, D. V. (2015). The cultural assessment framework. In R. M. Huff, M. V. Kline, & D. V. Peterson (Eds.), *Health promotion in multicultural cultural populations: A handbook for practitioners and students* (3rd ed., pp. 127–149). Thousand Oaks, CA: Sage Publications, Inc.
- Huff, R. M., Kline, M. V., & Peterson, D. V. (2015). *Health promotion in multicultural populations: a handbook for practitioners and students*. Los Angeles: SAGE.
- Jarvis, D. C. (1996). *Folk medicine: An almanac of natural health care*. New York, NY: Galahad Books.
- Kagawa-Singer, M., & Kho, C. (2015). Asian American health and disease. In R. M. Huff, M. V. Kline, & D. V. Peterson (Eds.), *Health promotion in multicultural populations: A handbook for practitioners and students* (3rd ed., pp. 292–332). Thousand Oaks, CA: Sage Publications, Inc.
- Kleinman, A. (1980). *Patients and healers in the context of culture*. Berkeley: University of California Press.
- Kleinman, A., & Benson, P. (2006, October). Anthropology in the clinic: The problem of cultural competency and how to fix it. *PLoS Medicine*, 3(10), 1673–1676.
- Mitchem, S. Y. (2007). *African American folk healing*. New York and London: New York University Press.
- Ortiz, B. L., Shields, K. M., Clauson, K. A., & Clay, P. G. (2007). Complimentary and alternative medicine use among hispanics in the United States. *The Annals of Pharmacotherapy*, 41(6), 994–1004. Retrieved from http://www.medscape.com/viewarticle/557983_print
- Rhoades, E. R. (2009). The Indian health service and traditional medicine. *Virtual Mentor*, 11(10), 793–798. Retrieved from <http://journalofethics.ama-assn.org/2009/10/mhst1-0910.html>
- Spector, R. E. (2013). *Cultural diversity in health and illness* (8th ed.). Upper Saddle River, NJ: Pearson Education.
- World Health Organization. (n.d.). *Traditional medicine: Definitions*. Retrieved 2016 from <http://www.who.int/medicines/areas/traditional/definitions/en/>.

Suggested Reading

- Comas-Diaz, L. (2012). *Multicultural care: A clinician's guide to cultural competence*. Washington, DC: American Psychological Association.
- Heinerman, J. (2001). *Folk medicine in America today: A guide for a new generation of folk healers*. New York, NY: Kensington Publishing Corp.
- Sandner, D. F., & Wong, S. H. (1997). *The sacred heritage: The influence of shamanism on analytical psychology*. New York, NY: Routledge.

Training in Health Psychology

Kevin T. Larkin

Department of Psychology, West Virginia University, Morgantown, WV, USA

Shortly after the Division of Health Psychology was recognized by the American Psychological Association (APA) in 1978 and the parameters for the specialty area defined (Matarazzo, 1980), the founding leaders of this emerging area of professional practice met to discuss and formulate training guidelines for those who desired to pursue careers as health psychologists. From its inception, there was a recognition that those psychologists desiring careers involving research and/or practice of health psychology needed to acquire competencies beyond those typically trained in existing doctoral programs. Founded on the biopsychosocial model of health (Engel, 1977), it was obvious to early leaders that additional competencies were required for psychologists who desired to function as health psychologists. Although knowledge of psychological theories and skills was a regular component of doctoral training programs in psychology for decades, expertise in the biological and social foundations of health was sorely absent in most doctoral programs training psychologists in the 1980s. To remedy this problem and develop training guidelines, 57 psychologists with a commitment to the emerging specialty of health psychology joined together to participate in the National Working Conference on Education and Training in Health Psychology held in May of 1983 at Arden House in Harriman, New York.

The Arden House Conference

The National Working Conference on Education and Training in Health Psychology, commonly referred to as the Arden House Conference, was a 4-day meeting that was funded largely through the support of the Carnegie Foundation, the MacArthur Foundation, and the Kaiser Family Foundation. The Conference was chaired by Stephen M. Weiss, one of the early leaders of the specialty of health psychology. Among the highlights from Arden House was the recognition that there were two types of training programs comprising the specialty area, scientist and scientist-practitioner (Miller, 1983; for comprehensive coverage of the Arden

House Conference, see Stone et al., 1987). Both types of programs were based upon the biopsychosocial model of health that ensured broad-based training of the science regarding biological, psychological, and social determinants of health and health behaviors and the development of research skills that fostered discovery of new findings. Trainees emerging from scientist programs (or what are now called health psychology training programs) were prepared to launch research careers aimed at advancing the understanding of the multiplicity of factors that influence health and the treatment of health conditions. In contrast, trainees graduating from scientist-practitioner programs (or what are now called clinical health psychology training programs) received training in the application of this knowledge with patients seen in healthcare settings as well as acquiring the foundational research competencies. In this regard, a hierarchical model of competency acquisition was outlined, with (a) fundamental knowledge of psychological principles and theories commonly taught in existing doctoral programs at the base; (b) knowledge of biological, psychological, and social determinants of health and research skills to discover new information comprising the next level required for those entering the workforce as health psychologists; and (c) additional clinical skills to enable the competent application of the knowledge base of health psychology into healthcare practice at the final level for trainees with aims of becoming clinical health psychologists.

Although many recommendations voiced by participants of the Arden House Conference regarding the distinctive set of competencies associated with the specialty were adopted by consensus, one area of disagreement among meeting participants pertained to the level of training required to apply evidence-based training with patients seen in healthcare settings. On the one hand, it was argued that due to the vast amount of knowledge required for the competent practice of clinical health psychology, only doctoral psychologists should be credentialed to function as clinical health psychologists. In contrast, it was argued that properly trained behavioral healthcare providers from other training backgrounds could be credentialed to assess and treat patients presenting with specific healthcare problems to meet what would certainly be a rapidly expanding demand for these services. Indeed, the limited number of training programs available to train clinical health psychologists who delivered competent behavioral healthcare services has long been recognized as a problem for a healthcare system that claims to treat and prevent death and disability largely caused by problems humans have in developing and maintaining proper health behaviors (e.g., eating balanced diets, engaging in regular exercise, using safe sexual practices, moderating alcohol use, etc.).

Rather than meeting on a single occasion to devise and adopt recommendations regarding the training of health and clinical health psychologists, the Arden House participants envisioned the creation of a vehicle to promote the ongoing dialogue regarding education and training in health psychology that would monitor training guidelines that would certainly need to adapt to the changing healthcare environment. The body constructed to ensure this ongoing conversation was the Council of Health Psychology that was a nonprofit organization composed of training programs in health psychology at doctoral, internship, and postdoctoral levels. Representatives from these training programs met annually to discuss relevant issues regarding training the next generation of health and clinical health psychologists for over a decade before falling into a state of dormancy by the end of the twentieth century.

The Tempe Summit on Education and Training in Clinical Health Psychology

Recognizing the important role of behavioral factors in the etiology of many diseases and sources of disability, the APA redefined itself as a health profession (Anderson, 2003) by voting

to add the objective “promoting health” to its vision statement. This event coincidentally occurred as the importance of behavioral factors in the etiology, assessment, diagnosis, and treatment of a broad range of diseases and disorders was being recognized by professions long affiliated with healthcare (e.g., medicine, dentistry, nursing, etc.). As healthcare systems looked to psychology to assist in managing the behaviors at the root of most of the problems seen regularly in their clinics and hospitals, inadequate numbers of clinical health psychologists were readily available, so the healthcare system turned to other types of psychologists and then to other related professions (e.g., social work, health counselors) to provide behavioral healthcare in these medical settings. Unfortunately, many of these individuals lacked the requisite knowledge of the biological, behavioral, and social factors that influenced health and health outcomes and the clinical-applied competencies for functioning within the healthcare environment. Even those trained in the top doctoral clinical or counseling psychology programs learned that they lacked the essential competencies to meet the unique demands encountered in medical settings. It became quickly apparent that some additional work needed to be conducted to describe the parameters of knowledge and specify the skills required to identify as a clinical health psychologist capable of functioning competently within the modern healthcare environment.

Based upon the foundational work conducted at the Arden House Conference and the growing focus on articulating competencies within the broader profession of psychology, Division 38 (Health Psychology) of the APA planned and held the Tempe Summit on Education and Training in Clinical Health Psychology in 2007 (see France et al., 2008). The primary goal of this meeting of 20 leaders from the education and training community in clinical health psychology was to articulate the competencies required for entry-level practice of clinical health psychology in six areas: (a) assessment/diagnosis/case conceptualization, (b) intervention, (c) research/evaluation, (d) consultation, (e) supervision/teaching, and (f) management/administration. It is beyond the scope of this chapter to enumerate all of the competencies on the list here, but the interested reader is referred to France et al. (2008) for a depiction of the initial list and to Larkin and Klonoff (2014) for the most recent iteration of it. Representative examples of some of the competencies of clinical health psychologists that distinguish them from other types of practicing psychologists are shown in Table 1.

A quick perusal of Table 1 easily reveals some of the unique attributes of the modern healthcare environment in which health and clinical health psychologists operate. Unfortunately, many of these competencies are not acquired in traditional doctoral training programs and internships where mental health issues are given more attention than health problems generated by other organ systems in the body. Conversations among Tempe Summit participants acknowledged that the primary distinction between health and clinical health psychology was in the extensive list of competencies comprising the applications cluster (i.e., assessment, intervention, and consultation) and knowledge and skills essential for the practice of clinical health psychology, but not required for functioning as health psychologists.

An additional outcome of the Tempe Summit was the reestablishment of an active training council. In order to ensure that the next generation of clinical health psychologists acquired the requisite competencies for functioning in the rapidly evolving healthcare system, the training community recognized that they had to commit to meeting annually to keep up with new developments in medicine and healthcare. The Council of Health Psychology established at Arden House was revived, given a new name, and established a primary goal to ensure that behavioral healthcare providers identified as clinical health psychologists possessed all of the competencies required to function effectively in the changing healthcare environment. Composed of directors of all training programs in clinical health psychology across doctoral, internship, and postdoctoral fellowship programs, the Council of Clinical Health Psychology Training Programs (CCHPTP) (acronym pronounced “chip-tip”) was incorporated in 2007.

Table 1 Examples of some of the distinctive competencies of clinical health psychologists.

<i>Competency cluster</i>	<i>Clinical health psychology competency</i>	<i>Example of means of demonstrating competence</i>
Science	Knowledge of pathophysiology of disease and biomedical treatments specific to medical specialty or environment in which the practice will occur*	Earning an A or B in a human physiology class
Professionalism	Identification and ability to address the distinctive ethical issues encountered in clinical health practice, particularly if these are in conflict with the ethical code of other members of the healthcare team**	Ability to discuss ethical issues pertaining to making end-of-life decisions and/or determining capacity to consent for medical treatments
Relationships	Development of facilitative and collaborative relationships with professionals from a variety of healthcare disciplines including medicine, nursing, physical therapy, social work, etc.*	Completion of practice in which trainees interact regularly with other healthcare professionals
Applications	Ability to evaluate the presenting problem and to select and administer empirically supported biopsychosocial assessments appropriate for the patient's physical illness, injury, or disability*	Completion of practice in a hospital, clinic, or other healthcare setting
Education	Supervision of clinical health psychology skills, conceptualizations, and interventions for psychologists, psychology trainees, and behavioral health providers from other health professions*	Experience supervising others with less training in health psychology, including those in other disciplines
Systems	Leadership within an interprofessional team or organization in the healthcare setting*	Demonstration of effective leadership skills during research or clinical team meetings

*Adapted from Competencies in Clinical Health Psychology. France et al. (2008).

**Adapted from Competencies for Psychology Practice in Primary Care. McDaniel et al. (2014).

The Council of Clinical Health Psychology Training Programs (CCHPTP)

Launched at the Tempe Summit, CCHPTP members have met twice annually for purposes of making sure that training programs in clinical health psychology are keeping up with the demands of the contemporary practice of clinical health psychology within the context of the modern healthcare environment. Based upon the spirit of collaboration and community observed at both Arden House and Tempe Summit conferences, CCHPTP sponsors working meetings that frequently result in published articles aimed at disseminating the most current information to the entire educational community (e.g., Kerns, Berry, Frantsve, & Linton, 2009; Larkin, 2009; Larkin, Bridges, Fields, & Vogel, 2016; Larkin & Klonoff, 2014; Masters, France, & Thorn, 2009; Nash & Larkin, 2012; Nash, McKay, Vogel, & Masters, 2012; Nicholas & Stern, 2011). Early CCHPTP meetings focused on refining the core list of competencies articulated at the Tempe Summit, but more recent meetings have focused on other extensions of education and training in this specialty area, including the enumeration of the distinctive competencies required for working in primary care medical settings, where flexibility is essential and rapid pace of healthcare is the norm.

Most recently, CCHPTP has focused on establishing a standard method for describing the range of clinical health psychology training programs that exist among doctoral, internship, and postdoctoral levels of training. By addressing this issue, prospective learners have informed consent regarding the depth and breadth of training in clinical health psychology to assure that the designated program matches their desired training objectives. Using the structural framework of the *Taxonomy for Education and Training in Professional Psychology Health Service Specialties* (APA, 2012), CCHPTP outlined criteria for academic programs at four levels of training (doctoral, internship, postdoctoral, and post-licensure) for designation as (a) a major area of study (e.g., an internship in which 50% of supervised practice is in clinical health psychology), (b) an emphasis (e.g., a doctoral program that offers two courses and two practicum experiences in clinical health psychology), (c) an experience (e.g., a doctoral program that offers one course and one practicum in clinical health psychology, and (d) exposure to the specialty area (e.g., 10–20% of a postdoctoral program is in clinical health psychology). The complete taxonomy of clinical health psychology training programs is available on the CCHPTP website (see <http://cchptp.org>).

The Specialization of Clinical Health Psychology

Periodically, specialty areas are reviewed by the Commission for the Recognition of Specialties and Proficiencies in Professional Psychology (CRSPPP) of the APA to ensure that they continue to meet the requirement of providing advanced, specialty-specific scientific knowledge. Clinical health psychology last underwent this scrutiny in 2011, and its status as a specialty was reconfirmed (see APA, 2011). Part of the documentation for the redesignation as a specialty area requires that the area state whether specialty training occurs at the doctoral level, the postdoctoral level, or both. Although the specialty practice of psychology in most areas was historically conducted during postdoctoral training, the specialization of clinical health psychology reported in their petition that training was now done at both levels. In full recognition of the growth of specialization within the profession of psychology, trainees interested in pursuing advanced, specialty-specific training now start acquiring specialty competencies during their doctoral training years alongside the acquisition of broad and general competency acquisition (Nash & Larkin, 2012). In the past, students had opportunities to secure internships and postdoctoral fellowships in clinical health psychology with very little exposure to the specialty area during their doctoral training years. This is no longer the case. Entrance into most accredited internships and postdoctoral training programs in clinical health psychology is so competitive that some exposure and experience in clinical health psychology is almost a requirement for advanced specialty training. To fulfill the need to produce trainees who had acquired some specialty competencies during their doctoral training years, more doctoral programs began offering tracks or areas of emphasis in specialty areas, and clinical health psychology or its related discipline of behavioral medicine is one of the most popular tracks among all accredited doctoral programs (Perry & Boccaccini, 2009).

Although the specialty area of clinical health psychology acknowledges that training is conducted at both doctoral and postdoctoral levels, accreditation of clinical health psychology programs is only recognized for postdoctoral programs in the specialty area. This is largely done because most of the doctoral programs that offer opportunities to acquire specialty competencies in clinical health psychology are embedded in existing clinical or counseling psychology training programs (Larkin, 2009). As such, some, but not all, trainees in these programs acquire specialty-specific competencies in clinical health psychology. That said, there

are now several programs that exclusively train clinical health psychology students at the doctoral level of training and ensure that all graduates of these programs acquire coursework, practicum, and research experiences relevant to clinical health psychology. This same state of affairs exists at the internship level, where over 500 internship sites provide rotations in clinical health psychology (Masters, France, & Thorn, 2009).

The foundational work on articulating entry-level competencies of the clinical health psychologist was cited prominently in the petition to CRSPPP for re-recognition as a specialty area. Most formidably, competencies in conducting research were highlighted as credentials of clinical health psychologists that make them stand out among all other professions represented in the modern healthcare environment. As such, clinical health psychologists become natural leaders of interdisciplinary research programs and quality improvement evaluation efforts. The fact that the Affordable Care Act, once fully implemented, will require demonstration of positive treatment outcomes in order to obtain optimal compensation for services will position clinical health psychologists as indispensable members of staffs of academic medical centers and hospitals.

Expansion of Clinical Health Psychology Specialty Training

It is clearly recognized that expertise in behavioral healthcare is highly valued in the modern healthcare arena. The extent to which these services will be provided and/or supervised by specialists in clinical health psychology has yet to be known. The dilemma confronting Arden House participants, whether adequate numbers of doctoral clinical health psychologists to provide these services exist, continues to confront the field. Naturally, cost-benefit calculations also enter into these equations as healthcare organizations consider the value of hiring doctoral level clinical health psychologists. In this regard, it is clear that clinical health psychologists of the future will not be hired only as frontline behavioral healthcare providers. Their value becomes truly recognized through their extensive training in research, consultation, team management, and clinical supervision.

Regardless of how specific hospitals and healthcare systems resolve the workforce decisions associated with hiring doctoral level clinical health psychologist and their research-oriented health psychology peers, the domain of health psychology has been expanding. For example, pediatric psychology reflects a somewhat newer specialty area that certainly has its roots in clinical health psychology and those psychologists who chose to provide behavioral healthcare and conduct research in pediatric clinics. Likewise, early clinical neuropsychologists applied their solid behavioral assessment skills acquired in traditional clinical or counseling training programs with patients visiting neurology clinics and rehabilitation hospitals. More recently, considerable attention has been given to psychologists who integrate the provision of behavioral healthcare and practice-based research into the daily medical practice in primary care settings (e.g., McDaniel et al., 2014; Nash et al., 2012). The unique set of interdisciplinary systems, practice management, and supervisory skills with which clinical health psychologists are equipped makes them highly competitive for these types of positions. As the field becomes more specialized, even more subspecialty areas that have their roots in clinical health psychology can be envisioned (e.g., psycho-oncology, cardiac psychology, pain psychology).

Although the addition of health promotion to APA's vision statement accurately acknowledged the fact that mind and body are interconnected, it has, in some respects, blurred the distinction between clinical health psychology and other types of professional psychology. Nowhere is this more apparent in the decision to replace the term "professional psychology" with "health service psychology" (Health Service Psychology Education Collaborative, 2013).

Health service psychology should not be confused with clinical health psychology. Health service psychology refers to psychologists who are licensed to provide services within the scope of their competencies and to offer and bill for these services through various health plans. In this regard, a clinical health psychologist is one type of health service psychologist. The inverse statement, however, is not true. Not all health service psychologists possess the competencies to practice as clinical health psychologists and should not attempt to practice beyond the scope of their competencies. To the extent that the enumeration of clinical health psychology competencies has informed the construction of the competencies of all health service providers, it has become clear that all practicing psychologists, clinical health psychologists included, will need to learn more about interdisciplinary healthcare provision, biological diseases and treatments that influence human functioning, and practice management within healthcare systems.

Clinical health psychologists are well positioned to play critical roles in the healthcare teams of the future. They possess the knowledge of behavior change that should be at the root of almost all treatment plans of patients seeking care from all health professionals today. They are uniquely suited for developing and testing interventions in healthcare settings, training and supervising those who use them, leading interdisciplinary research teams, and consulting with other members of the healthcare teams regarding patients with complicated medical presentations. Educators comprising professional training programs have long been committed to working with trainees to ensure that the next generation of clinical health psychologists possess the competencies needed to practice effectively in the modern healthcare arena.

See Also

Biopsychosocial Practice; History of Clinical Health Psychology; Integrated Primary Care; Interprofessional Development and Competencies; Training Non-Psychologists in the Health Care Setting

Author Biography

Kevin T. Larkin, PhD, ABPP, is a professor and department chair in the Department of Psychology at West Virginia University. As a board certified clinical health psychologist, he also holds an appointment in the Department of Behavioral Medicine and Psychiatry at the Robert C. Byrd Health Sciences Center in Morgantown, WV. His primary works of scholarship focus on applied psychophysiology and cardiovascular behavioral medicine. He served as the chair of CCHPTP during its first 6 years and has written extensively on the training and education of clinical health psychologists.

References

- American Psychological Association. (2011). Petition for the recognition of a specialty in professional psychology for clinical health psychology. Minutes from the *Annual Report of the Commission for the Recognition of Specialties and Proficiencies in Professional Psychology (CRSPPP)*. Washington, DC: American Psychological Association.
- American Psychological Association. (2012). *Education and training guidelines: A taxonomy for education and training in professional psychology health service specialties*. Retrieved from <http://www.apa.org/ed/graduate/specialize/taxonomy.pdf>

- Anderson, N. B. (2003). Psychology as a health profession. *The APA Monitor on Psychology*, 34, 9.
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196, 129–136.
- France, C. R., Masters, K. S., Belar, C. D., Kerns, R. D., Klonoff, E. A., Larkin, K. T., ... Thorn, B. E. (2008). Application of the competency model to clinical health psychology. *Professional Psychology: Research and Practice*, 39, 573–580. doi:10.1037/0735-7028.39.6.573
- Health Service Psychology Education Collaborative. (2013). *A blueprint for health service psychology education and training*. Washington, DC: American Psychological Association.
- Kerns, R. D., Berry, S. S., Frantsve, L. M. E., & Linton, J. C. (2009). Life-long competency development in clinical health psychology. *Training and Education in Professional Psychology*, 3, 212–217. doi:10.1037/a0016753
- Larkin, K. T. (2009). Variations of doctoral training programs in clinical health psychology: Lessons learned at the box office. *Training and Education in Professional Psychology*, 3, 202–211. doi:10.1037/a0016666
- Larkin, K. T., Bridges, A. J., Fields, S. A., & Vogel, M. E. (2016). Acquiring competencies in integrated behavioral health care in doctoral, internship, and post-doctoral programs. *Training and Education in Professional Psychology*, 10, 14–23. doi:10.1037/tep0000099
- Larkin, K. T., & Klonoff, E. A. (2014). *Specialty competencies in clinical health psychology*. New York, NY: Oxford University Press.
- Masters, K. S., France, C. R., & Thorn, B. E. (2009). Enhancing preparation among entry-level clinical health psychologists: Recommendations for “best practices” from the first meeting of the Council of Clinical Health Psychology Training Programs (CCHPTP). *Training and Education in Professional Psychology*, 3, 193–201. doi:10.1037/a00116049
- Matarazzo, J. D. (1980). Behavioral health and behavioral medicine: Frontiers for a new health psychology. *American Psychologist*, 35, 807–817. doi:10.1037/0003-066X.35.9.807
- McDaniel, S. H., Grus, C. L., Cubic, B. A., Hunter, C. L., Kearney, L. K., Schuman, C. C., ... Johnson, S. B. (2014). Competencies for psychology practice in primary care. *American Psychologist*, 69, 409–429. doi:10.1037/a0036072
- Miller, N. E. (1983). Some main themes and highlights of the conference. *Health Psychology*, 2, 11–14.
- Nash, J. M., & Larkin, K. T. (2012). Geometric models of competency development in specialty areas of professional psychology. *Training & Education in Professional Psychology*, 6, 37–46. doi:10.1037/a0026964
- Nash, J. M., McKay, K. M., Vogel, M. E., & Masters, K. S. (2012). Functional and foundational characteristics of psychologists in integrated primary care. *Journal of Clinical Psychology in Medical Settings*, 19, 93–104. doi:10.1007/s10880-011-9290-z
- Nicholas, D. R., & Stern, M. (2011). Counseling psychology in clinical health psychology: The impact of specialty perspective. *Professional Psychology: Research and Practice*, 42, 331–337. doi:10.1037/a0024197
- Perry, K. M., & Boccaccini, M. T. (2009). Specialized training in APA-approved clinical psychology doctoral programs: Findings from a review of program websites. *Clinical Psychology: Science and Practice*, 16, 348–369. doi:10.1111/j.1468-2850.2009.01173.x
- Stone, G. C., Weiss, S. M., Matarazzo, J. D., Miller, N. E., Rodin, J., Belar, C. D., ... Singer, J. E. (Eds.). (1987). *Health psychology: A discipline and a profession*. London: The University of Chicago Press.

Suggested Reading

- Belar, C. D. (2008). Clinical health psychology: A health care specialty in professional psychology. *Professional Psychology: Research and Practice*, 39, 229–233. doi:10.1037/0735-7028.39.2.229
- Belar, C. D., & Deardorff, W. W. (2009). *Clinical health psychology in medical settings: A practitioner's guidebook* (2nd ed.). Washington, DC: American Psychological Association.

Training Non-psychologists in the Healthcare Settings

Cesar A. Gonzalez¹, Kristine M. Diaz², and
Richard J. Seime¹

¹*Mayo Clinic Minnesota, Rochester, MN, USA*

²*Walter Reed National Military Medical Center, Bethesda, MD, USA*

Historical Context of Psychology in the Healthcare Setting

In the United States, psychology first began to hold a prominent role in healthcare settings around 1945 through the development of the Department of Veterans Affairs' psychology program. The inclusion of psychologists into the Veterans Affairs promoted the advancement of multisite and interdisciplinary research and facilitated the inclusion of behavioral treatments in healthcare settings. In 1977, George L. Engel published *The Need for a New Medical Model: A Challenge for Biomedicine* and introduced the biopsychosocial model into medicine, which, in addition to the biological determinants of disease, also included the psychological and social determinants in the contribution to the development and progression of disease (Engel, 1977).

The World Health Organization describes determinants of health as being the context in which individuals are born, develop, live, work, and age that influence the shape and trajectory of health and mortality (WHO, 2010). An individual's behavioral and sociocultural context is estimated to account for 10–50% of the cause of chronic diseases and early mortality. Health psychology uses the scientific method to generate and apply knowledge related to the interplay between the determinants of disease in the biopsychosocial model in order to prevent, treat, rehabilitate, and maintain the health and well-being of individuals, communities, and populations. Since the 1990s, in the United States, there has been a movement toward integrated care characterized by colocation and deliberate collaboration among healthcare professionals with the goal of integrating the biopsychosocial model into patient care to optimize the trajectory of care and health.

Given the rapidly changing healthcare system, psychologists are uniquely positioned to serve as behavioral health experts; health educators and consultants; and researchers across primary, secondary, and tertiary healthcare settings. In addition to training licensed health professionals in these settings, psychologists train undergraduate, graduate, and postgraduate learners.

Psychologist as Educators With Non-psychologists

While psychologists hold specialized training to provide direct patient care, the role of the psychologist in healthcare settings is likely to shift to one that is focused on the delivery of training and supervision for other healthcare professionals. In 2014, the “Competencies for Psychology Practice in Primary Care” published in the *American Psychologist* (McDaniel et al., 2014) identified teaching and supervision as a competency of psychologists in these settings, serving as educators in faculty and staff development as well as educating and supervising health trainees. Working with various health professionals, finding a common language and focus contributes to successful education and training goals with non-psychologists.

Family medicine is one field of medicine that has demonstrated success in the integration of psychologists as educators with non-psychologists in primary care, utilizing accreditation standards in the provision of behavioral health in primary care. With identified core competencies and training milestones, emerging physicians, attending physician faculty members, and allied health providers are able to treat patients with complex medical histories. In fact, the triumph of the integration of psychology into family medicine serves as an educational model for psychologists working with non-psychologists. Accreditation-driven graduate medical education in family medicine has contributed to a system-based approach to the inclusion of psychologists in traditionally physician-dominated areas of medicine, and family medicine is one of the first specialty areas of medicine to demonstrate interprofessional collaborative clinical practice.

With the innovation of family medicine to embed educational requirements of non-psychologists in their field through accreditation standards, the field has created an inclusive learning environment, building a common language for the sustainability of psychology as integral to the practice of family medicine.

Enhancement of Learning Environment in the Education of Non-psychologists

The American Psychological Association published the *2015 Survey of Psychology Health Service Providers*, which suggested that over half of psychologists engage in collaboration with at least four different healthcare professionals: 60% of psychologists reported collaboration with physicians and surgeons, 50% reported collaboration with nurse practitioners, and 45% reported collaboration with nurses. Moreover, the survey found that approximately 24% of psychologists in the United States identified their primary work setting to be in a hospital setting or organized human service settings (American Psychological Association, 2015).

In 2016, the US-based Interprofessional Education Collaborative, which is inclusive of 60 health professional fields, identified four fundamental domains and competencies that aim

to promote interprofessional collaboration in order to improve individual and population health outcomes:

- 1 *Values/Ethics for Interprofessional Practice*: Work with individuals of other professions to maintain a climate of mutual respect and shared values.
- 2 *Clarity around Roles/Responsibilities*: Use the knowledge of one's own role and those of other professions to appropriately assess and address the health care needs of patients and to promote and advance the health of populations.
- 3 *Interprofessional Communication*: Communicate with patients, families, communities, and professionals in healthcare.
- 4 *Engage in Teams and Teamwork*: Apply relationship-building values and the principles of team dynamics to perform effectively in different team roles to plan, deliver, and evaluate patient/population-centered care and population health programs and policies that are safe, timely, efficient, effective, and equitable. With the increase in team-based interprofessional healthcare, psychologists' skills are well matched to facilitate and deliver the core competencies across healthcare settings and disciplines (Interprofessional Education Collaborative, 2016).

The expanding role of psychologists within the healthcare systems demands standards of practice and guidelines for the integration of behavioral health. However, there remain limited resources for using a common set of competencies in the integration of behavioral health into integrated care. To address this gap, the Substance Abuse and Mental Health Services Administration and the Health Resources and Services Administration Center for Integrated Health Solutions (CIHS) developed the *Core Competencies for Integrated Behavioral Health and Primary Care* as a resource for integrated practice relevant to behavioral health and primary care providers (Hoge et al., 2014). CIHS seeks to advocate for the advancement in the treatment of patients with mental health and substance use issues in either integrated primary or behavioral health settings. Published in January 2014, the *Core Competencies for Integrated Behavioral Health and Primary* serves as a resource for educators in curriculum development and training in integrated care using the following competencies: (a) interpersonal communication, (b) collaboration and teamwork, (c) screening and assessment, (d) care planning and care coordination, (e) intervention, (f) cultural competence and adaptation, (g) systems-oriented practice, (h) practice-based learning and quality improvement, and (i) informatics. This framework serves as an opportunity to unite all health providers under a shared mission and values.

Behavioral anchors allow the educator to identify specific educational topics and medical knowledge to address in curriculum development. The Institute of Medicine (IOM) identified a lack of educational resources to identify relevant behavioral and social science curriculum content, instructional strategies, and educational research in undergraduate medical education (Institute of Medicine [US] Committee on Behavioral and Social Sciences in Medical School Curricula, 2004). Thus, IOM identified 26 educational topics in the following 6 behavioral and social science knowledge domains in *Improving Medical Education: Enhancing the Behavioral and Social Science Content of Medical School Curricula*: (a) mind–body interactions in health and disease, (b) patient behavior, (c) physician role and behavior, (d) physician–patient interaction, (e) social and cultural issues in healthcare, and (f) health policy and economics. The use of behavioral anchors (or short-term objectives) will lead to an increase in positive outcomes in obtaining long-term training goals (achieve core competencies).

The use of a comprehensive behavioral and social science curriculum and core competencies in the education and training of non-psychologists serves an opportunity to highlight the psychologist's expertise. The additional role as an educator within healthcare will demonstrate the utility of a psychologist in the education and training of non-psychologists in integrated care. Education and training to further one's competency in behavioral and social science curriculum development and interprofessional education exists in various medical specialty areas and health psychology.

Conclusion

The ever-changing culture of healthcare has contributed to an evolving role of psychologists in medicine. Psychologists historically have served a dual role within healthcare systems providing clinical education and training and psychological services. The current emphasis of behavioral health integration in primary care has contributed to psychologists' responsibilities for integrating mental health, substance abuse, and health behavior change to meet the community's needs. Within the context of integrated care, there are resources available to help psychologists translate their psychology expertise into obtainable educational learning goals for non-psychologists, furthering the integration of psychology into medicine.

See Also

Biopsychosocial model; History of Clinical Health Psychology; History of Behavioral Medicine/Health Psychology; Integrated primary care; Interprofessional development and competencies; Training in health psychology.

Author Biographies

Cesar A. Gonzalez, PhD, LP, ABPP, is an assistant professor of psychology and family medicine at Mayo Clinic; he is board certified in clinical psychology by the American Board of Professional Psychology and practices in the areas of primary care psychology and transgender health. He is an associate program director of the Family Medicine Residency at Mayo Clinic; in this role he promotes behavioral science and interprofessional education and practice into the family medicine residency curriculum and faculty development.

Kristine M. Diaz, PsyD, is a behavioral scientist with professional and clinical experience in primary care, community mental health, and hospital, VA, and corrections settings. Dr. Diaz has provided curriculum development in behavioral science in both undergraduate and graduate medical education. Her research focuses in medical education, specifically LGBTY health and behavioral science education.

Richard J. Seime, PhD, LP, ABPP, is board certified in clinical health psychology and clinical psychology by the American Board of Professional Psychology. He is an emeritus consultant at Mayo Clinic in the Department of Psychiatry and Psychology and emeritus associate professor of psychology in the Mayo Clinic College of Medicine and Science. His practice in academic health centers has spanned primary care psychology and consultative specialty care. He is chair of the Clinical Health Psychology Specialty Council, president of Council of Specialties in Professional Psychology, and past president of the American Board of Clinical Health

Psychology. A former president of the Association of Psychologists in Academic Health Centers, he was recipient of the APAHC 2011 Ivan Mensh Award for Distinguished Teaching. He is a fellow of the American Psychological Association, the Society of Clinical Psychology, and Society for Health Psychology.

References

- American Psychological Association. (2015). *2015 APA survey of psychology health service providers*. Washington, DC: Author. Retrieved from <http://www.apa.org/workforce/publications/15-health-service-providers/index.aspx?tab=1>
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196, 129–136.
- Hoge, M. A., Morris, J. A., Laraia, M., McManis, A., Pomerantz, A., & Farley, T. (2014). *Core competencies for integrated behavioral health and primary care*. Washington, DC: SAMHSA—HRSA Center for Integrated Health Solutions. Retrieved from http://www.integration.samhsa.gov/workforce/Integration_Competencies_Final.pdf
- Institute of Medicine (US) Committee on Behavioral and Social Sciences in Medical School. Curricula. (2004). In P. A. Cuff & N. Vanselow (Eds.), *Improving medical education: Enhancing the behavioral and social science content of medical school curricula*. Washington, DC: National Academies Press (US). Retrieved from <https://www.nap.edu/catalog/10956/improving-medical-education-enhancing-the-behavioral-and-social-science-content>
- Interprofessional Education Collaborative. (2016). *Core competencies for interprofessional collaborative practice: 2016 update*. Washington, DC: Author. Retrieved from <http://www.aacn.nche.edu/education-resources/IPEC-2016-Updated-Core-Competencies-Report.pdf>
- McDaniel, S. H., Grus, C. L., Cubic, B. A., Hunter, C. L., Kearney, L. K., Schuman, C. C., ... Johnson, S. B. (2014). Competencies for psychology practice in primary care. *American Psychologist*, 69, 409–429. doi:10.1057/a0036072
- World Health Organization. (2010). *Framework for action on interprofessional education and collaborative practice*. Geneva, Switzerland: WHO Press. Retrieved from http://whqlibdoc.who.int/hq/2010/WHO_HRH_HP_N_10.3_eng.pdf

Suggested Reading

- Behavioral science in family medicine residencies. : Part I. Teachers and curricula. The STFM task force on behavioral science. (1985). *Family Medicine*, 17(2), 64–69.
- Behavioral science in family medicine residencies. : Part II. Teacher roles, relationships, and rewards. The STFM task force on behavioral science. (1985). *Family Medicine*, 17(2), 69–73.
- World Health Organization and World Organization of Family Doctors. (2008). *Integrating mental health into primary care: A global perspective*. Geneva, Switzerland: WHO Press. Retrieved from http://www.who.int/mental_health/resources/mentalhealth_PHC_2008.pdf

Transgender Health

DeAnna L. Mori^{1,2}, Colleen A. Sloan^{1,2},
Heather Walton Flynn^{1,3}, and Claire Burgess^{1,2,3}

¹*VA Boston Healthcare System, West Roxbury, MA, United States*

²*Department of Psychiatry, Boston University School of Medicine, Boston, MA, USA*

³*Department of Psychiatry, Harvard Medical School, Boston, MA, USA*

Social Context

Within the past two decades, transgender lives and experiences have received more attention within sociocultural–political contexts and relatedly within healthcare. Transgender is a term used to describe people whose gender identity (sense of maleness, femaleness, or other non-binary identities) or gender expression (how one communicates their gender) differs from that typically associated with their sex assigned at birth, although not all people who have this experience will identify themselves as transgender. Regardless of how they label themselves, transgender people collectively have unique experiences and healthcare needs, which are greatly impacted by social context and issues of stigma. Stigma, defined as the shame or disgrace attached to something regarded as socially unacceptable, manifests both via negative attitudes and treatment toward transgender people and within transgender people via internalization of negative beliefs and feelings. These effects impact physical and mental health and contribute to well-documented health and healthcare disparities for transgender people. Moreover, some transgender people experience identity-specific distress (known as gender dysphoria) and may choose to alter their bodies and expressions to be more consistent with their identities, a process usually termed “transitioning” and which may also involve utilization of specific healthcare services. Of note, not all gender transitions involve medical interventions. Some transgender people may opt to socially transition by altering their gender expression and gender role to be more consistent with their self-identified gender identity. Regardless of the intervention or change employed, the goal is to reduce distress and to promote congruity with physical body and sense of self.

Understanding of transgender stigma and associated consequences within the larger social context is necessary to fully comprehend transgender health disparities. Stigma regarding transgender identities and experiences manifests systemically, at multiple levels—within legislation, media, communities, environments of care, and via interpersonal and intrapersonal processes—and therefore is experienced as chronic and pervasive. Consequently, transgender people are at risk of experiencing a specific type of chronic stress, termed *gender minority stress* (Hendricks & Testa, 2012), an extension of *minority stress* (Meyer, 2003). Such stress increases vulnerability to health problems, which are elevated within the transgender population. The US Transgender Survey (USTS) conducted in 2015 is the largest survey of transgender people in the United States, with over 27,000 participants in all 50 states and US territories (James et al., 2016). Survey respondents endorsed experiencing mistreatment, discrimination, harassment, and violence in all areas of life including housing, employment, education, personal/family life, health and healthcare, and experiences with the criminal justice system. Moreover, results revealed transgender individuals experienced mistreatment in these areas at a higher rate than cisgender (i.e., self-identified gender is congruent sex assigned at birth) individuals. One of the most alarming findings is the high rates of violence and victimization perpetrated by family, employers/coworkers, law enforcement officials, and even healthcare providers. Relatedly, results revealed elevated rates of unemployment and homelessness and decreased rates of health insurance coverage, all of which are compounded by absence of legal protections for transgender people in many of these areas. For example, anti-discrimination policies may or may not include gender identity as a protected component of identity. Lastly, one of the most notable findings from this survey is the strikingly high rates of suicide for transgender people. While the USTS only surveys transgender people, other sources have demonstrated that these suicide rates are significantly higher than rates for cisgender people. Collectively, these findings highlight the pervasiveness of transgender stigma and its profound impact on physical and mental health and general well-being. Additionally, these disparities highlight an apparent dilemma with respect to health and healthcare—limited to no access to healthcare may lead to solicitation of care that is of poor quality (e.g., traveling surgeons, “doc in the box”), which in turn contributes to poorer health.

Access to Healthcare

Regarding healthcare in particular, specific and unique disparities have been indicated for transgender people. According to USTS, roughly one-fourth reported having experienced at least one problem with their insurance related to transgender identity, with over 50% being denied for transition-specific surgical procedures and 25% being denied for hormone therapy. Additionally, over one-third of those who sought care from a healthcare professional during the past year reported having had a negative experience including refusal of care, verbal harassment, or experience of physical or sexual assault, all of which were higher for transgender people of color and/or with disabilities. Likewise, almost one-fourth of respondents reported avoiding healthcare due to fear of mistreatment based on transgender identity, which further perpetuates health disparities. Negative experiences notwithstanding, healthcare providers often do not have adequate education and training regarding transgender health, as this area is not reliably included in medical or mental health curricula. Inadequate education and training leads to lack of knowledge and competency, which in turn can negatively impact provision of care (or lead to lack of care). For example, transgender people often use pronouns that

differ from the ones typically associated with sex assigned at birth. When others refer to transgender people using incorrect pronouns, an example of “misgendering,” this may communicate both a lack of knowledge and a lack of respect, both of which negatively impact provision of care and general interaction. Relatedly, healthcare facilities rarely have visual cues and/or other materials (e.g., intake forms, reading material) that depict or communicate directly to the transgender population. These types of issues contribute to concern in transgender individuals about the availability of competent and respectful care and sometimes lead to avoidance of healthcare. Collectively these factors highlight the multifaceted nature of transgender disparities within healthcare.

Health-Related Issues

Transgender individuals are at increased risk for certain types of health-related problems, diseases, and mental health problems. This is largely impacted by the issues discussed above, including but not limited to minority stress, poor access to healthcare, and lack of culturally competent providers (Makadon, Mayer, Potter, & Goldhammer, 2015). The following are key health problems for which transgender individuals are at increased risk.

Alcohol and Substance Use Disorders

Results of observational studies in the United States show that alcohol and substance use (both marijuana and illicit substances) in this population is high. A recent empirical study found that gender dysphoria was significantly associated with excessive alcohol use for transgender women, cannabis use among both transgender women and men, and illicit (non-cannabis) drug use among transgender women (Gonzalez, Gallego, & Bockting, 2017). Gender minority stressors were independently associated with excessive alcohol use among transgender men and cannabis use among transgender women, suggesting that minority stress partially accounts for the increased use. Transgender individuals should be screened for problematic alcohol and drug use and should be referred to appropriate resources for achieving recovery and learning healthy coping skills.

Cancer

There is little information on cancer outcomes for transgender people because of an absence of large-scale prospective studies investigating cancer incidence and mortality in lesbian, gay, bisexual, and transgender people. Anecdotal evidence and case reports suggest that transgender individuals’ rates of cancer may be higher than that of the general population. Transgender men continue to have a risk of developing breast, ovarian, or uterine cancer if those parts of their body have not been removed. Similarly, transgender women still have a risk of prostate cancer. It is important to note that while transgender individuals should be treated and addressed according to their self-identified gender, they also need to receive screenings that align with their sex assigned at birth to detect any problems related to the anatomy with which they were born as well as any organs they have at present. These screenings may not occur because transgender individuals or their providers may not realize the need for them, because of problems with healthcare access, or due to discrimination. Having culturally competent, sensitive care available can help individuals realize the need for these screenings and address the barriers that often interfere with receiving this care.

Cardiovascular Disease

Transgender persons are often at higher risk for heart disease. Several factors may contribute to this disparity, including higher rates of tobacco use, obesity, and reduced physical activity. Transgender individuals should be encouraged to follow up with routine physical exams that include blood pressure and cholesterol checks and to engage in healthy behaviors that can protect against cardiovascular disease. Due to the increased risk, transgender individuals also should be educated about the signs and symptoms of heart disease and stroke.

Depression and Suicidality

Transgender individuals have higher rates of depression and anxiety compared with cisgender individuals, including an increased risk for suicide. These problems can be exacerbated in those who lack social support or feel unable to “come out” and express their gender identity. Transgender individuals should be screened for depression and suicidality and referred for treatment. Untreated depression can also interfere with engagement in healthy behaviors and medical care, which further contributes to the development of health problems and medical illness.

Sexually Transmitted Infections (STIs)

Transgender individuals, especially women of color, are at high risk for sexually transmitted infections (STIs), including those for which there are effective cures (e.g., chlamydia, gonorrhea, pubic lice, or syphilis), as well as those for which treatments are more limited (e.g., hepatitis, HIV, human papillomavirus). This risk may be related, in part, to higher rates of alcohol and drug use. In addition, given the high rates of homelessness and unemployment, transgender individuals are more likely to support themselves by engaging in underground economy activities, such as sex work, which increases risk for STIs. Transgender individuals who are sexually active should be encouraged to be screened for sexually transmitted diseases.

Smoking/Tobacco Use

The limited information that exists regarding transgender individuals and tobacco use suggests that transgender adults smoke cigarettes and use tobacco products at a higher rate than the general population of adults. Transgender individuals should be screened for tobacco use and offered tobacco cessation treatment or resources. Identification of factors that contribute to tobacco use (e.g., depression, minority stress) and treatment to address the underlying stressors may help reduce tobacco use in this population.

Weight

Although the data regarding transgender individuals’ weight and exercise habits are limited, current findings suggest that transgender individuals are more likely to be either underweight or obese compared with cisgender individuals. Obesity may be impacted by other co-occurring risk factors, such as depression and minority stress. On the other end of the spectrum, a subset of the transgender population may be underweight in an attempt to achieve a body type that is more consistent with their gender identity. Transgender individuals may benefit

from providers approaching discussions about their weight in a manner that is supportive and affirming of gender identity while also promoting the importance of healthy diet and exercise.

Transition-Related Health Issues

In addition to being at disproportionately high risk for the health issues described above, some transgender individuals experience health-related issues related to medical treatments for gender dysphoria or discomfort or distress that is caused by a discrepancy between a person's gender identity and sex assigned at birth (Coleman et al., 2012). A range of medical treatment options are available that can help individuals move toward achieving changes that are more consistent with their self-identity and reduce or alleviate their gender dysphoria (Coleman et al., 2012). These treatments range from hormone therapy and hair removal to a number of surgical interventions that include feminizing procedures such as vaginoplasty, breast augmentation, or facial alteration or masculinizing procedures that include phalloplasty, scrotoplasty, metoidioplasty, chest surgery, or liposuction. Transgender people make highly individual decisions about which procedures or steps to pursue to achieve their gender transition needs. Some individuals do not pursue any medical interventions, while there are others who may pursue many.

The World Professional Association for Transgender Health (WPATH) is a multidisciplinary organization whose mission is to “promote evidence-based care, education, research, advocacy, public policy, and respect for transgender health” (Coleman et al., 2012). Consistent with this mission, the WPATH draws from current research and expert opinions to develop *Standards of Care (SOC) for the Health of Transsexual, Transgender, and Gender Nonconforming People* (Coleman et al., 2012). Importantly, the WPATH has determined that medical treatments, including hormones and surgical interventions, are medically necessary to treat gender dysphoria in many people.

There are many health-related implications of these treatments that must be considered. It is important to ensure that individuals who are pursuing these medically necessary treatments for gender dysphoria are well informed about the procedure and course of treatment, have a good understanding of the associated risks, have realistic expectations regarding outcomes, and have the ability to adhere to their medical regimen. In addition, given the individualized and complex nature of each person's treatment plan, having a good working relationship with an interdisciplinary, culturally competent team of providers can help transgender individuals achieve their treatment goals while prioritizing their overall health and well-being, thus enhancing outcomes.

Hormone Therapy

Gender-affirming hormone therapy is a commonly sought medical intervention, and it allows individuals to develop secondary sex characteristics that are more consistent with their gender identity and can help to minimize some characteristics that are associated with sex assigned at birth.

There are a number of risk factors associated with hormone use. For feminizing hormones, the most prominent side effects are venous thromboembolic disease, gallstones, elevated liver enzymes, weight gain, and hypertriglyceridemia. There is also a risk of cardiovascular disease,

particularly in the presence of other risk factors. For masculinizing hormones, the most significant side effects are polycythemia, weight gain, acne, balding, and sleep apnea. Promoting healthy behaviors and good medical adherence can help to minimize some of these risks. Some individuals may also be reluctant to report side effects if they are concerned that they will be taken off of hormones. As noted above, having a trusting relationship with a culturally competent provider who promotes open communication can help minimize this risk.

Surgical Procedures

While many transgender and gender-nonconforming individuals do not desire or pursue surgical interventions, for many others, the modification of their primary and/or secondary sex characteristics is medically necessary to address their gender dysphoria. As many of these surgical interventions are irreversible, surgeons often require careful decision making. While a mental health evaluation is not always required to initiate hormone treatment, the WPATH recommends that an appropriate mental health assessment is conducted prior to genital and chest surgeries, and criteria for each different surgical intervention are provided in the SOC. For all procedures, evidence of persistent gender dysphoria is recommended, and for some surgeries, criteria include being on hormone therapy (unless medically contraindicated) and living in the gender role that is consistent with their gender identity for a specified period of time.

Unregulated Treatments

Some individuals who are not able to obtain gender-affirming medical treatments by qualified providers or those who are worried that they will not meet criteria may attempt to obtain treatments in an unregulated manner. For example, these individuals may buy hormones without a prescription from pharmacies online or from other unregulated sources without appropriate medical oversight. There are multiple risks associated with this approach: the hormones purchased may be unsafe or counterfeit, and use of hormones without appropriate medical monitoring could lead to health-related issues that go undetected or untreated.

Injectable silicone is another treatment that is sometimes pursued by transgender women who want to achieve feminizing body changes rapidly. While injectable silicone can be used safely when administered by a trained healthcare professional for more minor enhancements, it can be dangerous and potentially lethal when used by unqualified individuals. The FDA warns against the use of silicone for larger-scale body contouring, which includes injecting large quantities of silicone in hopes of quickly achieving a more feminine body type. When silicone is obtained through unregulated methods, there is also the risk that the silicone used is contaminated or not FDA approved. Ideally, individuals should only receive care from qualified licensed providers and be skeptical if the treatments are offered in nonmedical settings (e.g., private homes or hotel rooms).

Transgender individuals may opt to travel long distances, even overseas, to obtain gender-affirming surgeries. While cost and other barriers to care may drive these decisions, it is important that individuals understand the medical, psychological, and legal risks associated with traveling out of country to obtain care. Facilities may have varying quality of care, their promotional claims may be unregulated, and it can be difficult to assess outcomes from some

of these facilities. For continuity of care and to maximize positive outcomes, it is important that individuals return with a comprehensive postoperative plan and treatment providers whom they can enlist to provide follow-up care. Facilitating communication between providers and facilities (e.g., carrying medical records in both directions) may also allow for smoother transition of care.

Future Directions

In order to promote health and enhance care for transgender people, a number of actions can be taken. Most vital to this effort is for healthcare providers to be educated about best practices to ensure well-informed and culturally competent care. The importance of competence in provision of care is highlighted by the finding that more than 70% of psychology graduate students and psychologists reported unfamiliarity with transgender patients' experiences (American Psychological Association Task Force on Gender Identity and Gender Variance, 2009). To address this need, the American Psychological Association (APA) Council of Representatives on the Resolution on Transgender, Gender Identity, and Gender Expression Non-Discrimination called upon psychologists to provide appropriate nondiscriminatory treatment and to take an active role in leading work against discrimination toward transgender individuals.

WPATH is a leader in providing evidence-based standards of care that can be used as a guiding resource for both providers and patients, providing comprehensive recommendations for transgender health considerations. In addition, the APA has published *Guidelines for Psychological Practice with Transgender and Gender Nonconforming People* (APA, 2015), which were written in accordance with Endocrine Society and WPATH guidelines. These guidelines were developed with the purpose of promoting culturally competent, developmentally appropriate, and trans-affirmative care.

Healthcare systems can also enhance care for transgender individuals by ensuring that affirmative standards of care are mandated. Large healthcare systems can also receive certification through the Human Rights Campaign on the Healthcare Equality Index (HEI) if they demonstrate that they are competent to provide transgender care and promote affirmative workplace practices.

Although there is increasing awareness of the health issues that transgender individuals experience, the research is at a nascent stage. While there has been debate about the inclusion of transgender individuals and their health needs in broad-based surveys, it is notable that Healthy People 2020, the national health promotion goals set by the US Department of Health and Human Services, recommended an increase in the number of surveys that collect information on transgender health outcomes. The number of national representative surveys of transgender individuals has remained unchanged since 2008. Furthermore, much of the transgender health research has been based on convenience samples that do not necessarily represent the diversity of the transgender population within the United States (APA, 2015). Future studies using broader representative samples will provide a more complete understanding of the health-related issues and experiences of the transgender population.

In order to advance knowledge about healthcare needs of the transgender population, research funding by governmental and other large mechanisms is critical. In the United States, NIH funding of research dedicated to transgender health outcomes has historically been low: less than .04% of NIH funding from 1989 to 2011 (Coulter, Kenst, Bowen, & Scout, 2014).

Broad-based surveys that identify the healthcare needs and barriers to treatment of the transgender population can be used to highlight the necessity of greater funding to address the healthcare needs and disparities of this marginalized population.

Cross-References

See Also

Sex differences and health, Sexual minority populations and health

Author Biographies

DeAnna L. Mori, PhD, is the director of behavioral medicine at VA Boston Healthcare System and an assistant professor of psychiatry at Boston University School of Medicine. She is a member of the Interdisciplinary Transgender Treatment Team at VA Boston and provides training for psychology interns and fellows about transgender health.

Colleen A. Sloan, PhD, is a clinical psychologist at VA Boston Healthcare System and an assistant professor of psychiatry at Boston University School of Medicine. Dr. Sloan chairs the Interdisciplinary Transgender Treatment Team at VA Boston and serves as the psychologist team member of a national expert training and consultation team for transgender health in VA.

Heather Walton Flynn, PhD, is a staff psychologist at VA Boston Healthcare System and an instructor at Harvard Medical School. She is experienced in providing psychotherapy to transgender patients and trains psychology interns and psychiatry residents and is a member of the Interdisciplinary Transgender Treatment Team at VA Boston.

Claire Burgess, PhD, has interests in improving behavioral health services for minority populations. She has clinical expertise in working with transgender and gender nonconforming veterans through her work as a Veteran Care Coordinator and serves on the Interdisciplinary Transgender Treatment Team at VA Boston.

References

- American Psychological Association. (2015). Guidelines for psychological practice with transgender and gender nonconforming people. *American Psychologist*, 70(9), 832–864.
- American Psychological Association Task Force on Gender Identity and Gender Variance. (2009). *Report of the task force on gender identity and gender variance*. Washington, DC: Author. Retrieved from <http://www.apa.org/pi/lgbt/resources/policy/gender-identity-report.pdf>
- Coleman, E., Bockting, W., Botzer, M., Cohen-Kettenis, P., DeCuypere, G., Feldman, J., ... Zucker, K. (2012). Standards of care for the health of transsexual, transgender, and gender-nonconforming people, Version 7. *International Journal of Transgenderism*, 13, 165–232. doi:10.1080/15532739.2011.700873
- Coulter, R. W., Kenst, K. S., Bowen, D., & Scout. (2014). Research funded by the National Institutes of Health on the health of lesbian, gay, bisexual, and transgender populations. *American Journal of Public Health*, 104(2), e105–e112.
- Gonzalez, C., Gallego, J., & Bockting, W. (2017). Demographic characteristics, components of sexuality and gender, and minority stress and their associations to excessive alcohol, cannabis, and illicit (non-cannabis) drug use among a large sample of transgender people in the United States. *The Journal of Primary Prevention*, 38, 419–445. doi:10.1007/s10935-017-0469-4

- Hendricks, M., & Testa, R. (2012). A conceptual framework for clinical work with transgender and gender nonconforming clients: An adaptation of the Minority Stress Model. *Professional Psychology: Research and Practice*, 43(5), 460. doi:10.1037/a0029597
- James, S., Herman, J. L., Rankin, S., Keisling, M., Mottet, L., & Anafi, M. (2016). *Executive summary of the report of the 2015 U.S. transgender survey*. Washington, DC: National Center for Transgender Equality.
- Makadon, H., Mayer, K., Potter, J., & Goldhammer, H. (Eds.) (2015). *Fenway guide to lesbian, gay, bisexual, and transgender health* (2nd ed.). Philadelphia, PA: American College of Physicians.
- Meyer, I. H. (2003). Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: Conceptual issues and research evidence. *Psychological Bulletin*, 129(5), 674.

Suggested Reading

- Byne, W., Karasic, D. H., Coleman, E., Eyler, A. E., Kidd, J. D., Meyer-Bahlburg, H. F. L., ... Pula, J. (2018). Gender dysphoria in adults: An overview and primer for psychiatrists. *Transgender Health*, 3(1), 57–70. doi:10.1089/trgh.2017.0053
- Endocrine Society. (2017). *Endocrine treatment of gender-dysphoria/gender-incongruent persons*. Retrieved from <https://www.endocrine.org/guidelines-and-clinical-practice/clinical-practice-guidelines/gender-dysphoria-gender-incongruence>
- Kauth, M., & Shipherd, J. (2018). *Adult transgender care: An interdisciplinary approach for training mental health professionals*. New York, NY: Taylor & Francis Group.
- Matsuno, E., & Israel, T. (2018). Psychological interventions promoting resilience among transgender individuals: Transgender resilience intervention model (TRIM). *The Counseling Psychologist*, 46(5), 632–655. doi:10.1177/0011000018787261
- Valentine, S. E., & Shipherd, J. (In Press). A systematic review of social stress and mental health among transgender and gender non-conforming people in the United States. *Clinical Psychology Review*. doi:10.1016/j.cpr.2018.03.003

Veterans' Health Issues

Cynthia J. Willmon

Reno VA Sierra Nevada Health Care System, Reno, NV, USA

Military Background Information

In order to understand issues concerning veterans, it may be helpful to briefly review contextual information pertaining to the military. In the United States, there are five active duty branches of the military or armed forces: the Army, the Navy, the Air Force, the Marine Corps, and the Coast Guard (US Military, 2017). Each branch has its own specified focus areas and eligibility requirements. Additionally, a person can join the military as an enlisted member or as an officer, depending upon one's experience and qualifications, and the person is typically employed full time with one's branch for a specified time period. It is likely that the differences in stated missions and cultures of the branches factor into which branch a person chooses to join along with other motivations for being in the armed forces (e.g., family legacy, to see the world, get away from home, avoid legal problems, path to citizenship, etc.). There are also National Guard and Reserve units connected to the military branches, and individuals who choose to go into the guard or reserves typically have civilian jobs, and they may be called into active duty military service if needed. Moreover, a person's military experience can often include initiation into the military (i.e., boot camps, basic training), specialized training in one or more occupations, multiple duty station assignments, and possible deployments to different countries or combat zones. How one leaves the military also matters and the type of military discharge a person receives can impact if he or she receives post-military benefits or services, such as qualifying for care through the Department of Veterans Affairs (VA) (Szymendera, 2016).

Definition of Veteran and Other Demographic Information

To a layperson, a veteran can generally be defined as a person who served in the military. According to national statute, a veteran is defined as a "person who served in the active

military, naval, or air service, and who was discharged or released therefrom under conditions other than dishonorable” (Szymendera, 2016, p. 1). Additionally, to qualify to receive VA benefits, a veteran must meet specific criteria, including having been in active service, meet minimum length of service requirements (especially if one joined the military on or after September 8, 1980), and not have had a dishonorable discharge (Szymendera, 2016). Veterans represent a small percentage of the US population. In 2014, it was estimated that there were 251.4 million adults ages 17 and above living in the United States and Puerto Rico and 19.4 million of these adults were identified as being veterans (US Department of Veteran Affairs, National Center for Veterans Analysis and Statistics [NCVAS], 2016). The vast majority of veterans are male, with only about 1.6 million of the 19.4 million veterans being female (NCVAS, 2016).

Veterans’ Health Issues

Veterans face unique health concerns based upon their military experiences. Some of these concerns may be associated with events occurring during the era in which a person served (Fontana & Rosenheck, 2008; Olenick, Flowers, & Diaz, 2015), and these concerns often include a combination of mental and physical health problems (Frayne et al., 2010; Fredricks & Nakazawa, 2015; Olenick et al., 2015; Spelman, Hunt, Seal, & Burgo-Black, 2012). Additionally, although a significant number of veterans qualify to receive healthcare from a VA facility, veterans do seek out healthcare services through the community or private sector (Fredricks & Nakazawa, 2015; Olenick et al., 2015; Taylor et al., 2012), and it is important for VA and non-VA providers alike to be aware of issues that may be common or prevalent in the veteran population (Spelman et al., 2012).

Chronic Pain

Pain is a common presenting problem for people seeking medical care (Kaur, Stechuchak, Coffman, Allen, & Bastian, 2007; Kerns, Otis, Rosenberg, & Reid, 2003), and this finding is consistent in the veteran population. Research in a primary care setting found that 48% of veterans reported having pain on a regular basis, and these veterans rated their health during the past year as being significantly worse than those who had no pain (Kerns et al., 2003). Additionally, chronic pain can be defined as pain that persists after the amount of time it takes for one’s body to heal from an acute injury (Kaur et al., 2007), and 90% of veterans in a primary care setting had pain conditions lasting longer than 6 months (Reid, Crone, Otis, & Kerns, 2002). Some of the most common chronic pain conditions found within the veteran population include joint pain, back pain, headaches, abdominal pain, and limb pain (Kaur et al., 2007), and the prevalence of low back pain in veterans increased about 5% annually from 2000 to 2007 (Sinnott & Wagner, 2009).

Age and gender differences have been found with regard to chronic pain. In a study comparing pain experiences in younger veterans (i.e., ages 64 and below) vs. older veterans (i.e., ages 65 and above), significant differences were found pertaining to the location of the worst experienced pain (Reid et al., 2002). Specifically, 32% of younger veterans experienced the most discomfort from low back pain, whereas 32% of older veterans identified leg pain as being the most uncomfortable. This study also found that older veterans report experiencing

more consistent or constant pain, whereas younger veterans rated their intermittent pain levels as being more intense. Additionally, gender differences in chronic pain exist. Specifically, female veterans experience significantly more headaches, abdominal pain, and limb pain than males, while male veterans have higher rates of back and joint pain than females (Kaur et al., 2007). Female veterans with chronic pain also have higher rates of comorbid mental health conditions than male veterans (Kaur et al., 2007).

Problems with physical, emotional, and other functioning are associated with chronic pain (Kerns, Sellinger, & Goodin, 2011). A significant number of veterans were found to be service-connected for pain-related conditions (Reid et al., 2002), and one of the requirements for VA service connection includes significant impairment in physical, occupational, or social functioning. Chronic pain was also found to be associated with reductions in veterans' performance of their roles at home and at work (Helmer et al., 2009). Additionally, veterans experiencing pain attend more outpatient medical appointments than veterans without pain (Kerns et al., 2003), and women veterans with chronic pain attended a higher number of medical visits than male veterans with chronic pain (Kaur et al., 2007). Moreover, high comorbidities have been found between chronic pain and multiple mental health disorders, including depression and posttraumatic stress disorder (PTSD) (Helmer et al., 2009; Kaur et al., 2007; Otis, Keane, & Kerns, 2003). Having a mental health diagnosis was associated with veterans' higher attendance of medical appointments for pain concerns, and these were especially true for veterans diagnosed with chronic pain and depression or PTSD (Kaur et al., 2007). Another group of researchers cited a study that found about 10% of veterans referred to a pain clinic had symptoms consistent with PTSD, and these researchers hypothesized that the comorbidity of chronic pain and PTSD is likely higher than this, especially in cases where one's pain is associated with one's experience of trauma (Otis et al., 2003). Furthermore, a recent study of OEF/OIF veterans (i.e., veterans who were deployed to Iraq and Afghanistan) in a VA polytrauma clinic found that about 72% of the sample met criteria for PTSD, about 88% experienced significant pain, and 65% had comorbid PTSD and pain (Lang, Veazey-Morris, Berlin, & Andrasik, 2016). This study found that diagnosis was related to healthcare utilization, with veterans with PTSD symptoms participating in significantly higher numbers of both mental and medical healthcare visits, while veterans with significant pain had high rates of attendance of medical appointments. Finally, associations were hypothesized between substance use and chronic pain. Specifically, veterans who experience regular pain have higher rates of tobacco use than veterans without pain (Kerns et al., 2003), and this could lead to the exacerbation of other health problems. Interestingly enough, the presence of pain was not found to be related to excessive alcohol use (Kerns et al., 2003) or alcohol misuse (Helmer et al., 2009).

Numerous recommendations have been made about how best to approach treatment with veterans who experience pain. Specifically, treatment should include a thorough assessment of one's pain, psychological assessments for all reporting pain, and the use of integrative interventions (Kerns et al., 2003). Integrative approaches can include a combination of treatments, including pharmacology, behavioral interventions, and complementary or alternative treatments (Spelman et al., 2012). Additionally, psychological treatments of pain may include self-regulatory approaches (e.g., biofeedback, relaxation training, hypnotherapy, or mindfulness), behavioral approaches (e.g., operant behavioral therapy or fear avoidance), cognitive behavioral therapy, or acceptance and commitment therapy. Finally, veterans with comorbid mental health diagnoses may benefit from participation in mental health services in addition to pain-focused treatments.

Sleep Problems

Disturbed sleep is an important area for consideration when working with veterans. The prevalence of problems related to sleepiness, snoring, and risk for sleep apnea is higher in veterans compared with those for nonveteran populations (Mustafa, Erokwu, Ebose, & Strohl, 2005). Specifically, 50% of veterans in a primary care setting reported problems with falling asleep, staying asleep, or sleeping too much during the past year, while 35% reported insomnia during the past week (Shepardson, Funderburk, Pigeon, & Maisto, 2014). In a different primary care study using different sleep measures, 40% of veterans reported symptoms consistent with a diagnosis of insomnia (Mustafa et al., 2005). High rates of sleep apnea have also been found, with 76% of veterans referred to a specialty sleep clinic for possible sleep problems being diagnosed with obstructive sleep apnea and 47% of those with this disorder had severe sleep apnea (Samson, Casey, Knepler, & Panos, 2012). Veterans also report receiving insufficient sleep more often than nonveterans, and they are more likely to get less than 7 hr of sleep each night (Faestel, Littell, Vitiello, Forsberg, & Littman, 2013). Additionally, OEF/OIF veterans with and without mental health diagnoses were found to experience significantly more sleep impairments, nightmares, and distressing dreams than nonveterans (Ulmer, Van Voorhees, Germain, Voils, & Beckham, 2015).

Sleep problems are associated with problems with health and functioning. Insufficient sleep in veterans was found to be significantly related to factors, including younger age, lower income, inability to work, frequent mental distress, current tobacco use, and binge-drinking behaviors (Faestel et al., 2013). Similarly, veterans who reported sleeping 5 hr or less per night had higher rates of PTSD, major depressive disorder (MDD), panic disorder, and risky health behaviors, including drug misuse, alcohol misuse, and suicidal ideation, than veterans sleeping between 7 and 8 hr per night (Swinkels, Ulmer, Beckham, Buse, & Calhoun, 2013). High rates of comorbidities exist between insomnia and other disorders, including depression, anxiety, PTSD, chronic pain, and traumatic brain injury (TBI) (Bramoweth & Germain, 2013). Sleep disturbance may also play a role in completed suicides in veteran populations (Pigeon, Britton, Ilgen, Chapman, & Conner, 2012). Additionally, insomnia has been found to be related to increased severity of PTSD and depressive symptoms in combat veterans, and insomnia can be viewed as a chronic condition, especially since insomnia symptoms in these veterans persisted over time, even after reductions in PTSD and depressive symptoms occurred (Pigeon, Campbell, Possemato, & Ouimette, 2013). Moreover, veterans who do not have diagnosable mental health problems were found to experience poorer sleep quality, shorter sleep duration, and more severe insomnia than nonveterans (Ulmer et al., 2015). Finally, veterans identified as sleeping too much (i.e., sleeping 9 hr per night or more) had higher rates of PTSD and MDD diagnoses and increased tobacco use than those identified as being average sleepers (Swinkels et al., 2013).

Given the prevalence of sleep problems in the veteran population, the following treatment recommendations are suggested. Assessment of sleep problems in primary care settings is recommended (Mustafa, 2015), and it is important to address sleep problems with all veterans with and without mental health diagnoses (Ulmer et al., 2015). Additionally, insufficient sleep and insomnia should be viewed as chronic conditions deserving of clinical attention, assessment, and referral for appropriate treatment (Faestel et al., 2013), and assessing for sleep duration (especially in regard to getting too little or too much sleep) could help identify other potential comorbid mental health conditions or risky health behaviors (Swinkels et al., 2013). Veterans can also be referred to specialty sleep clinics for further diagnostic testing if warranted. Moreover, the use of sleep-focused cognitive behavioral treatments and the

medication prazosin have resulted in significant reductions in sleep complaints, including insomnia (Trockel, Karlin, Taylor, & Manber, 2014) and frequency of nightmares (Germain et al., 2012). Furthermore, Shepardson et al. (2014) researched veterans' treatment preferences for insomnia and veterans identified trying to deal with sleep problems on their own or talking to their primary care provider about their problems as being their preferred treatment methods. Veterans were less likely to want to meet with a behavioral health provider or be prescribed a sleeping medication. Finally, veterans expressed a preference for individualized treatment as opposed to participation in group treatments for sleep problems (Shepardson et al., 2014).

Traumatic Brain Injuries

TBI within the veteran population has been a focus of attention in recent years. Aspects of military service including training situations and deployment to combat zones place veterans at risk for TBI (King & Wray, 2012), and blast-related TBIs may be unique to combat service, especially when exposed to improvised explosive devices (IEDs) (Elder, 2015). Additionally, OEF/OIF veterans are more likely to survive events causing TBIs due to improvements in military protective gear than veterans serving in previous conflicts (Vasterling, Verfaellie, & Sullivan, 2009), and it has been estimated that 20% of OEF/OIF veterans have experienced a TBI (Tanielian & Jaycox, 2008). Rates of deployment-related TBIs have also been found to be similar between male and female veterans (Iverson et al., 2011). Moreover, TBIs vary in level of severity from mild to severe (King & Wray, 2012). Morissette et al. (2011) cited that about 77% of the TBIs identified in a military sample were rated as being mild, and mild TBIs (mTBIs) often go undetected when they occur (Elder, 2015). Various symptoms (e.g., headaches, dizziness, irritability, disturbed sleep, concentration problems, light sensitivity, etc.) may develop following an mTBI, and these symptoms typically resolve within a few months for the vast majority of people (King & Wray, 2012). However, a recent study of OEF/OIF veterans with a history of TBI found that 85% experienced post-concussive symptoms (including headaches, balance problems, and/or memory problems) persistently longer than expected for an mTBI, and these symptoms likely result in ongoing problems in social and occupational functioning (Morissette et al., 2011). Female OEF/OIF veterans also had more severe somatosensory (e.g., pain, headaches, nausea), cognitive (e.g., poor concentration, decision-making problems, forgetting), and vestibular (e.g., loss of balance, dizziness, and coordination problems) symptoms than male veterans (Iverson et al., 2011).

Furthermore, TBI appears to be related to emotional and cognitive problems. A review of research found that veterans with a past history of TBI are at higher risk of attempting and completing suicide (King & Wray, 2012). Comorbidities have also been found between TBI and mental health problems, including depression and PTSD (Elder, 2015; King & Wray, 2012). Researchers note that there has been some difficulty determining comorbidity rates of TBI and PTSD due to overlap in diagnostic criteria and post-concussive symptoms, although one study found that about 44% of soldiers had both mTBI and PTSD (King & Wray, 2012), whereas another study found 75% had both disorders (King, Wade, & Wray, 2013). Additionally, there are gender differences in prevalence rates of veterans with TBI and other comorbid mental health disorders (Iverson et al., 2011). Specifically, female veterans had higher rates of diagnoses of depression, anxiety disorders, and comorbid depression and PTSD. Male veterans were more likely to be diagnosed with PTSD and substance use disorders. Moreover, moderate and severe TBIs have been associated with chronic cognitive

problems, and veterans with a history of these TBIs may be at risk for developing dementia and chronic traumatic encephalopathy (Elder, 2015). Finally, veterans with a history of TBI were found to utilize healthcare more frequently, and they were more likely to be referred for specialty care than those with no TBI (King et al., 2013). Veterans also attended significantly more behavioral health appointments if they had both TBI and PTSD than if they were diagnosed with only PTSD or only TBI.

King and Wray (2012) offer multiple treatment recommendations for providers seeing veterans with a history of TBI, especially in primary care settings. Specifically, they encourage all veterans to be screened for TBI, and a current assessment of a veterans' current cognitive, emotional, and somatic symptoms be done. They also recommend that veterans be informed about the likely course and recovery of symptoms associated with TBI and about other factors that could impact their symptoms, such as comorbid mental or physical health problems. Moreover, King and Wray (2012) suggest veterans be encouraged to develop coping skills, avoid future brain injuries if possible, and be referred for specialty care when needed (such as cognitive rehabilitation, polytrauma, mental health services, substance use programs, and pain clinics).

Posttraumatic Stress Disorder (PTSD)

PTSD is a mental health disorder that is often associated with veterans, and PTSD is believed to be more common in veteran than nonveteran populations. Recent studies estimate the lifetime prevalence rate of PTSD in veteran populations is between 6.3 and 8% (Smith, Goldstein, & Grant, 2016; Wisco et al., 2014), and it is estimated that 5% of veterans likely have current PTSD (Wisco et al., 2014). Lifetime prevalence rates have also been found to be significantly higher in female veterans and in younger veterans (especially those under the age of 30; Smith, Goldstein, et al., 2016; Wisco et al., 2014). Additionally, the majority of veterans experience at least one traumatic event during their lifetimes, and the most common traumatic events reported were death of a close friend or family member (61.3%), witnessing a sudden death or bad injury (37.7%), combat exposure (34.3%), and natural disasters (33.9%; Wisco et al., 2014). A recent study found that about 86% of OEF/OIF veterans were exposed to a life-threatening event during their recent deployment (Asnaani, Reddy, & Shea, 2014). Lifetime prevalence rates for PTSD were high for veterans who were sexually abused as an adult (37.3%), had heavy combat exposure (34.9%), or reported sexual abuse as a child (25.9%) (Wisco et al., 2014).

PTSD within the veteran population has been associated with numerous problems. Veterans with reexperiencing symptoms have been found to have more problems with pain and physical health functioning, while those with hyperarousal symptoms have lower energy and poorer reports of mental well-being (Asnaani et al., 2014). Trauma exposure and PTSD symptoms also were associated with poorer physical health in women veterans (Smith, Tyzik, & Iverson, 2015). Additionally, sleep problems are common in veterans with PTSD. A recent study found that 76% of recent combat veterans with PTSD have persistent insomnia, while more than half of these veterans report having distressing nightmares (Pigeon et al., 2013). This study also found that veterans with insomnia had more severe symptoms of PTSD and depression than those without, and the insomnia symptoms tended to persist, even after PTSD and depressive symptoms reduced or resolved. Moreover, exacerbation of PTSD symptoms has been found to occur in about 10% of veterans as they age, and this may be due to exposure to a greater number of traumas, increased cognitive difficulties, loneliness, lower social support,

and increased stress (Mota et al., 2016). Lifetime prevalence of PTSD also was associated with current suicidal ideation and past history of suicide attempts (Wisco et al., 2014). Finally, veterans with PTSD often have other comorbid mental health problems, including depressive disorders (e.g., MDD, dysthymia, etc.), anxiety disorders, substance use disorders, and personality disorders (Smith, Goldstein, et al., 2016; Vojvoda, Stefanovics, & Rosenheck, 2014; Wisco et al., 2014).

Given the increased prevalence of PTSD in veteran populations, it may be helpful for providers to regularly assess for PTSD when working with veterans. Additionally, PTSD can develop in response to numerous different types of trauma, with sexual assault and heavy combat exposure being the most common. Some of the most effective evidence-based treatments for PTSD include prolonged exposure (PE) and cognitive processing therapy (CPT) (Hamblen, Schnurr, Rosenberg, & Eftekhari, 2017). In addition, treatments to enhance coping skills and/or other identified problems, such as anger, pain, and physical health concerns, may be beneficial, and referral for appropriate medical care may be warranted. Moreover, insomnia was found to persist, even after PTSD symptoms resolve (Pigeon et al., 2013), and referral for insomnia treatment is likely appropriate. Finally, veterans should continue to be assessed for PTSD over time, given that about 10% experience an exacerbation in symptoms as they age.

Military Sexual Trauma (MST)

Military sexual trauma (MST) refers to threatening sexual harassment or sexual assault that occurred while an individual is in the military (US Department of Veterans Affairs, 2004). MST can include unwanted sexual attention (e.g., touching, sexual comments, cornering, or pressure for sexual favors) or being threatened with the use of force and/or actually being forced into sexual contact without consent (Klingensmith, Tsai, Mota, Southwick, & Pietrzak, 2014). In a brief review of the literature, Suris et al. (2013) reported that about 20% of female veterans and 1% of male veterans receiving care in the VA healthcare system experienced MST, and these rates are similar to the lifetime prevalence rates of sexual assault in nonveteran populations. Of note, MST only occurs during the time one is in the military as opposed to across one's lifetime, which suggests individuals in the military are at an increased risk for sexual assault compared with civilians (Klingensmith et al., 2014; Suris et al., 2013). Additionally, a recent national survey study found that approximately 7.6% of US veterans likely experienced MST, with female veterans having higher rates (32.4%) than male veterans (4.8%; Klingensmith et al., 2014). Another interesting finding is that the number of male and female veterans experiencing MST is actually pretty similar; however, MST impacts a larger percentage of female veterans given that the military is predominantly male.

MST has been associated with numerous problems within the veteran population. While both men and women can experience MST, the majority of research has focused on female veterans with MST, and it is believed that men and women experience similar mental health outcomes related to sexual assault (Suris & Lind, 2008). Specifically, MST is linked to increased mental health problems, including PTSD, depression, generalized anxiety, social phobia, eating disorders, and substance use disorders (Klingensmith et al., 2014; Pavao et al., 2013; Suris & Lind, 2008). One study of female veterans found that 76% of the women who met criteria for PTSD experienced MST (Kintzle et al., 2015), whereas a different study found that male veterans with MST were more likely to have mood disorders than those without MST (Mondragon et al., 2015). MST also has been linked to worse quality of life, poorer emotional

and cognitive functioning, increased risks for current suicidal ideation and history of suicide attempts, and interpersonal difficulties (Klingensmith et al., 2014; Mondragon et al., 2015). Additionally, reviews of the research literature indicate that MST is associated with physical health problems, including cardiovascular disease, pain, genitourinary conditions, gastrointestinal problems, headaches, fatigue, and chronic pulmonary and liver disease (Suris et al., 2013; Suris & Lind, 2008). Finally, high rates of MST have been found in both female and male homeless veterans (Pavao et al., 2013).

It has been recommended that all female and male veterans be assessed for MST (Klingensmith et al., 2014). Identifying individuals with MST and getting them engaged in appropriate care in a timely manner is important (Suris et al., 2013). Additionally, the vast majority of veterans do not seek treatment immediately after their MST experience; however, these individuals tend to utilize higher rates of healthcare services (including mental health services) presently, which demonstrates the ongoing impact MST may have on their lives (Kintzle et al., 2015; Klingensmith et al., 2014; Pavao et al., 2013). Moreover, educating veterans about the availability of treatment for problems associated with their MST is important, and veterans and non-VA providers alike may not be aware that mental and medical care associated with MST experiences is free at VA facilities (Suris et al., 2013). Finally, providing veterans with MST with integrated clinical care is important, given the mental, physical, and cognitive difficulties often associated with MST (Klingensmith et al., 2014).

Suicide

Suicide is a problem found in both veteran and civilian populations. Recent estimates indicate that 20 US veterans commit suicide each day (Shane & Kime, 2016), and the research literature is unclear about whether the prevalence of suicide in veterans is higher than that found in the American nonveteran population (Katz, McCarthy, Ignacio, & Kemp, 2012). Suicidal ideation is likely associated with suicide attempts. A national cohort study found that about 10% of veterans reported suicidal ideation within the past 2 weeks, and it also found that rates of suicidal ideation vary over time (Smith, Mota, et al., 2016). Specifically, about 5% of veterans experienced chronic suicidal ideation over a 2-year period, about 4% of veterans no longer had suicidal ideation after 2 years, and about 5% of veterans developed suicidal ideation over this time period. Additionally, a recent study found that about 9% of veterans in primary care setting have suicidal ideation (Magruder, Yeager, & Brawman-Mintzer, 2012), and increased suicidal ideation was associated with deployment-related TBI, mental health diagnoses (e.g., depression, PTSD, generalized anxiety disorder, and substance use disorders), head and musculoskeletal pain, and poorer physical, psychological, and social functioning (Gradus et al., 2015; Magruder et al., 2012; Smith, Goldstein, et al., 2016). Psychiatric distress and physical health problems were associated with the development and chronicity of suicidal ideation (Smith, Goldstein, et al., 2016), and pain-related functioning played a more significant role in suicidal thoughts than pain severity (Magruder et al., 2012). A qualitative study of OEF/OIF veterans also found suicidal ideation to be associated with aspects of military culture, difficult deployment experiences, and challenges adjusting to life after deployment (Denneson et al., 2014).

Multiple studies have been conducted examining people who completed suicide to better understand factors that may have contributed to these deaths, including veteran status, gender, age, presence of psychiatric symptoms, and healthcare utilization. The majority of veteran suicides are via firearms (Kaplan, McFarland, Huguet, & Valenstein, 2012), and suicide rates for

male veterans are significantly higher than for female veterans (Katz et al., 2012). Male veterans age 64 and younger were found to have a higher risk of suicide than nonveterans (Kaplan et al., 2012). Age differences were also found in veterans who completed suicide (Kaplan et al., 2012). Specifically, younger male veterans had more occupational, financial, legal, and intimate relationship problems prior to their deaths, they had a higher number of past suicide attempts, and they were more likely to be intoxicated at their time of death compared with older veterans. Male veterans age 65 and older had more health problems, and they also had higher rates of firearm-related suicides than younger veterans (83.5 vs. 61%; Kaplan et al., 2012).

Suicide rates were also found to differ in veterans based on their healthcare utilization. Katz et al. (2012) found that suicide rates were higher for male veterans who utilized care through the VA than those who did not, and it was hypothesized that this may be due to VA users having more problems resulting in them being at increased risk for suicide. However, a decline in suicide rates from 2005 to 2008 was found for male veterans under the age of 30 who utilized VA care compared with those who did not, and this decline may be related to an enhanced focus on suicide prevention within the VA (Katz et al., 2012). In a study examining characteristics of veterans who committed suicide while involved in VA care, 68% of veterans had clinician-documented psychiatric symptoms within the past year compared with 32% of veterans with none of these symptoms (Britton et al., 2012). Veterans with identified mental health symptoms had higher rates of reported suicidal ideation and plans, mental health treatment participation, sleep disturbance, and stressors, including occupational, relationship, legal, and housing problems. These veterans also had higher rates of death within 60 days of their last VA visit, and death rates of veterans who reported suicidal ideation within the past year were twice as high within 30 days of their last VA visit than those with no suicidal thoughts (Britton et al., 2012). Similarly, a different study found that veterans with sleep problems committed suicide more quickly after their last VA visit than those without disturbed sleep (on average 75 vs. 174 days; Pigeon et al., 2012). Veterans with and without psychiatric symptoms who committed suicide were found to have similar rates of chronic pain, and this suggests that pain may be a risk factor for suicide regardless of mental health status (Britton et al., 2012).

Given the prevalence of suicidal ideation and suicide within the veteran population, the following treatment recommendations are encouraged. All veterans should be periodically monitored for suicidal ideation because veterans at risk for suicide may not be easily identified (Britton et al., 2012) and suicidal ideation can change over time (Smith, Goldstein, et al., 2016). Additionally, identified risk factors for suicide in veterans include pain, sleep problems, health problems (especially in older veterans), life stressors, relationship problems (especially in younger veterans), and mental health diagnoses (Britton et al., 2012; Kaplan et al., 2012; Magruder, Kassam-Adams, Thoresen, & Olff, 2016). Suicide prevention strategies would likely benefit from being tailored to veterans' individual concerns (Denneson et al., 2014), and intervention strategies would likely include development of appropriate coping skills (Kaplan et al., 2012). Moreover, planning for safety (including restricting access to weapons) is important in veteran populations, and non-VA providers should be educated about this (Kaplan et al., 2012).

Conclusion

Overall, US veterans experience a number of physical and mental health concerns that may be associated with their military experience, including chronic pain, sleep problems, TBI, PTSD, MST, and suicidal ideation and/or attempts. Although many of these problems are not unique

to the veteran population, many of these health concerns have been found to occur at higher rates in veteran populations and with significant impact on one's level of functioning. Additionally, it is important for VA and non-VA providers alike to be informed about issues experienced by veterans, given that a large number of veterans participate in healthcare through the private or community sector (Fredricks & Nakazawa, 2015; Olenick et al., 2015; Spelman et al., 2012; Taylor et al., 2012).

References

- Asnaani, A., Reddy, M. K., & Shea, M. T. (2014). The impact of PTSD symptoms on physical and mental health functioning in returning veterans. *Journal of Anxiety Disorders*, 28, 310–317.
- Bramoweth, A. D., & Germain, A. (2013). Deployment-related insomnia in military personnel and veterans. *Current Psychiatry Reports*, 15, 1–13.
- Britton, P. C., Ilgen, M. A., Valenstein, M., Knox, K., Claassen, C. A., & Conner, K. R. (2012). Differences between veteran suicides with and without psychiatric symptoms. *American Journal of Public Health*, 102, S125–S130.
- Denneson, L. M., Teo, A. R., Ganzini, L., Helmer, D. A., Bair, M. J., & Dobscha, S. K. (2014). Military veterans' experiences with suicidal ideation: Implications for intervention and prevention. *Suicide and Life-Threatening Behavior*, 45(4), 1–16.
- Elder, G. A. (2015). Update on TBI and cognitive impairment in military veterans. *Current Neurology and Neuroscience Reports*, 15, 1–9.
- Faestel, P. M., Littell, C. T., Vitiello, M. V., Forsberg, M. S., & Littman, A. J. (2013). Perceived insufficient rest or sleep among veterans: Behavioral risk factor surveillance system 2009. *Journal of Clinical Sleep Medicine*, 9, 577–584.
- Fontana, A., & Rosenheck, R. (2008). Treatment-seeking veterans of Iraq and Afghanistan comparison with veterans of previous wars. *The Journal of Nervous and Mental Disease*, 196, 513–521.
- Frayne, S. M., Chiu, V. Y., Iqbal, S., Berg, E. A., Laungani, K. J., Cronkite, R. C., ... Kimerling, R. (2010). Medical care needs of returning veterans with PTSD: Their other burden. *Journal of General Internal Medicine*, 26(1), 33–39.
- Fredricks, T. R., & Nakazawa, M. (2015). Perceptions of physicians in civilian medical practice on veterans' issues related to health care. *The Journal of the American Osteopathic Association*, 115(6), 360–368.
- Germain, A., Richardson, R., Moul, D. E., Mammen, O., Haas, G., Forman, S. D., ... Nofzinger, E. A. (2012). Placebo-controlled comparison of prazosin and cognitive-behavioral treatments for sleep disturbances in US military veterans. *Journal of Psychosomatic Research*, 72, 89–96.
- Gradus, J. L., Wisco, B. E., Luciano, M. T., Iverson, K. M., Marx, B. P., & Street, A. E. (2015). Traumatic brain injury and suicidal ideation among U.S. operation enduring freedom and operation Iraqi freedom veterans. *Journal of Traumatic Stress*, 28, 361–364.
- Hamblen J. L., Schnurr P. P., Rosenberg, A., & Eftekhar, A. (2017). Overviews of psychotherapy for PTSD. Retrieved from <https://www.ptsd.va.gov/professional/treatment/overview/overview-treatment-research.asp>.
- Helmer, D. A., Chandler, H. K., Quigley, K. S., Blatt, M., Teichman, R., & Lange, G. (2009). Chronic widespread pain, mental health, and physical role function in OEF/OIF veterans. *Pain Medicine*, 10(7), 1174–1182. doi:10.1111/j.1526-4637.2009.00723.x
- Iverson, K. M., Hendricks, A. M., Kimerling, R., Krengel, M., Meterko, M., Stolzmann, K. L., et al. (2011). Psychiatric diagnoses and neurobehavioral symptom severity among OEF/OIF VA patient with deployment-related traumatic brain injury: A gender comparison. *Women's Health Issues*, 21, S210–S217.
- Kaplan, M. S., McFarland, B. H., Huguet, N., & Valenstein, M. (2012). Suicide risk and precipitating circumstances among young, middle-aged, and older male veterans. *American Journal of Public Health*, 102, S131–S137.

- Katz, I. R., McCarthy, J. F., Ignacio, R. V., & Kemp, J. (2012). Suicide among veterans in 16 states, 2005 to 2008: Comparisons between utilizers and non-utilizers of Veterans Health Administration (VHA) services based on data from the National Death Index, the National Violent Death Reporting System, and VHA administrative records. *American Journal of Public Health, 102*, S105–S110.
- Kaur, S., Stechuchak, K. M., Coffman, C. J., Allen, K. D., & Bastian, L. A. (2007). Gender differences in health care utilization among veterans with chronic pain. *Society of General Internal Medicine, 22*, 228–233.
- Kerns, R. D., Otis, J., Rosenberg, R., & Reid, M. C. (2003). Veterans' reports of pain and associations with ratings of health, health-risk behaviors, affective distress, and use of the healthcare system. *Journal of Rehabilitation Research and Development, 40*(5), 371–380.
- Kerns, R. D., Sellinger, J., & Goodin, B. R. (2011). Psychological treatment of chronic pain. *Annual Review of Clinical Psychology, 7*, 411–434.
- King, P. R., Wade, M., & Wray, L. O. (2013). Health care utilization in OEF/OIF veterans with closed TBI. *Military Behavioral Health, 1*, 74–80.
- King, P. R., & Wray, L. O. (2012). Managing behavioral health needs of veterans with traumatic brain injury (TBI) in primary care. *Journal of Clinical Psychology in Medical Settings, 19*, 376–392.
- Kintzle, S., Schuyler, A. C., Ray-Letourneau, D., Ozuna, S. M., Munch, C., Xintarianos, E., ... Castro, C. A. (2015). Sexual trauma in the military: Exploring PTSD and mental health care utilization in female veterans. *Psychological Services, 12*, 394–401.
- Klingensmith, K., Tsai, J., Mota, N., Southwick, S. M., & Pietrzak, R. H. (2014). Military sexual trauma in US veterans: Results from the national health and resilience in veterans study. *Journal of Clinical Psychiatry, 75*, 1133–1139.
- Lang, K. P., Veazey-Morris, K., Berlin, K. S., & Andrasik, F. (2016). Factors affecting health care utilization in OEF/OIF veterans: The impact of PTSD and pain. *Military Medicine, 181*, 50–55.
- Magruder, K. M., Kassam-Adams, N., Thoresen, S., & Olff, M. (2016). Prevention and public health approaches to trauma and traumatic stress: A rationale and a call to action. *European Journal of Psychotraumatology, 7*. Article 29715
- Magruder, K. M., Yeager, D., & Brawman-Mintzer, O. (2012). The role of pain, functioning, and mental health in suicidality among veterans affairs primary care patients. *American Journal of Health, 102*, S118–S124.
- Mondragon, S. A., Wang, D., Pritchett, L., Graham, D. P., Plasencia, M. L., & Teng, E. J. (2015). The influence of military sexual trauma on returning OEF/OIF male veterans. *Psychological Services, 12*, 402–411.
- Morissette, S. B., Woodward, M., Kimbrel, N. A., Meyer, E. C., Kruse, M. I., Dolan, S., & Gulliver, S. B. (2011). Deployment-related TBI, persistent postconcussive symptoms, PTSD, and depression in OEF/OIF veterans. *Rehabilitation Psychology, 56*(4), 340–350.
- Mota, N., Tsai, J., Kirwin, P. D., Harpaz-Rotem, I., Krystal, J. H., Southwick, S. M., & Pietrzak, R. H. (2016). Late-life exacerbation of PTSD symptoms in US veterans: Results from the national health and resilience in veterans study. *Journal of Clinical Psychiatry, 77*, 348–354.
- Mustafa, F. A. (2015). The Safewards study lacks rigour despite its randomised design. *International Journal of Nursing Studies, 52*(12), 1906–1907. doi:10.1016/j.ijnurstu.2015.09.002
- Mustafa, M., Erokwu, N., Ebose, I., & Strohl, K. (2005). Sleep problems and the risk for sleep disorders in an outpatient veteran population. *Sleep & Breathing, 9*, 57–63.
- Olenick, M., Flowers, M., & Diaz, V. J. (2015). US veterans and their unique issues: Enhancing health care professional awareness. *Advances in Medical Education and Practice, 6*, 635–639.
- Otis, J. D., Keane, T. M., & Kerns, R. D. (2003). An examination of the relationship between chronic pain and post-traumatic stress disorder. *Journal of Rehabilitation Research and Development, 40*(5), 397–406.
- Pavao, J., Turchik, J. A., Hyun, J. K., Karpenko, J., Saweikis, M., McCutcheon, S., ... Kimerling, R. (2013). Military sexual trauma among homeless veterans. *Journal of General Internal Medicine, 28*, S536–S541.
- Pigeon, W. R., Britton, P. C., Ilgen, M. A., Chapman, B., & Conner, K. R. (2012). Sleep disturbance preceding suicide among veterans. *American Journal of Public Health, 102*, S93–S97.

- Pigeon, W. R., Campbell, C. E., Possemato, K., & Ouimette, P. (2013). Longitudinal relationships of insomnia, nightmares, and PTSD severity in recent combat veterans. *Journal of Psychosomatic Research, 75*, 546–550.
- Reid, M. C., Crone, K. T., Otis, J., & Kerns, R. D. (2002). Differences in pain-related characteristics among younger and older veterans receiving primary care. *Pain Medicine, 3*(2), 102–107.
- Samson, P., Casey, K. R., Knepler, J., & Panos, R. J. (2012). Clinical characteristics, comorbidities, and response to treatment of veterans with obstructive sleep apnea, Cincinnati veterans affairs medical center, 2005–2007. *CDC-Preventing Chronic Disease, 9*, 110–117.
- Shane, L., & Kime, P. (2016). New VA study finds 20 veterans commit suicide each day. *Military Times*. Retrieved from <http://www.militarytimes.com/story/veterans/2016/07/07/va-suicide-20-daily-research/86788332/>.
- Shepardson, R. L., Funderburk, J. S., Pigeon, W. R., & Maisto, S. A. (2014). Insomnia treatment experience and preferences among veterans affairs primary care patients. *Military Medicine, 179*, 1072–1076.
- Sinnott, P., & Wagner, T. H. (2009). Low back pain in VA users. *Archives of Internal Medicine, 169*(3), 1338–1339.
- Smith, B. N., Tyzik, A. L., & Iverson, K. M. (2015). Age-related differences in trauma exposure, PTSD symptomatology, and functional health and well-being in women veterans. *Traumatology, 21*, 128–135.
- Smith, N. B., Mota, N., Tsai, J., Monteith, L., Harpaz-Rotem, I., Southwick, S. M., & Pietrzak, R. H. (2016). Nature and determinants of suicidal ideation among U.S. veterans: Results from the national health and resilience in veterans study. *Journal of Affective Disorders, 197*, 66–73.
- Smith, S. M., Goldstein, R. B., & Grant, B. F. (2016). The association between post-traumatic stress disorder and lifetime DSM-5 psychiatric disorders among veterans: Data from the National Epidemiologic Survey on Alcohol and Related Conditions-III (NESARC-III). *Journal of Psychiatric Research, 82*, 16–22.
- Spelman, J. F., Hunt, S. C., Seal, K. H., & Burgo-Black, A. L. (2012). Post deployment care for returning combat veterans. *Journal of General Internal Medicine, 27*(9), 12001209.
- Suris, A., Holliday, R., Weitlauf, J. C., North, C. S., & the Veteran Safety Initiative Writing Collaborative. (2013). Military sexual trauma in the context of veterans' life experiences. *Federal Practitioner, 30*, 16–20.
- Suris, A., & Lind, L. (2008). Military sexual trauma: A review of prevalence and associated health consequences in veterans. *Trauma, Violence & Abuse, 9*, 250–269.
- Swinkels, C. M., Ulmer, C. S., Beckham, J. C., Buse, N., VA Mid-Atlantic MIRECC Registry Workgroup, & Calhoun, P. S. (2013). The association of sleep duration, mental health, and health risk behaviors among U.S. Afghanistan/Iraq era veterans. *Sleep, 36*, 1019–1025.
- Szymendera, S. D. (2016). Who is a "veteran"? Basic eligibility for veterans' benefits. Congressional Research Service, 1–7. Retrieved from <https://fas.org/sgp/crs/misc/R42324.pdf>.
- Tanielian, T., & Jaycox, L. H. (2008). *Invisible wounds of war: Psychological and cognitive injuries, their consequences, and services to assist recovery*. Santa Monica, CA: RAND Corporation.
- Taylor, B. C., Hagel, E. M., Carlson, K. F., Cifu, D. X., Cutting, A., Bidelsbach, D. E., & Sayer, N. A. (2012). Prevalence and costs of co-occurring traumatic brain injury with and without psychiatric disturbance and pain among Afghanistan and Iraq war veteran VA users. *Medical Care, 50*(4), 342–346.
- Trockel, M., Karlin, B. E., Taylor, C. B., & Manber, R. (2014). Cognitive behavior therapy for insomnia with veterans: Evaluation of effectiveness and correlates of treatment outcomes. *Behavior Research and Therapy, 53*, 41–45.
- Ulmer, C. S., Van Voorhees, E., Germain, A. E., Voils, C. I., Beckham, J. C., & VA MidAtlantic Mental Illness Research Education and Clinical Center Registry Workgroup. (2015). A comparison of sleep difficulties among Iraq/Afghanistan theater veterans with and without mental health diagnoses. *Journal of Clinical Sleep Medicine, 11*, 995–1005.

- United States Military. (2017). US Armed Forces Overview. Retrieved from <http://www.military.com/join-armed-forces/us-military-overview.html>.
- US Department of Veteran Affairs. *National Center for Veterans Analysis and Statistics*. (2016). Profile of Veterans: 2014. Data from the American Community Survey. Retrieved from https://www.va.gov/vetdata/docs/SpecialReports/Profile_of_Veterans_2014.pdf.
- US Department of Veterans Affairs. (2004). Military sexual trauma. Retrieved from https://www.publichealth.va.gov/docs/vhi/military_sexual_trauma.pdf.
- Vasterling, J. J., Verfaellie, M., & Sullivan, K. D. (2009). Mild traumatic brain injury and posttraumatic stress disorder in return veterans: Perspectives from cognitive neuroscience. *Clinical Psychology Review*, 29(8), 674–684.
- Vojvoda, D., Stefanovics, E., & Rosenheck, R. A. (2014). Treatment of veterans with PTSD at a VA medical center: Primary care versus mental health specialty care. *Psychiatric Services in Advance*, 65(10), 1–6.
- Wisco, B. E., Marx, B. P., Wolf, E. J., Miller, M. W., Southwick, S. M., & Pietrzak, R. H. (2014). Posttraumatic stress disorder in the US veteran population: Results for the national health and resilience in veterans study. *Journal of Clinical Psychiatry*, 75, 1338–1346.

Team Science in Health

Sasha Cukier, Ekaterina Klyachko, and Bonnie Spring

Department of Preventive Medicine, Northwestern University, Evanston, IL, USA

The Science and Praxis of Team Science: Definitions

Team science (TS) is the study and application of principles about how to assemble, optimize, and lead groups of people who collaborate on a shared endeavor. A science team in particular is composed of two or more individuals who have different roles and who work interdependently to accomplish a common research goal. In unidisciplinary teams, researchers from a single discipline work together to solve a shared problem. In multidisciplinary teams, researchers from different disciplines work separately on different parts of a research problem, each applying their own discipline specific expertise. The parts are then integrated at the end in an additive manner. Finally, interdisciplinary/transdisciplinary teams work in a more integrative, interactive manner. Team members work jointly to understand each other's disciplinary frameworks. They develop new language and a shared mental model that enables them to approach the shared research problem from a more deeply integrated perspective.

Two aspects of TS are of great contemporary interest: the science of TS and the praxis of TS. A 2015 report from the National Academy of Science (NAS) defined the science of TS as a new interdisciplinary field that empirically examines the processes by which scientific teams organize, communicate, and conduct research to achieve scientific breakthroughs that would not be attainable by either individual or simply additive efforts (NAS, 2015). To date, the science of TS has been largely concerned with describing features that appear to facilitate or hinder high impact research. Most of this research has been correlational in nature, concerned with identifying conditions that covary with bibliometric indicators of scholarly impact (i.e., citations). In parallel, research on the praxis of TS examines how to apply the TS knowledge base pragmatically so as to optimize the assembly, launch, and maturation of interdisciplinary research teams. Research on TS praxis includes not only observational studies characterizing the sociopsychological processes that successful teams display but also experimental studies of interventions aiming to improve team functioning.

The Emergence of Team Science

Throughout much of history, the dominant image of a scientist has been a lone individual secluded away in a lab, single-handedly trying to solve the most pressing scientific problems. This romantic notion of the lone scientist as “Renaissance person” (i.e., someone very accomplished at many different things) has a few prototypic exemplars. Consider Leonardo da Vinci who, alongside painting the Mona Lisa, made important scientific discoveries in engineering, weapons design, flight, and the understanding of human anatomy. Or contemplate Galileo Galilei, another Renaissance era polymath, whose studies of sunspots, planetary motion, temperature, gravity, and inertia contributed not only to astronomy and mathematics but also to physics. Jumping ahead to the seventeenth century brings to mind philosopher-architect Robert Hooke, who discovered and coined the term for “cell” while solving major problems in mechanics and potential energy, gravitation, astronomy, and paleontology. A few solo “Renaissance” scientists can even be identified in the nineteenth century. An example is the English physician Thomas Young, who lived from 1773 to 1829 and who has been called “the last man who knew everything.” In addition to deciphering Egyptian hieroglyphs on the Rosetta Stone, Young made significant contributions to the fields of vision, solid mechanics, energy, physiology, language, and musical harmony.

By the mid-twentieth century, however, the social ecology of scientific knowledge production had fundamentally changed, as the paradigm of the solo scientist lost ground. Discovery of the DNA double helix, unarguably one of the most significant breakthroughs of the twentieth century, emerged from a group effort between two English crystallographers (Rosalind Franklin and Maurice Wilkins), an American biologist (James Watson), and an English physicist (Francis Crick), albeit that Franklin’s main contribution to the collaboration occurred unknowingly when her discovery was shared without her consent. Another prominent TS example that occurred during WWII was the top secret Manhattan Project, which engaged physicists, mathematicians, and chemists from 19 different locations to collaborate on producing the first atomic bomb.

By 1955, 50% of science and engineering publications were authored by two or more individuals; by 2015, the percent of papers that were co-authored reached 90% (NAS, 2015). A 2007 analysis of nearly 20 million papers published over 5 decades and 2.1 million patents demonstrated that research is increasingly being done by teams in all branches of science (Wuchty, Jones, & Uzzi, 2007). Only in the arts and humanities does the solo author remain prevalent. Moreover, teams produce more frequently cited research than individuals do, and this team advantage has increased over time. The same growing dominance of teams that is observed in the physical sciences and engineering also is evident in the basic biological and social sciences, medicine, and health. Also consistent is the finding that National Institutes of Health (NIH)-funded extramural research produces primarily multi-authored papers (Lauer, 2018). Moreover, research teams are growing larger. The number of authors per paper is increasing over time in all areas of science, including health (Guimera, Uzzi, Spiro, & Amaral, 2005; Lauer, 2018). Finally, collaboration is becoming more geographically distributed across institutions, and, at least among relatively high-tier universities, the scholarly impact of multi-institution projects is higher than single-institute ones (Jones, Wuchty, & Uzzi, 2008).

What was true of the twentieth century is even more so in the twenty-first century. More and more, major scientific discoveries are made by larger teams. Mega-team authorship has become the norm in some research areas, such as genetics. For example, the initial report of the human genome sequence had more than 1000 authors. The Human Genome Project involved collaboration between scientific and medical communities from 20 institutions in

6 countries. The Large Hadron Collider at CERN, the world's biggest and most powerful particle accelerator, is a collaboration involving more than 10,000 scientists and engineers from over 100 countries, as well as hundreds of universities and laboratories.

Why Team Science?

As the body of scientific knowledge has grown, it has become increasingly difficult for a lone researcher to maintain deep expertise in more than one area. More than 50 million scientific papers have been published since the mid-1600s, at a current rate of about 2.5 million new scholarly publications every year. The pace of knowledge growth also has increased exponentially since the 1600s when it was still feasible for some Renaissance scientists to become both proficient in the arts and expert in several scientific disciplines. Moreover, the rate of knowledge proliferation continues to rise to a current state at which global scientific output doubles every 9 years (Bornmann & Mutz, 2015).

To keep up, science and scientists became more specialized, acquiring deeper knowledge of a more circumscribed domain. By the twentieth century, the growing specialization in science forced research to be carried out largely in disciplinary silos. Because of the time and resources involved in developing expertise, young scientists were encouraged to master a very narrow and specific subset of topics, identifying a unique niche from which they could contribute the deepest possible domain expertise. In consequence, as the volume of scientific knowledge expanded, it became increasingly difficult for one researcher to have a deep understanding of all the parts involved in any one problem. Slowly, the realization dawned that addressing the major unresolved research questions would require scientists from a variety of different fields and diverse expertise to collaborate. The lone scientist sitting in his isolated lab no longer possesses all the knowledge needed to single-handedly lead scientific discovery, and the most famous discoveries are no longer attributable to a single name.

In 2005, 2014, and 2015, the NAS produced several reports attributing the twenty-first-century surge of interest in TS to several powerful drivers (NAS, 2005, 2014, 2015). The first driver is the increasing impossibility for any individual to remain expert in multiple scientific disciplines. The second driver concerns the complexity of pressing unresolved societal and natural problems (e.g., poverty, global warming, violence, cancer) that command global policy attention. The knowledge and skills needed to address these outstrip the domain expertise of any single scientific discipline. Instead, such complex problems call for collaborative efforts that leverage the strengths and expertise of professionals trained in different fields. Whereas traditional single-investigator driven approaches suffice for solving well-defined problems of circumscribed scope, complex social problems with multiple causes call for coordinated teams of investigators with diverse skills and knowledge. A third driver of interest in TS is the availability of powerful but expensive new technologies whose use is optimally shared.

The 2005 NAS report on *Facilitating Interdisciplinary Science* sounded a clarion call signaling an urgent need to stimulate a more interdisciplinary research climate in the nation's universities (NAS, 2005). The goal was to advance fundamental understanding or to solve problems whose solutions are beyond the scope of a single discipline or field. Interdisciplinary research was defined as an approach integrating information, data, techniques, tools, perspectives, concepts, and theories from two or more disciplines or bodies of specialized knowledge. The 2005 NAS report opened eyes to the fact that academia lagged far behind industry and government laboratories in familiarity with and commitment to interdisciplinary research. Both industry and government research facilities had a track record of agile problem-driven

research whereby scientists could assemble in interdisciplinary teams and move flexibly between laboratories as the research problem and professional development needs warranted it. In contrast, even though the greatest density of scientific talent exists in the nation's universities, entrenched institutional customs in academia create a persistent "drag" on researchers who would like to do interdisciplinary research and teaching.

Interdisciplinary Team Science Initiatives

The major increase in federal funding for TS research that has occurred during the past four decades strongly incentivizes universities to encourage the formation of interdisciplinary research teams. The most recent NAS report on TS (NAS, 2015) highlights this trend. Both the National Science Foundation (NSF) and the NIH have increased the proportion of funding they award to research overseen by more than one principal investigator (PI). The number of NIH multiple PI (MPI) grants increased from 3 in 2006 (when such grants were first allowed) to 1,098 in 2013, or 15–20% of major grants awarded. Beginning in the mid-1980s, both NIH and NSF also increased their funding of research centers, which involve multiple related research projects and more loosely linked "U" networks of topically related grant projects.

Some NIH Center grant initiatives have been very large, and analysis of their productivity outcomes provides much of what is known about the science of funding TS. One example is the Transdisciplinary Tobacco Use Research Centers (TTURC), a group of seven university-based centers and a coordinating center co-funded in 1999 by the National Cancer Institute, National Institute on Drug Abuse, and the Robert Wood Johnson Foundation to advance understanding and reduce use of tobacco. A subsequent NCI initiative, the Transdisciplinary Research on Energetics and Cancer Centers (TREC) supported eight research centers and a coordination center in two separate funding cycles, 2005–2010 and 2011–2016, with aim of accelerating progress toward reducing cancers associated with obesity, physical inactivity, and poor diet. An even more expansive and international collaboration was the Human Genome Project, which aimed to determine the DNA sequence of the entire human genome. Launched 1990 and declared complete in 2003, this highly interdisciplinary project identified 22,300 genes in the human genome, ultimately yielding 2,000 genetic tests for adverse health conditions and 350 biotechnology products in clinical trials.

A major currently ongoing transdisciplinary NIH initiative is the Brain Research through Advancing Innovative Neurotechnologies (BRAIN) Initiative. Launched in 2014 with an initial annual allocation of \$100 million, this large-scale undertaking expanded to a budget of \$434 million for fiscal year 2017. The extensive interagency collaboration, involving 15 NIH institutes and centers, NSF, Defense Advanced Research Projects Agency (DARPA), Intelligence Advanced Research Projects Activity (IARPA), the Food and Drug Administration, and the Department of Energy (DOE), aims to develop tools to create a new dynamic picture of the brain that shows how individual cells and complex neural circuits interact in time and space.

Practical Challenges of Interdisciplinary Team Science

Mastering the praxis of TS sufficiently to optimize team performance is of great importance, especially given sizeable federal investment in large-scale interdisciplinary research. A group of

individuals needs to progress through the series of developmental stages needed to function well as a team. Bruce Tuckman described the team development sequence as forming–storming–norming–performing (Tuckman, 1965). Members of a new interdisciplinary team need to become oriented to who is on the team, what knowledge and skills they possess, and, on that basis, who will be responsible to undertake which tasks. Particularly because team members come from different disciplinary training backgrounds as well as having different personalities and demographic characteristics, the getting-acquainted process takes time, requires conversation, and is impeded by distance. In the late 1970s, research by Robert Allen identified a principle known in communication theory as the Allen curve. Allen found a strong negative correlation between physical distance and the frequency of communication between workers. He found that collaboration effectiveness declines when coworkers are more than 30–50 m apart, because they do not interact regularly. Although one might wonder whether the same principle holds true today in this era of easier virtual communication, Allen has argued in the affirmative, finding that we are more likely to telephone or use additional communication channels to contact those whom we often see in person (Allen & Henn, 2007).

To reach the next level of productivity, team members need to dialogue openly enough to earn each other's trust, a process that may initially cause the conflict that Tuckman characterized as storming. Teams can struggle between the application of their own knowledge and the willingness to listen to other points of view and discover new approaches. One discipline's efforts to adhere to standards of practice in their field can cause dispute with group members encouraging adoption of another discipline's methods and values. Eventually, to engage in productive problem solving about their shared project, teams need to progress toward generating a shared language and mental model of the problem at hand. Productive collision of viewpoints fosters the constructive disagreement needed to challenge conventional ways of thinking. It is defined as "a process by which parties who see different aspects of a problem can constructively explore their differences and search for solutions that go beyond their own limited vision of what is possible" (Gray, 1989).

Once a shared image has emerged of who knows what and how team members think differently, teams can progress to the next stage. During norming, team members become more open to each other's ideas and less inclined to take criticism personally. The team collaborates on establishing rules of conduct, values, standards, and methods. These shared normative expectations help the team to function more efficiently, segueing into the next stage of optimal performance when team members work collegially toward their shared objective.

Now try to imagine facilitating this stage-wise developmental progression for a very large team representing multiple disciplines and whose members are dispersed across multiple universities. Even though such cross-institutional teams can produce very high impact research (Jones et al., 2008), there is also evidence that the more universities involved in a collaboration, the fewer are the project outcomes such as patents, grant awards, and publications (Cummings & Kiesler, 2007). The discrepancy between these two apparently contradictory findings can be explained by variation in the team's degree of investment in coordination activities. Time allocated to communication and discussion among team members is as essential, if not more so, in distributed teams as it is in colocated ones. Substantial investment in coordination activities and tools is necessary to help cross-institutional teams establish a shared vision, divide responsibilities, and communicate effectively. Project management tools, regular teleconferences, in-person meetings, student exchanges, and co-authored manuscripts are all effective approaches to maintain coordination. Another essential ingredient is patience. That truism was well demonstrated in a longitudinal analysis that Kara Hall and colleagues conducted to compare the scientific productivity and impact of very large NIH transdisciplinary research

center grants versus traditional investigator-initiated NIH R01 grants in the same area. The results showed that although productivity initially lagged for the center grants, it eventually outstripped that for R01 grants both because of a higher rate of publication and a larger number of co-authors per paper (Hall et al., 2012).

Training Team Science Competencies

A number of training resources have been developed to facilitate skills development for those interested in working on interdisciplinary teams. One freely available online training resource is the COALESCE online learning modules available at <https://www.teamscience.net>. This web tool created by Bonnie Spring, Arlen Moller, and Holly Falk-Krzesinski at Northwestern University's Clinical and Translational Science Institute includes four modules. One provides an overview of the science of TS through animations and short videos featuring many of the experts who contributed to the NAS's 2015 volume on TS. The other three interactive modules present case scenarios that enable learners to practice foundational TS skills as applied to problems in clinical medicine, basic biological science, and behavioral science while assuming the roles of a junior scientist, a senior scientist, and a research development officer. Comparison of pretest with posttest scores of those completing the modules indicates that the training increases knowledge, attitudes, and self-efficacy about implementing TS practices (Klyachko et al., 2018).

A different training approach, sponsored by NSF, is the Toolbox Project (see <http://toolbox-project.org>) developed by philosophers Michael O'Rourke, Steven Crowley, and their colleagues. This facilitated in-person 2-hr group workshop prompts attendees to engage in philosophical dialogue about their fundamental assumptions regarding science, ways of knowing, values, and the nature of the world. Although the Toolbox Approach has not been formally evaluated, anecdotal evidence suggests that the workshop meets its goal of encouraging participants to thoughtfully consider other points of view.

Alternative training models take the approach of training TS competencies implicitly by embedding young investigators in the TS context most relevant to their research aspirations. Bill Ameredes and colleagues at the University of Texas Medical Branch took this approach to train early career investigators in interinstitutional collaboration (Ameredes et al., 2015). Self-report survey responses gathered from a subset of trainees suggest that participating in a multi-institutional science team increased participants' confidence in specific research competencies, including research design and statistical analysis. Another frequently used format is the weeklong immersive training experience. Trainees attend a retreat where they are grouped into teams to develop an interdisciplinary research proposal. They hear didactic lectures in relevant content areas and collaborate with project team members who have complementary skills in the other disciplines needed to design the research successfully. One such training experience is the very well-subscribed NIH-sponsored mHealth Summer Training Institute held at UCLA (see <https://mhealth.md2k.org/mhealth-training-institute>). The institute receives more than 300 applicants annually for its 30 positions. Pre-post comparison of self-reported knowledge, attitude, and skill changes reported by participants shows positive changes in appreciation of interdisciplinary science and confidence about working in teams.

Institutional Culture

The risks and the rewards of engaging in interdisciplinary TS have been well described for the individual investigator (Rhoten & Parker, 2004). In their 2014 report on Convergence, the

NAS went on to consider institutional barriers that impede integrative transdisciplinary science (NAS, 2014). The department-based budgeting structure of most universities is one tradition that has discouraged research and mentoring collaborations from crossing disciplinary lines. University departments compete to acquire their allocation of faculty positions, laboratory and office space, and seed funding from their school's central administration. Departments seek to justify their resource allocation by being credited for the grant funding, teaching, and service accomplishments contributed to the university by their departmental faculty. Even today, many years after recognizing and trying to correct institutional barriers to cross-disciplinary work, not all universities have figured out how to allocate shared credit and resources for research grants, teaching, and mentorship accomplished by faculty from more than one department. In such settings, university faculty may feel subtly discouraged from pursuing collaborations with colleagues in other departments. On a more positive note, most research-intensive universities have by now implemented sharing policies that support cross-departmental implementation of research grants.

The residual TS barrier that has remained most challenging to resolve exists at the level of the academic promotion and tenure system (Klein & Falk-Krzesinski, 2017). Because interdisciplinary science inherently occurs at the boundary of adjoining fields, but most distinguished senior scientists were trained as specialists, it can be a daunting task for review committees to acquire requisite letters of endorsement from external authorities who fully appreciate a candidate's interdisciplinary science. Augmenting that difficulty is the multi-authored nature of multidisciplinary publications, coupled with the promotion committee's historic bias to downgrade the importance of contributions other than first, last, and, in some communities, second authorship.

Optimizing Institutional Culture to Support Team Science

The National Center for Advancing Translational Sciences (NCATS), founded in 2011 as 1 of 27 institutes and centers at the NIH, is a major national driver for interdisciplinary TS. Broadly, NCATS's goal is to transform the translational process so that new treatments and cures for disease can be delivered to patients faster. NCATS regards TS as integral to its mission because of the need to "encourage partnerships that cross many scientific disciplines and research sectors in pursuit of translational success." Consequently institutions applying for CTSA designation are required to describe the mechanisms they have put in place to foster interdisciplinary science collaborations as well as engagement of researchers with community stakeholders.

A few institutions are changing institutional policy to facilitate TS. For example, in 2011, Northwestern University's Feinberg School of Medicine created a faculty track for team scientists. To quote from the University's handbook, the track is designed for "non-clinical faculty who make substantial contributions to the research and/or educational missions of the medical school. Faculty members whose primary activity is in research will typically engage in team science. Their skills, expertise and/or effort play a vital role in obtaining, sustaining and implementing programmatic research.... Typically such faculty provide critical expertise to a program or group of research teams as a co-investigator with contributions that do not necessarily require or result in independent grant funding, but some faculty on this track may serve as principal investigator on related research. Faculty on this track do not perform clinical work but do contribute to the education and service missions of the medical school.... For candidates on the Team Scientist track engaged in research and related academic activities, Feinberg recognizes the critical importance of collaboration in research and scholarly activity and that the contributions of middle authors in multi-authored publications are often seminal and of

the highest quality. It is an expectation that faculty on this track who choose the research domain will generally be members of a research team and that their contributions to publications will often be as middle authors. When research and/or scholarship is pursued in a collaborative fashion and results in multi-authored publications, the specific contributions of the candidate should be made clear. The school also recognizes the critical contributions of collaborators in the acquisition of grant funding to collaborative research projects.” Initiatives like the creation of the TS faculty track pave the way for institutions to support and reward the collaboration that is integral to TS.

Conclusion

Burgeoning government investment in interdisciplinary research reflects an urgent need to improve the public’s health by pressing forward the frontiers of transdisciplinary science. On one hand, an association between multiple cross-institutional authorship and citation impact has been documented, and it has become rare for scientific research to be conducted by single investigators. On the other hand, the existing evidence base gives scant guidance regarding best practices to follow when assembling, launching, and optimizing a science team. For now, the policies followed to assemble large cross-institutional interdisciplinary research teams must inevitably outstrip the science base about TS praxis. On the other hand, the process of iteratively trying to improve these teams’ performance affords a rich learning lab to enhance the knowledge base about how to optimize team performance. A first priority is to develop rigorous metrics that can track teams’ short-term and long-term team performances, making it feasible to understand how well and poorly performing teams evolve over time and the patterns that predict superior scientific productivity and impact. Having a grasp of how outstanding teams evolve should point to critical team development processes that can potentially be optimized. A bright future for interdisciplinary TS rests upon progressing the science of TS from descriptive to interventional studies that build a practical knowledge base translatable into high impact collaboration.

Author Biographies

Sasha Cukier graduated from Emory University where she majored in neuroscience and behavioral biology and minored in predictive health. Sasha worked as a research study assistant in the Spring Laboratory at Northwestern University, Feinberg School of Medicine, on the Opt-IN, SMART, and Sense2Stop studies exploring her research interests in preventive medicine and behavioral interventions. While working with diverse groups and teams spanning multiple institutions across the country, Sasha became interested in the science of team science and engaged in projects exploring what constitutes effective team-based research.

Dr. Ekaterina Klyachko is the team science program administrator at the Northwestern University Clinical and Translational Sciences Institute where her primary role is to support researchers in the development of multidisciplinary research teams. Prior to joining the TS program, she organized and managed multidisciplinary research programs and grant applications in the Northwestern University Feinberg Cardiovascular Research Institute. Katya holds a PhD in Microbiology from University of Illinois and a master’s in clinical research and regulatory administration from Northwestern University. Dr. Ekaterina Klyachko has been the recipient of multiple grant awards, and she has published numerous peer-reviewed manuscripts.

Dr. Bonnie Spring is professor of preventive medicine, psychology, psychiatry, and public health at Northwestern University and director of the Center for Behavior and Health – Institute for Public Health and Medicine. She also serves as team science director for NUCATS, Northwestern’s CTSI, and co-leader for the Cancer Prevention Program. She studies technology-supported interventions to promote healthy change in multiple chronic disease risk behaviors (particularly poor quality diet, overeating, physical inactivity, and smoking), and her research has been funded continuously for more than 30 years. Current mHealth trials ongoing by her group involve the use of wearable sensors to predict and preempt relapse to smoking, MOST factorial, SMART, and JITAI designs to optimize treatment for obesity and a cluster randomized trial to prevent loss of cardiovascular health among college students. A past president of the Society of Behavioral Medicine (SBM), she is a recipient of SBM’s Distinguished Research Mentor, Research to Practice Translation, and Distinguished Leadership awards and founding editor of its journal, *Translational Behavioral Medicine: Practice, Policy, Research*. A winner of The Obesity Society’s e-Health Pioneer Award and past chair of American Heart Association’s Behavior Change Committee, she chairs the NIH Psychosocial Risk and Disease Prevention standing study section. An original member of APA’s Advisory Steering Committee to establish Practice Guidelines and past chair of its Board of Scientific Affairs, she was awarded an American Psychological Association Presidential Citation for Innovative Research and Leadership in Health Psychology and for Vision in Incorporating Technology into Practice and Training. She has served as primary mentor for 42 training awards. Her NIH-funded learning modules on evidence-based practice (www.ebbp.org) and the science of team science (www.teamscience.net) are freely available online and have been used by more than 50,000 learners worldwide.

References

- Allen, T., & Henn, G. (2007). *The organization and architecture of innovation: Managing the flow of technology*. Boston, MA: Elsevier.
- Ameredes, B. T., Hellmich, M. R., Cestone, C. M., Wooten, K. C., Ottenbacher, K. J., Chonmaitree, T., ... Brasier, A. R. (2015). The multidisciplinary translational team (MTT) model for training and development of translational research investigators. *Clinical and Translational Science*, 8(5), 533–541. doi:10.1111/cts.12281
- Bornmann, L., & Mutz, R. (2015). Growth rates of modern science: A bibliometric analysis based on the number of publications and cited references. *Journal of the Association for Information Science and Technology*, 66(11), 2215–2222. doi:10.1002/asi.23329
- Cummings, J. N., & Kiesler, S. (2007). Coordination costs and project outcomes in multi-university collaborations. *Research Policy*, 36(10), 1620–1634. doi:10.1016/j.respol.2007.09.001
- Gray, B. (1989). *Collaborating: Finding common ground for multiparty problems*. Jossey-Bass management series. (1st ed.). San Francisco, CA: Jossey-Bass.
- Guimera, R., Uzzi, B., Spiro, J., & Amaral, L. A. (2005). Team assembly mechanisms determine collaboration network structure and team performance. *Science*, 308(5722), 697–702. doi:10.1126/science.1106340
- Hall, K. L., Stokols, D., Stipelman, B. A., Vogel, A. L., Feng, A., Masimore, B., ... Berrigan, D. (2012). Assessing the value of team science: A study comparing center- and investigator-initiated grants. *American Journal of Preventive Medicine*, 42(2), 157–163. doi:10.1016/j.amepre.2011.10.011
- Jones, B. F., Wuchty, S., & Uzzi, B. (2008). Multi-university research teams: Shifting impact, geography, and stratification in science. *Science*, 322(5905), 1259–1262. doi:10.1126/science.1158357
- Klein, J. T., & Falk-Krzesinski, H. J. (2017). Interdisciplinary and collaborative work: Framing promotion and tenure practices and policies. *Research Policy*, 46(6), 1055–1061. doi:10.1016/j.respol.2017.03.001

- Klyachko, E., Rak, P., McFadden, H., Pfammatter, A., Hedeker, D., Siddique, J., & Spring, B. (2018). Online team science training for health and medical professionals: teamscience.net. Annual Meeting of the Science of Team Science, Galveston, TX.
- Lauer, M. (2018). Impact of teams receiving NIH funding. *Open Mike* (blog). Retrieved from <https://nexus.od.nih.gov/all/2018/04/04/theres-no-i-in-team-assessing-impact-of-teams-receiving-nih-funding>
- National Academies of Science. (2005). *Facilitating interdisciplinary research*. Washington, DC: The National Academies Press.
- National Academies of Science. (2014). *Convergence: Facilitating transdisciplinary integration of life sciences, physical sciences, engineering, and beyond*. Washington, DC: The National Academies Press.
- National Academies of Science. (2015). Introduction. In N. J. Cooke & M. L. Hilton (Eds.), *Enhancing the effectiveness of team science* (pp. 19–46). Washington, DC: The National Academies Press.
- Rhoten, D., & Parker, A. (2004). Risks and rewards of an interdisciplinary research path. *Science*, 306(5704), 2046–2046. doi:10.1126/science.1103628
- Tuckman, B. W. (1965). Developmental sequence in small-groups. *Psychological Bulletin*, 63(6), 384–399. doi:10.1037/h0022100
- Wuchty, S., Jones, B. F., & Uzzi, B. (2007). The increasing dominance of teams in production of knowledge. *Science*, 316(5827), 1036–1039. doi:10.1126/science.1136099

Suggested Reading

- Bennett, L. M., Gadlin, H., & Levine-Finley, S. (2018). Collaboration & team science: A field guide. *NIH Publication No. 18-7660*.
- National Academies of Science. (2015). In N. J. Cooke & M. L. Hilton (Eds.), *Enhancing the effectiveness of team science*. Washington, DC: The National Academies Press.

Family and Health

Brittany N. Sherrill¹, Janet Deatruck², and Keith Sanford¹

¹*Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA*

²*Department of Family & Community Health, University of Pennsylvania School of Nursing, Philadelphia, PA, USA*

There are many ways in which family characteristics are associated with physical health. Family structure, family relationships, and family behaviors are associated with physical health behaviors and physical health outcomes. Also, the medical condition of a family member can place demands on other family members, and this in turn can influence the well-being of other family members and influence family functioning. Conversely, if a family member has a medical condition, family coping can influence the medical and emotional sequela of the medical condition. Before considering links between family characteristics and physical health, it is necessary to clarify the scope of the topic by defining both physical health and families. First, although there are many dimensions to health, it is useful to delimit the scope of physical health to include areas of well-being or disease that are defined primarily in terms of body organ systems. This definition makes a somewhat artificial distinction that excludes dimensions of mental-behavioral health, which pertains to areas of well-being or disease defined primarily in terms of behavior, emotion, and cognition. Second, although there are many diverse types of families, researchers have commonly defined families to include at least one of two basic components: intergenerational provision of care (such as between parents and children) and/or committed domestic partnerships (such as marriage or cohabitation relationships).

When considering ways that family and health are related, four overarching questions are salient. First, how are family-related variables associated with health behavior and health outcomes in the general population? Second, how does having a family member with a medical condition influence the health and well-being of other members of the family unit? Third, what types of family interaction behaviors help or hinder a family's ability to cope when one member has a medical condition? Fourth, what types of family interventions are effective in improving outcomes when one member has a medical condition? Each of these questions will be considered in detail below.

Associations Between Family Characteristics and Health Variables

The first overarching question pertains to correlations between family characteristics and health variables within a general population. This question is unique in that it is best addressed in studies of the general population and not in studies that focus exclusively on families that have a member with a medical condition. There is a large body of research investigating how health is related to family structure, such as being married or having children. There is also a body of research specifically investigating the effects of family conflict on health, as well as research specifically investigating how family relationships may predict outcomes that involve health behavior. In general, people tend to be the healthiest when they have supportive family relationships characterized by low levels of family conflict and when they live with other family members who engage in healthy behaviors.

Marriage

Research indicates that married individuals enjoy better physical health outcomes throughout their lifespan compared with their unmarried counterparts. Morbidity and mortality reduction in married individuals includes reduced levels of heart attacks, cancer, and surgery. This effect may be partially due to selection effects of healthy individuals getting married, as well as due to protective benefits of marriage related to access to resources, reduced stress levels, less engagement in risky health behaviors, and reduced social isolation. By reducing stress levels, marriage indirectly acts physiologically to improve health benefits by reducing cortisol levels, improving immune function, and reducing risk for cardiovascular disease. Although evidence of a link between marriage and health has been found across different economic status and culture groups, the link appears to be stronger in men than in women. A possible explanation for this trend is that men may tend to have narrower social networks outside of marriage; therefore, the effects of being married might be especially consequential.

Family Structure and Aging

Both marital status and having children are associated with health-related variables in aging populations. Marriage is associated with a lower disease risk as well as fewer functional limitations as individuals age. Additionally, a lack of children in old age is associated with riskier health behaviors due to a lack of the social control functions of parenthood. The presence of a single grandchild increases contact between parents and grandparents, which may mitigate the health effects of social isolation. There are also associations between family structure and the provision of healthcare to aging parents by their adult children. Research suggests that divorce and formation of stepfamilies reduce the extent to which children will provide healthcare for aging parents.

Conflict

Although marriage is generally associated with positive health outcomes, people in highly conflictual or distressed relationships are at greater risk for negative outcomes. While the health effects of simply being married may tend to be stronger for men, the effects regarding relationship conflict and distress tend to be stronger for women. This might reflect a tendency for women to be more attentive than men to issues regarding relationship quality. A large body of research on relationship conflict and health has focused on physiological stress

responses that occur when people experience relationship conflict. Recalling past conflicts has been shown to elevate blood pressure, while spouses who report low levels of cohesion in their marriage experience higher blood pressure simply from being in contact with their spouses (Baker et al., 1999). Both correlational and experimental research have found that exposure to relationship conflict is associated with impaired immune system functioning and prolonged inflammation responses. In part, this physiological response may be a direct consequence of the stress associated with relationship conflict. It also may be an indirect outcome of a mediated pathway that includes other variables associated with relationship conflict—such as depression, poor sleep, decreased physical activity, and unhealthy diet (Kiecolt-Glaser, Gouin, & Hantsoo, 2009). The physiological effects of family conflict are not limited to those family members who are directly involved in a conflict. Relationship conflict between parents is often noticed and a source of stress for their children. For instance, research has found that adolescents experience increased cortisol production at times when their parents report relationship conflict.

Family Influence on Health Behaviors

Family members also have the potential to influence the health behaviors of one another. Children's health beliefs and lifestyle behaviors are influenced by parent modeling, including parent involvement in exercise and healthy eating. These modeling effects last even beyond adolescence (Norton, Froelicher, Waters, & Carrieri-Kohlman, 2003). Negative parent health behaviors are often emulated by children. A case in point is children of parent smokers, who have an increased risk of becoming smokers in adolescence. Married partners also tend to share similar health behaviors pertaining to smoking, drinking, and exercise. Research suggests that this similarity is both due to people selecting similar partners to marry and to partners becoming more similar to each other after getting married. This type of pull toward partner similarity is also evidenced in the fact that former smokers are more likely to relapse in the presence of a partner who smokes. Research also finds that family structure is related to health behavior. Merely having a partner is associated with a decreased likelihood of smoking. Finally, child-birth is associated with changes in health behaviors in new parents. Physical activity tends to decrease in new parents and tends to remain low even several years after the birth of a child.

Family Outcomes When One Member Has a Medical Condition

The second overarching question relates to how families are influenced by having a member with a medical condition. This question is unique in that it is best answered with research comparing families where all members are healthy with families having one member with a medical condition. Research in this area has focused on two general types of family variables that may be affected when one member has a medical condition. These include (a) increases in feelings of stress among family members and (b) changes in the ways that family members interact with each other. The literature suggests that when one family member has a health condition, other members sometimes experience increased stress, while family interactions are sometimes also characterized by increased understanding toward the member with the health condition. However, research on this particular question sometimes produces results that are inconsistent across different studies or that are nonsignificant. This likely reflects the fact that there are substantial differences between types of medical conditions and between families in how they cope and respond to medical conditions. Compared with other questions regarding

families and health, it may be especially difficult to draw strong generalizations about how families are typically influenced by having a member with a medical condition.

Family Stress When One Member Has a Medical Condition

When one member of a family experiences a significant adverse health event, there is sometimes an increased risk that other family members will report feelings of stress, anxiety, or depression. When one member of a couple is diagnosed with cancer, it is common for the patient's partner to experience a similar level of psychological distress to the patient. Several studies have also investigated the extent to which parents experience stress when their children have medical conditions. Mothers and fathers of children who experience serious burn events report significant stress reactions. Posttraumatic stress disorder symptoms have been noted in parents of survivors of childhood leukemia (Kazak et al., 1997). These symptoms have also been observed in parents of children diagnosed with other types of cancer, as well as type 1 diabetes. It is important to note, however, that strong stress reactions are not consistently observed across studies or across medical conditions: one study found that the presence of juvenile rheumatoid arthritis had only small effects related to parents worrying about children's health. The intensity of the stress reaction is likely moderated by other variables. Some studies find that mothers report higher stress compared with fathers.

Family Interactions When One Member Has a Medical Condition

Research has found only a small number of consistent results regarding ways that family interactions may be influenced when one member has a medical condition. Compared with parents of healthy children, parents of children with medical conditions tend to be more excusing toward their children and to have more positive affective responses to child behavioral and academic problems. Along this line, one study found that, among children with phenylketonuria, the higher the phenylalanine levels were in the child, the more likely parents were to attribute child behavior and academic problems to external sources (Antshel, Brewster, & Waisbren, 2004). Aside from research showing that parents make excusing attributions for children with medical conditions, research has been mostly inconclusive regarding other possible ways that families typically respond when a child has a medical condition. Studies investigating this issue often fail to find significant results. One study comparing families with and without a child with juvenile rheumatoid arthritis found only minimal differences between families. In another study of families of children with a variety of health conditions including cystic fibrosis, sickle cell anemia, and epilepsy, no significant differences related to family functioning were observed.

Notably, it is sometimes suggested that having a child with a medical condition can lead to increased levels of family conflict. Along this line, one study found that among parents of children with epilepsy, the presence of recent seizures in the child was associated with high levels of expressed emotion in the parent, a type of interaction characterized by criticism and emotional over-involvement. However, there are few other studies suggesting this type of negative effect. Moreover, general research on married parents of children with disabilities has often (though not always) failed to find associations between child disability status and relationship satisfaction between parents. In sum, it is difficult to draw clear conclusions about how families typically respond when a child has a medical condition. Given differences between medical conditions and diversity in family experience, it may be especially difficult to make meaningful generalizations about the ways in which families typically respond to medical stressors.

It is also important to consider how couple relationships change when one partner has a medical condition. This association may be especially salient when a medical condition causes changes in areas that are relevant to the relationship, such as changes in physical appearance, sexual functioning, self-care ability, cognitive ability, and ability to perform previous responsibilities. However, as with research on families of children with medical conditions, it is difficult to draw clear generalizations regarding how couples respond. Studies investigating levels of relationship satisfaction often find that couples facing medical conditions have similar levels of satisfaction to couples that are not; however, this is not always the case. For example, one study of stem cell transplant cancer patients and their caregiving partners found that relationship satisfaction tended to decline over the course of treatment for female caregivers, but not for male caregivers or for patients (Langer, Yi, Storer, Syrjala, 2010). In general, the divorce rate for couples when one partner has a serious medical condition is similar to that of the general population. However, divorce rates are substantially higher when women have serious medical conditions, compared with when men have serious medical conditions. Thus, in infrequent situations where partner abandonment occurs, it is most likely to involve a healthy man abandoning a woman with a medical condition.

Family Interaction Behavior That Helps or Hinders Family Coping

The third overarching question involves examining research that identifies types of family interaction behaviors that help or hinder a family's ability to cope with a member's medical condition. Researchers investigating this topic have focused on three basic goals: (a) identifying the important dimensions of family interaction behavior, (b) investigating correlations between family interaction behavior and other dimensions of family functioning, and (c) investigating correlations between family interaction behavior and aspects of well-being outside of family functioning. As discussed below, this research finds that when a family member has a medical condition, positive family behavior and negative family behavior are not opposites and that negative family behavior may be especially consequential in predicting outcome variables regarding feelings of stress and well-being.

Key Dimensions of Family Interaction Behavior

Researchers have proposed many different models identifying types of family interaction behavior that are important when one member has a medical condition. A commonly used model of social support makes a distinction between the provision of emotional support (such as providing comfort to a family member) and instrumental support (such as completing a task to assist a family member with medical care). In another model focused on dyadic coping in couples, Bodenmann (1997) identifies four important components: common coping (behaviors that two partners do together), supportive coping (behaviors that one partner does to comfort, encourage, or advise the other), delegated coping (tasks that one partner completes at the request of the other), and negative coping (behaviors that are hostile, ambivalent, or superficial). Coyne and Smith (1991) identified a distinctive and potentially problematic type of behavior called "protective buffering" where one family member attempts to shield another family member from negative information. Other models have emphasized variables such as congruence between family members' preferred coping styles, the ways in which a family communicates about medical issues, and the ways in which family members may lead each other to have specific types of thoughts about or emotional reactions to a member's medical

condition. Other more general models that are not specific to families facing medical conditions suggest that important types of family interaction may include overall levels of family conflict, cohesion, and adaptability.

Despite the widely varied number of models put forth, recent research suggests that the types of family interaction described in these models can be understood as reflecting just two basic dimensions. One dimension involves the presence of positive behavior, and the other involves the absence of negative behavior. This research suggests that most types of *negative* family behavior are similar in function and most types of *positive* family behavior are similar in function, but there are important differences between the function of negative and positive behavior, respectively. In other words, they do not function as mere opposites.

According to the mobilization–minimization hypothesis (Taylor, 1991), it is adaptive for people to respond strongly and quickly to threatening events (such as negative family interactions). In comparison with positive events (such as positive family interactions), negative events elicit more intense affective, cognitive, and behavioral responses. Thus, negative family behavior may be most important because it elicits immediate stress responses that are memorable and distressing. This type of stress response is evident in the fact that interpersonal conflict is associated with physiological stress symptoms such as impaired immune system functioning and prolonged inflammation responses. In contrast, the function of positive behavior may be to facilitate the gradual building of relationship cohesion, which in turn is useful for emotion regulation. According to relational regulation theory, people are best able to regulate their emotions in the context of positive interpersonal interactions that include ordinary, routine conversations and shared activities with other significant people in their lives (Lakey & Orehek, 2011). In sum, negative interactions may function as stressors, whereas positive interactions may function to help members build cohesion and closeness.

Consistent with this model, a line of research suggests that when families face medical stressors, family members perceive their family interactions in a way that makes clear distinctions between the presence of positive interaction and the absence of negative interaction (Sanford, Backer-Fulghum, & Carson, 2016). This research began with a study that identified a large pool of different relationship behaviors that people naturally notice and experience in their relationships during a range of stressful life events. This pool included most of the behaviors previously identified as being important in models of family resilience and dyadic coping. Subsequently, a series of factor analytic studies indicated that this pool of behaviors reflects two basic underlying dimensions: first, a positive behavior dimension that includes family members providing support and companionship, contributing skills, and helping each other feel optimistic and second, a negative behavior dimension that includes family members being irritable, withdrawn, or avoidant, or failing to engage in expected positive behaviors. This research found high correlations between all the different types of positive behavior and high correlations between all the different types of negative behavior, while the correlation between positive and negative behavior was close to zero. This means that the presence of positive behavior is not necessarily associated with the absence of negative behavior, and vice versa. In fact, when families face stressful medical situations, there is a possibility that members may increase the use of both positive and negative behavior—such as being both more comforting and more irritable.

Family Interaction Behavior and Other Dimensions of Family Functioning

A large body of research covering a wide variety of medical conditions has investigated associations between different types of family coping behavior and other aspects of family

functioning. In general, this research finds that variables measuring different aspects of family functioning are correlated. In couples that have a child with a medical condition, and in couples where one partner has a medical condition, the use of positive couple coping behavior is strongly correlated with couple relationship satisfaction. Other research on families with a child with a medical condition finds that when families report low levels of family hardiness, the parents are likely to view their children as engaging in negative coping strategies. However, this line of research is difficult to interpret because it is mostly based on studies where people complete questionnaires asking about different aspects of family functioning. Due to a process called “sentiment override,” family members sometimes rely upon their global, overall feelings regarding family quality as a basis for how they respond to all self-report questionnaires measuring different aspects of family functioning. If people respond to all the questionnaire scales in a study on the basis of sentiment override, then all the scales will be correlated. Thus, it is not clear if this research tells us about the effects of family coping or if it merely shows us the extent to which responses to questions about family functioning are influenced by sentiment override.

Family Interaction Behavior and Well-Being

Research on families facing medical stressors has found that family interaction patterns are associated with emotional well-being. Several studies have investigated samples of couples where one partner is facing a serious medical condition such as breast cancer, lung cancer, prostate cancer, or a cardiac event. These studies have found that dimensions of couple interaction behavior are correlated with measures of emotional well-being ranging from depression and social dysfunction to illness-related distress. Other research with couples has specifically distinguished between positive and negative interaction behavior. In samples of couples where one member has a medical condition, as well as in samples of parents of a child with a medical condition, research has found that negative couple behavior is correlated with perceived stress, reduced well-being, and reduced quality of life. Notably, this negative behavior tends to be more important than positive behavior in predicting these outcomes. This is consistent with the mobilization–minimization hypothesis (discussed above) suggesting that negative and positive behavior have different functions and that negative behavior elicits particularly strong stress reactions.

In addition to research on couples, studies investigating parent–child relationships have also found links between family coping and well-being. In families of children with type 1 diabetes, children who rated their mothers as being uninvolved tended to be less likely to take recommended medications and to report poorer quality of life. In families of children with juvenile rheumatoid arthritis, mothers that describe their families as low in supportiveness and high in conflict are at greater risk of high levels of mental health symptoms.

There is also a large body of literature regarding family interactions when a family member experiences chronic pain. Several models of pain focus on the concept of pain behavior, in which a person verbally or nonverbally expresses or displays his or her pain, for example, by verbal complaint or facial expressions indicating pain. If a family member engages in pain behavior and this behavior is reinforced or supported by other family members, it can lead to increased pain behavior and, ultimately, higher levels of pain. This pattern has been observed in couples where one partner experiences chronic pain and in parent–child interactions where the child experiences pain. Additionally, research finds that parent reinforcement of their children’s pain behavior is related to school absences and that parent use of symptom-related talk is associated with increases in child somatic complaints. While reinforcement of pain behavior

is typically regarded as maladaptive, research with couples suggests that it is also beneficial for partners to be able to have intimate conversations about pain. Specifically, conversations characterized by discussion of emotions, as well as provision of empathy, are associated with less pain and less pain-related distress (Cano & Williams, 2010).

Family Interventions

The fourth overarching question has to do with identifying types of family interventions that improve outcomes when one member has a medical condition. These interventions include programs designed to help patients and their families cope with and manage medical conditions, and they fall into two broad categories. First, couple-based interventions are intended for couples where one partner has a medical condition and they target both members of the couple. Second, parent-based interventions are intended for families where a child has a medical condition and they include a substantial parent component. All these family interventions may target a range of different goals such as decreasing symptoms of the medical condition, improving adherence to a treatment plan, improving emotional well-being in the patient and in family members, and improving family relationships overall. In general, research in this area is still in its early stages. As might be expected for a new line of research seeking to develop effective treatments, the number of initial studies is still quite limited, and these initial studies often produce treatment effects that are small or nonsignificant.

Although several different types of couple-based interventions have been developed, many of these interventions are designed to address five different areas (for a review, see Baucom, Porter, Kirby, Hudepohl, 2012). First, they have an educational component where both members of a couple are provided with medical information. Second, they build intimate and clear communication in which partners share their thoughts and feelings regarding the medical condition. Third, they help partners negotiate the process of making medical decisions together. Fourth, they help partners make adjustments in their relationships to accommodate changes that may have been caused by a medical condition, including changes in physical ability, appearance, and sexual functioning. Fifth, they help couples improve their general relationship functioning.

At this point, evidence supporting the effectiveness of these types of interventions remains sparse. One small pilot study of women with early-stage breast cancer suggested that a couple-based relationship intervention might be beneficial in several areas, including increased acceptance and relationship satisfaction, as well as decreased pain and fatigue. In contrast, a randomized clinical trial of a caregiver-assisted cognitive skills training program for couples with a partner with lung cancer failed to find significant treatment benefits for emotional distress, quality of life, and caregiver stress. One of the largest randomized clinical trials for a couple-based intervention tested a partner-assisted emotional disclosure program for patients with gastrointestinal cancer. This study found that the treatment had nonsignificant effects on emotional variables but did have a small positive effect on relationship satisfaction. A follow-up analysis suggested that this effect was primarily noticeable in couples that initially did not talk about cancer-related concerns and emotions before treatment. As it stands, there remain many unanswered questions about which types of interventions will be most effective for which types of people.

Research regarding the effectiveness of parent-based interventions is also in its early stages. A meta-analysis identified three types of parent-based interventions that have been tested in randomized clinical trials (Law, Fisher, Fales, Noel, & Eccleston, 2014). First, cognitive

behavioral approaches teach all family members to change thoughts and behaviors that may exacerbate symptoms of a medical condition or feelings of distress. Second, problem-solving approaches focus on helping family members develop problem-solving skills related to the illness. Third, systemic approaches help family members alter potentially problematic patterns of interaction. This meta-analysis found a small but significant effect (.25 standard deviations of difference between treatment and control groups) indicating that, in general, parent-based interventions produced positive changes in parent behavior. However, this meta-analysis failed to find significant effects for other important outcomes such as parent and child mental health, family functioning, and child medical symptoms. Other reviews of parent-based interventions have suggested that cognitive behavioral approaches may reduce medical symptoms and improve family functioning in youth with chronic pain and that systemic approaches may reduce medical symptoms and improve family functioning in youth with diabetes. In sum, although some results are promising, there remains a need for further research and development of parent-based approaches for families of children with medical conditions.

See Also

Adapting to chronic illness, close relationships, coping styles, dissolution of intimate relationships, intimate relationships, marital and family distress, resilience, social factors in stress, social isolation/loneliness, social support.

Author Biographies

Brittany N. Sherrill, BA, is a clinical psychology doctoral student at Baylor University. She received her undergraduate degree from the University of Texas at Austin. She is currently conducting research on families, social support, and health.

Janet A. Deatrack, PhD, RN, FAAN, Professor Emerita of Nursing, focusing on potentially vulnerable populations who are at risk for health care disparities, provides leadership regarding children with chronic conditions and cancer, family management styles, health-related decisions, advanced practice pediatric nursing, and qualitative research methods.

Keith Sanford, PhD, is a professor in the Department of Psychology and Neuroscience at Baylor University. He serves on the editorial board of *Journal of Family Psychology* and *Family Process*, and he teaches graduate and undergraduate courses on advanced data analysis. He received his PhD in clinical psychology from Michigan State University, and he specializes in the development and validation of assessment instruments. His research program focuses on couples and family psychology; communication, emotion, and cognition in close interpersonal relationships; and associations between interpersonal relationships and health.

References

- Antshel, K. M., Brewster, S., & Waisbren, S. E. (2004). Child and parent attributions in chronic pediatric conditions: Phenylketonuria (PKU) as an exemplar. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 45(3), 622–630. doi:10.1111/j.1469-7610.2004.00251.x
- Baker, B., Helmers, K., O’Kelly, B., Sakinofsky, I., Abelsohn, A., & Tobe, S. (1999). Marital cohesion and ambulatory blood pressure in early hypertension. *American Journal of Hypertension*, 12(2), 227–230. doi:10.1016/S0895-7061(98)00184-8

- Baucom, D. H., Porter, L. S., Kirby, J. S., & Hudepohl, J. (2012). Couple-based interventions for medical problems. *Behavior Therapy*, 43(1), 61–76. doi:10.1016/j.beth.2011.01.008
- Bodenmann, G. (1997). Dyadic coping: A systemic-transactional view of stress and coping among couples: Theory and empirical findings. *European Review of Applied Psychology*, 47(2), 137–141.
- Cano, A., & Williams, A. C. (2010). Social interaction in pain: Reinforcing pain behaviors or building intimacy? *Pain*, 149(1), 9–11. doi:10.1016/j.pain.2009.10.010
- Coyne, J. C., & Smith, D. A. F. (1991). Couples coping with a myocardial infarction: A contextual perspective on wives' distress. *Journal of Personality and Social Psychology*, 6(3), 404–412. doi:10.1037/0022-3514.61.3.404
- Kazak, A. E., Barakat, L. P., Meeske, K., Christakis, D., Meadows, A. T., Casey, R., ... Stuber, M. L. (1997). Posttraumatic stress, family functioning, and social support in survivors of childhood Leukemia and their mothers and fathers. *Journal of Consulting and Clinical Psychology*, 65(1), 120–129. doi:10.1037/0022-006X.65.1.120
- Kiecolt-Glaser, J. K., Gouin, J.-P., & Hantsoo, L. (2009). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews*, 35(1), 33–38. doi:10.1016/j.neubiorev.2009.09.003
- Lakey, B., & Orehek, E. (2011). Relational regulation theory: A new approach to explain the link between perceived social support and mental health. *Psychological Review*, 118(3), 482–495. doi:10.1037/a0023477
- Langer, S. L., Yi, J. C., Storer, B. E., & Syrjala, K. L. (2010). Marital adjustment, satisfaction, and dissolution among hematopoietic stem cell transplant patients and spouses: A prospective, five-year longitudinal investigation. *Psychooncology*, 19(2), 190–200. doi:10.1002/pon.1542
- Law, E. F., Fisher, E., Fales, J., Noel, M., & Eccleston, C. (2014). Systematic review and meta-analysis of parent and family-based interventions for children and adolescents with chronic medical conditions. *Journal of Pediatric Psychology*, 39(8), 866–886. doi:10.1093/jpepsy/jsu032
- Norton, D. E., Froelicher, E. S., Waters, C. M., & Carrieri-Kohlman, V. (2003). Parental Influence on Models of Primary Prevention of Cardiovascular Disease in Children. *European Journal of Cardiovascular Nursing*, 2(4), 311–322. doi:10.1016/s1474-5151(03)00072-0
- Sanford, K., Backer-Fulghum, L. M., & Carson, C. (2016). Couple resilience inventory: Two dimensions of naturally occurring relationship behavior during stressful life events. *Psychological Assessment*, 28, 1243–1254. doi:10.1037/pas0000256
- Taylor, S. E. (1991). Asymmetrical effects of positive and negative events: The mobilization-minimization hypothesis. *Psychological Bulletin*, 110(1), 67–85. doi:10.1037/0033-2909.110.1.67

Suggested Reading

- Bodenmann, G. (2005). Dyadic coping and its significance for marital functioning. In T. Revenson, K. Kayser, & G. Bodenmann (Eds.), *Couples coping with stress: Emerging perspectives on dyadic coping* (pp. 33–50). Washington, DC: American Psychological Association.
- Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: His and hers. *Psychological Bulletin*, 127(4), 471–503. doi:10.1037//0033-2909.127.4.472

Index

- 12-Item Short Form Survey (SF-12) 151
- 20-Item Short Form Survey (SF-20) 151
- 2015 Survey of Psychology Health Service Providers*
(APA) 214
- ableism 52
- acceptance and commitment therapy 71
- accident
 - prevention 61
 - term, associations 61
- accountable care organizations (ACOs)
 - growth 81
 - impact 107
- Accreditation Council for Graduate Medical
Education (ACGME)
 - competency assessment requirements 159
 - surgeon residency training 161
- acetate, involvement 36
- acquired immunodeficiency syndrome (AIDS)
 - community health worker/patient navigation
interventions 92–93
 - symptoms 83
- actigraphy, usage 166
- Active Living Research initiative 31–32
- acupressure, usage 201, 203
- acupuncture, usage 201
- acute hypoxia headache, inducing 129
- adenine (A) 35–36
- adolescents
 - development, sleep (importance) 119–120
 - pain, occurrence 120
- advance care planning 11
- Advanced Illness Management (Sutter Health
Program) 81
- advance directives
 - definition/types 10
 - grounding 11
- Advisory Committee on Interdisciplinary
Community-Base Linkages (ACICBL),
role 105
- African Americans
 - men, Tuskegee syphilis study 44
 - self-care, study 47
- ageism 52
- aging bodies/minds, infirmities 10
- aging, family structure (connection) 254
- alcohol
 - misuse, veterans (experience) 232
 - use disorders (transgender individuals) 221
- alkalosis, increase 129
- Allen curve 247
- Allen, Robert 247
- allopathic medicine 1–2
- allostatic load 52
 - discrimination, impact 52–53
 - increase 5
- alternative medicine 199
- ambulatory palliative care programs,
focus 81
- Ameredes, Bill 248
- American Board of Professional Psychology
(ABPP), medical board certification 162

- American Cancer Society
 - health psychology research funding 30
 - Report to the Nation* 91–92
- American College of Surgeons, accreditation 159
- American Health Care Act, passage 103
- American Heart Association
 - funding 31
 - health psychology research funding 30
- American Indian health beliefs/
 - practices 201–202
- American Psychological Association (APA)
 - 2015 Survey of Psychology Health Service Providers* 214
 - Division of Health Psychology,
 - recognition 205
 - Ethical Principles of Psychologists and Code of Conduct 118
 - telemental health guidelines 192
- American Reinvestment and Recovery Act,
 - economic stimulus 104–105
- American Society for Metabolic and Bariatric Surgery, accreditation 159
- American Telemedicine Association,
 - guidelines 192
- amyotrophic lateral sclerosis (ALS) 18
- anandamide, involvement 129
- anger, psychosocial factor 3
- anorexia, presence 84
- anti-discrimination policies 220
- antiretroviral therapy (ART)
 - adherence 92–93
 - medication refills, obtaining (likelihood) 93
- anxiety, psychosocial factor 3
- apnea 174
- apple cider vinegar, usage 202–203
- arachidonic acid, involvement 129
- Arden House Conference 205–206, 208
 - Council of Health Psychology,
 - establishment 207
- aromatherapy, usage 203
- Asian Americans, citizenship right
 - (deprivation) 44
- Assess, Advise, Agree, Assist, and Arrange
 - (5 A's) 70
- assisted suicide laws 17
- asthma, self-management adherence (pediatric
 - psychologist assistance) 121
- atherosclerosis, SDB (relationship) 175
- atrial fibrillation, SDB (relationship) 175
- balance, concept/impact 202
- behavioral anchors, usage 215
- behavioral conditions, incidence/prevalence
 - (provider education) 107–108
- behavioral factors, role (recognition) 206–207
- Behavioral Health Laboratory (BHL)
 - care management model 68–69
 - initiation assessment 68–69
- behavioral health, stigma (decrease) 67
- Behavior Change Consortium 31
- benzodiazepine diazepam, effectiveness
 - (reduction) 132
- Better Care Reconciliation Act, passage
 - (failure) 1032
- biological risk profile 52
- biomedical model
 - acceptance 9–10
 - Black American belief 202
 - flaw 1
 - holistic perspective, adoption 12–13
 - success 9
- biopsychosocial (BPS) model 1, 205
 - chronic pain 2–3
 - conceptualization 2
 - diathesis-stress component, significance
 - (acknowledgment) 3–4
 - utility 3
- Black Americans, health beliefs/traditions 202
- blood alkalosis, reduction 129
- blood oxygen saturation (SO₂)
 - reduction 174
 - stability 129
- bloodstoppers 203
- Blueprint for Health Service Psychology Education
 and Training, A* (Health Service
 - Psychology Education Collaborative) 107
- body (whole person component) 1
- bone marrow aspiration 120
- botanicals, usage 202–203
- brain
 - activity, studies 75
 - function, statistical history (representation) 74
 - imaging techniques, usage 131
 - regions, mapping 76
- Brain Research through Advancing Innovative
 - Neurotechnologies (BRAIN), NIH
 - initiative 246
- breast cancer (development), BRCA1/BRCA2
 - (implication) 38–39
- breastfeeding, community health worker/patient
 - navigation interventions 96
- breathlessness, palliative care role 84
- burn doctors 203
- burn dressing changes 120

- cachexia, presence 84
- calorie counts, truthfulness (government requirement) 22
- cancer
 - care, obstacles 91–92
 - community health worker/patient navigation interventions 94–95
 - transgender individuals 221
- carbon dioxide, excess (elimination) 129
- cardiometabolic conditions, PTSD (association) 38
- cardiometabolic diseases, community health worker/patient navigation interventions 95–96
- cardiopulmonary resuscitation, decrease 84–85
- cardiovascular disease (transgender individuals) 222
- cardiovascular health, SDB (relationship) 175, 176
- cardiovascular system, dysregulation 5
- caregiver distress, reduction 84
- Carter Center, healthcare recommendations 107–108
- Carter, Jimmy, national healthcare system/changes (advocacy) 101
- catechol-O-methyltransferase (COMT)
 - chronic pain gene 4
 - functional Val158Met polymorphism 132
- CB1 cannabinoid receptor antagonist, impact 129
- Centers for Disease Control and Prevention (CDC), health psychology research funding 29
- Centers for Excellence in Cancer
 - Communication Research, special initiatives 31
- Centers for Population Health and Health Disparities, special initiatives 31
- Centers of Excellence in Cancer Communication Research 31
- central command (central governor), notion (implication) 137
- central sensitization (CS), impact 5
- central sleep apnea (CSA) 174
- chattel slavery 44
- childhood
 - behaviors, development/impact 120–121
 - diarrhea, decrease 96
 - environmental exposures, importance 55
- children
 - cancer, survival rate 122
 - development, pediatric psychologist understanding (increase) 117
 - development, sleep (importance) 119–120
 - health, community health worker/patient navigation interventions 96–97
 - pain, occurrence 120
- cholecystokinin (CCK)
 - antagonist, impact 128–129
 - pro-nociceptive system, medication ability 129
- chromatin, navigation (problems) 36
- chronically ill, caring (disease continuum) 86
- chronic back pain (CBP), functional connectivity (impact) 75
- chronic illness
 - diagnosis/management, BPS model (utility) 3
 - diathesis-stress BPS model 4
 - pediatric psychology adjustment 118
 - prevalence 3
- chronic lung disease, palliative care role 84
- chronic obstructive pulmonary disease (COPD)
 - dyspnea, presence 84
 - symptoms 83
- chronic pain
 - biopsychosocial (BPS) model 2–3
 - treatment
 - assessment 2
 - behavioral psychological principles, application 2
 - veterans, experience 230–231
- chronic short sleep, metabolic effects (reversal) 170
- chronic sleepiness 169
- class bias 52
- client-provider relationship (televideo etiquette) 195
- clinical environment, psychologist considerations 161–162
- clinical health psychologists, competencies 208
- clinical health psychology
 - description, standard (CCHPTP establishment) 209
 - entry-level competencies, articulation 210
 - entry-level practice, competencies (articulation) 207
 - future 108–110
 - specialization 209–210
 - specialty area, re-recognition (CRSPPP petition) 210
 - specialty training, expansion 210–211
- clinical trials, industry support 30
- clinical video telehealth (CVT), intervention 71
- Clinton, Bill, national healthcare system/changes (advocacy) 101
- COALESCE online learning modules 248

- co-authored manuscripts, usage 247–248
- cognitive behavioral therapy (CBT)
 - impact 71
 - usage 69, 121
- cognitive behavioral therapy for insomnia (CBTI)
 - goals 173
 - usage 172–173
- cognitive coping skills 121
- cognitive development, vulnerability 55
- cognitive functioning, decrease 119
- cognitive processing therapy (CPT), usage 235
- cold illnesses, treatment 201
- Commission for the Recognition of Specialties and Proficiencies in Professional Psychology (CRSPPP) 209–210
- Commonwealth-Cummings project 14–16
- Commonwealth Fund 14
- communicable diseases, community health worker/patient navigation interventions 92–94
- community-based, interprofessional healthcare teams (establishment), ACA funding (availability) 105–106
- community health workers
 - international titles 91
 - interventions, benefits 95–96
 - patient navigation, relationship 91
- “Competencies for Psychology Practice in Primary Care” 214
- competency-based education (ACA) 106–107
- complementary medicine 199
- complex regional pain syndrome (CRPS), functional connectivity (impact) 75
- compound stressors 53
- conditioned response (CR) 132
- congestive heart failure (CHF), palliative care role 84
- consultation-liaison (CL) services 118
- contextual exposures 52
- continuous positive airway pressure (CPAP) therapy, usage 175–176
- coping skills
 - cognitive coping skills 121
 - usage 120
- Core Competencies for Integrated Behavioral Health and Primary Care* 215
- core particle 36
- Core Quality of Life Questionnaire (QLQ-C30) 152
- Core Quality of Life Questionnaire module for prostate cancer (QLQ-PR25) 152
- Coronary Drug Project 135
- Council of Clinical Health Psychology Training Programs (CCHPTP) 208–209
 - clinical health psychology training programs, description standard (establishment) 209
 - incorporation 207
- Council of Health Psychology, establishment 207
- couple-based interventions, development 260
- CpG dinucleotide 36
- CpG sites 37–38
- Crick, Francis 244
- criminal victimization 53
- cultural broker/interpreter (patient navigator qualification) 92
- cultural competency, concept (consideration) 203
- cultural mistrust
 - consideration, impact 47
 - measurement 46–47
 - role 43–46
- Cultural Mistrust Inventory (CMI) 46
- cumulative physiological dysregulation 52
- cupping, usage 201
- curanderismo* (evolution) 200–201
- Current Procedural Terminology (CPT) codes, televideo services determination 193
- cyclooxygenase (COX), involvement 129
- cystic fibrosis (CF), pediatric psychologists (impact) 122
- cytosine (C) 35–36
- D2/D3 receptors, activation 131*n*
- da Vinci, Thomas 244
- death/dying 9. *See also* good death
 - Japanese/Navajo beliefs 11
 - pain-filled trajectory 11
 - wishes 11–12
- “Decade of Pain Control and Research” 3
- de facto discrimination 53
 - LGBTQIA 55
 - sexual minorities, experience 55
- Defense Advanced Research Projects Agency (DARPA) 246
- de jure discrimination 52–53
 - LGBTQIA 55
 - sexual minorities, experience 55
- deoxyribonucleic acid (DNA) 35–36
 - double helix, discovery 244
 - methylation 36
 - patterns, impact 38
 - synthetic DNA, fluorescence 37

- Department of Defense, health psychology research funding 30
- Department of Energy (DOE) 246
- Department of Veterans Affairs
 - health psychology research funding 30
 - psychology program, development 213
- depression
 - insomnia, relationship 172, 173
 - management, PCP/care manager (involvement) 69
 - psychosocial factor 3
 - symptoms (alleviation), insomnia (improvement) 173
 - transgender individuals 222
 - veterans 231
- diabetes
 - glycemic control (improvement), community health workers (impact) 95–96
 - pediatric psychologists, impact 122–123
- Diabetes Prevention Program 32
- diabetic neuropathy, functional connectivity (impact) 75
- diathesis-stress BPS model 3–4, 4
- diathesis-stress component, significance (acknowledgment) 3–4
- didactic training, usage 159
- diet, health psychology issues 38
- directly observed treatment (DOT) 93–94
- discrimination
 - categories 52
 - de facto discrimination 53
 - de jure discrimination 52–53
 - impact 52–53
 - problems, association 53
 - racial discrimination 44–45, 52–53
- disease
 - natural history 127
 - prevention (ACA) 104
- disharmony, impact 202
- disinvestment 53
- disorderly transitional stressful event 55
- dizziness (treatment), apple cider vinegar (usage) 202
- doctor-patient relationship
 - augmentation 134
 - importance 135
 - neurobiological domain, integration 135
 - placebo effect 134–137
 - steps 135
- dominant group (Whites), mistrust 44
- do not resuscitate (DNR)
 - order 10
 - preferences 13
- dopamine, placebo involvement 129–130
- drug misuse, veterans experience 232
- dyadic coping 257
- dying (people)
 - caring, best practices/knowledge 14
 - goodbye, saying (time allotment) 17
 - treatment options, knowledge 17
- dysfunctional attitudes/beliefs 3
- dyspnea, presence 84
- economic demands, reality 15
- economic hardship 53
- eHealth, pediatric psychologist usage 123
- Eisenhower, Dwight, national healthcare system/changes (advocacy) 101
- elderly depressed patients, multisite RCT (insomnia results) 172
- electroencephalography (EEG), usage 132
- electronic health records (EHRs), American Reinvestment and Recovery Act stimulus 104–105
- emergency department (ED), palliative care (providing) 80–81
- emotional responses, trigger 2
- emotional support 257
- endocrine system, dysregulation 5
- end of life (EOL)
 - clinical outcomes (improvement), palliative care (impact) 81
 - palliative care, hospice (usage) 82
 - preparation, importance 16
- end-of-life care, SUPPORT study 13–14
- end-of-life matters, Hispanic beliefs/intentions 11
- end-of-life preferences 13
- endogenous cannabinoid ligands, involvement 129
- Engel, George L. 1, 213
- environmental exposures, importance 55
- epigenetics 36
 - analysis, techniques 37
 - impact 4
- Epworth Sleepiness Scale (trait sleepiness) 167
- eugenics 45
- Euro-American folk medicine, New World arrival 202–203
- European Organisation for Research and Treatment of Cancer (EORTC)
 - Core Quality of Life Questionnaire (QLQ-C30) 152
 - Core Quality of Life Questionnaire module for prostate cancer (QLQ-PR25) 152
- EuroQol-5 Dimension (EQ-5D) 150, 151

- evidence-based quality improvement (EBQI)
 - methods, usage 69
- evil eye 200
- existential phenomenology 12–13
- Expanded Prostate Cancer Index Composite for Clinical Practice (EPIC-CP) 152
- expectations, distortion 3
- explanatory models 199

- F2RL3, methylation levels 37–38
- Facilitating Interdisciplinary Science (NAS) 245–246
- faith healers 2032
- Falk-Krzesinski, Holly 248
- family
 - characteristics, health variables (associations) 254–255
 - conflict, impact 254–255
 - coping 257–260
 - functioning, dimensions 258–259
 - health, relationship 253
 - impact 255
 - interaction behavior
 - dimensions 257–258
 - impact 257–260
 - well-being, relationship 259–260
 - interactions, medical condition (impact) 256–257
 - interventions 260–261
 - marriage, association 254
 - medicine, psychologists (educator function integration) 214
 - negative/positive family behavior, comparison 258
 - outcomes, medical condition (presence) 255–257
 - stress, medical condition (impact) 256
 - structure, aging (connection) 254
- Family Educational Rights and Privacy Act, regulations 193
- Family Smoking Prevention and Tobacco Control Act (FSPTCA) 24
- fatty acid amide hydrolase (FAAH), gene coding 129
- fear avoidance 3
- Federal Register* (notices/public comment solicitations) 25
- “feeling sick” (doctor-patient relationship step) 136
- femaleness, sense 219
- feminizing hormones, side effects 223–224
- fibromyalgia, functional connectivity (impact) 75
- financial accountability (ACA) 108
- financial barriers (cancer care) 91–92
- “fire together, wire together” rule 75
- folk medicine 199
- Food and Drug Administration (FDA) 246
- forming-storming-norming-performing (team development sequence) 247
- frail elderly 10
- Franklin, Rosalind 244
- Freeman, Harold P. 92
- Frieden, Thomas 22–23
- functional connectivity 74
 - health psychology, relationship 75–76
 - impact 75–76
 - modification, possibility 75
- functional magnetic resonance imaging (fMRI) 73–74
 - clinical relevance, increase (process) 76

- Galilei, Galileo 244
- gastrointestinal symptoms, palliative care
 - role 84
- gate control theory of pain 2
- gender-affirming hormone therapy 223–224
- gender dysphoria 219
- gender minority stress 220
- gene expression, control 36
- generalist palliative care, specialist palliative care (contrast) 82–83
- generally recognized as safe (GRAS) list, trans fats removal 22–23
- “genetically inferior” populations, control 45
- genetics
 - analysis, techniques 37
 - health psychology, relationship 35
 - impact 38–39
- genome-wide association studies (GWAS) 37
 - summary statistics, usage 38
- glucose abnormalities, short sleep (impact) 169
- glucose intolerance, SDB (association) 175
- glucose tolerance impairment
 - sleep improvement, impact 170–171
 - sleep loss, impact 169–170
- good death 11–14
 - achievement, medical training (impact) 16
 - IOM stance 13
 - medical practice norm, recommendations 14
 - mixed-methods study, findings 16–17
 - suffering, impact 15–16
- Group-Based Medical Mistrust Scale (GBMMS) 46–47
- Guam, prescriptive authority for psychologists (RxP) allowance 140
- guanine (G) 35–36

- Hall, Kara 247
- harmful choices, obstacles (creation) 23
- harmony, concept/impact 202
- healing
 - shamanic healing 200
 - Southern Appalachia, folk medicine healers/practices (usage) 203
 - traditional healing practices/healers 199
- health
 - American Indian beliefs 201–202
 - beliefs 201–202
 - biological/psychological/social determinants, knowledge 206
 - biopsychosocial (BPS) model 205
 - determinants, WHO description 213
 - equity 51–52
 - family, relationship 253
 - Hispanic health beliefs/practices 201
 - improvement, regulation (strategy) 22
 - inequities
 - contemporary examples 54–55
 - historical examples 54
 - insomnia
 - CBTI, impact 172–173
 - relationship 172
 - treatment effects 172–173
 - insurance status, institutional factor 44
 - practices, problems 53
 - prevention 120–121
 - promotion 120–121, 207
 - APA vision statement addition 210–211
 - sleep hours, determination 165
 - promotion (ACA) 104
 - psychologists, role 155
 - regulation, role (support) 22–23
 - sleep
 - bidirectional relationship 165, 167–176
 - duration, bidirectional relationship 168–170
 - treatment effects 170–171
 - sleep-disordered breathing (SDB), relationship 174–176
 - team science 243
 - variables, family characteristics (associations) 254–255
- Health and Activity Limitation Index (HALex) 150, 151
- Health and Society Scholars Program 31–32
- health behavior
 - family, impact 255
 - federal/state regulation, role 21
 - regulation, context 22
- healthcare
 - access, limitation 53
 - providers, perceptions 47
 - reform, “triple aim” 102
 - services, usage (racial/ethnic differences) 47
 - setting
 - non-psychologists, training 213
 - psychology, historical context 213–214
 - structural changes (ACA) 107–108
 - system
 - federal responses 105
 - suspicion, GBMMS assessment 46–47
 - transgender access 220–221
 - workforce, education/training (Carter Center recommendations) 107–108
- healthcare power of attorney (HCPOA) 10
- health disparities 43–44, 51
 - action, call 56–57
 - conceptualizations 52–53
 - contextual exposures 52
 - cultural mistrust, role 43
 - life-course approach 52, 54
 - minority women, experience 55
 - research priorities 56
 - residential segregation 53
- Health Insurance Portability and Accountability Act (HIPAA)
 - client privacy/compliance, consideration 193
 - HIPAA-related NPP 196
 - personal health information, sharing requirements 105
- Health Maintenance Consortium 31
- health psychology. *See* clinical health psychology
 - assessment/treatment, genetics (impact) 38–39
 - clinical health psychology, future 108–110
 - contribution, process 24–25
 - development 30–31
 - fMRI clinical relevance, increase (process) 76
 - functional connectivity, relationship 75–76
 - genetics, relationship 35
 - issues, molecular genetics focus 37–38
 - knowledge base, application 206
 - Patient Protection and Affordable Care Act
 - components/implications 103–108
 - relationship 101
 - training 205
 - programs 206
- health psychology research
 - fundors 29–30
 - funding 29
 - challenges/opportunities 32
 - utilization 25

- health-related quality of life (HRQoL) 149
 - definition 149–150
 - generic self-report measures 150–152
 - interventions 153
 - measurement 150–152
 - specific self-report measures 152
- Health Resources and Services Administration
 - Center, *Core Competencies for Integrated Behavioral Health and Primary Care* 215
- health service psychology
 - interprofessional education, usage 106
 - term, usage 210–211
- Health Utilities Index Mark 2 (HUI2) 150, 151–152
- Health Utilities Index Mark 3 (HUI3) 150, 151–152
- healthy behaviors, government
 - encouragement 23
- Healthy Eating Research initiative 31–32
- heart
 - disease, symptoms 83
 - failure, SDB (relationship) 175
- herb doctors 203
- herbs, usage 201
- heterosexism 52
- hidden therapies, delivery 133–134
- high temporal resolution techniques, usage 132
- Hippocrates 108
- Hispanic health beliefs/practices 201
- histone acetylation/methylation 36
- historical trauma 54
- holism, roots 12–13
- home palliative care models, evolution 81
- honey, usefulness 203
- hoodoo, term (usage) 202
- Hooke, Robert 244
- hormone therapy (transgender individuals) 223–224
- hormone use, risk factors 223–224
- Horne-Ostberg Morningness-Eveningness Questionnaire (chronotype) 167
- hospice
 - movement 12
 - services, federal regulations 82
 - usage, increase 84–85
- hospice care
 - length, impact 14
 - philosophy 82
- hospital-based palliative care consultation 80
- human existence, dimensions 12
- human immunodeficiency virus (HIV),
 - community health worker/patient navigation interventions 92–93
- hypertension development risk (increase), SDB (impact) 175
- hypopnea 174
- hypothermia (prevention), skin-to-skin care (usage) 96
- hypoxemia 174
- Illinois, prescriptive authority for psychologists (RxP) allowance 140–141
- illness
 - beliefs 201
 - BPS model conceptualization 2
 - causality, categories 200
 - outcome, doctor-patient relationship (importance) 135
 - separation (Blacks, beliefs) 202
- immune mediators, conditioning (capability) 132
- immune system
 - dysregulation 5
 - function, impairment 258
- impetigo (treatment), apple cider vinegar (usage) 202
- improvised explosive devices (IEDS), veteran exposure 233
- incarceration, disorderly transitional stressful event 55
- Indiana, prescriptive authority for psychologists (RxP) allowance 140
- Indian Health Service, prescriptive authority for psychologists (RxP) allowance 140
- individual world (illness causality category) 200
- industry self-regulation, effectiveness (variation) 23
- inflammation responses, prolongation 258
- injury
 - decrease, interventions 64
 - definition 61
 - motor vehicle injuries 62–63
 - nonfatal injuries 62
 - prevention 61
 - behaviors, adoption (factors) 64
 - road traffic injuries 62–63
 - unintentional injuries 61–62
- Inouye, Daniel 139
- in-person meetings, usage 247–248
- in-person services, usage 69
- insomnia 119, 167, 171–173
 - chronicity 171
 - cognitive behavioral therapy for insomnia (CBTI) 172–173
 - depression, relationship 172, 173
 - health, relationship 172

- multisite randomized control trial (RCT), elderly depressed patients (results) 172
- occurrence 171
- secondary insomnia 171
- Insomnia Severity Index 167
- Institute of Medicine (IOM)
 - behavioral/social science knowledge domains 215
 - IPE perspective 106
 - “Living Well and Chronic Illness” 3
 - “Unequal Treatment” report 45–46
- institutional neglect 53
- instrumental support 257
- insulin resistance
 - SDB, relationship 176
 - short sleep, impact 169
- integrated care, curriculum development/training resource 215
- integrated primary care 67
- integrated primary care behavioral health (PCBH) 67
 - care, models 68–71
 - interventions, candidates 71
 - models, focus 68
 - treatment interventions 70–71
- Intelligence Advanced Research Projects Activity (IARPA) 246
- interdisciplinary team science (interdisciplinary TS)
 - challenges 246–248
 - initiatives 246
 - institutional culture, barriers 248–249
 - NCATS, impact 249
- interleukin 6 (IL-6), chronic pain gene 6
- Interorganizational Work Group (IWG) on Competencies for Primary Care Psychology Practice, individual competencies 106–107
- interpersonal conflict, physiological stress symptoms (association) 258
- interprofessional collaboration (promotion), domains/competencies (Interprofessional Education Collaborative) 214–215
- interprofessional communication 215
- interprofessional education (IPE)
 - integration, support 105
 - perspective, Institute of Medicine (IOM) 106
- Interprofessional Education Collaborative, interprofessional collaboration domains/competencies 214–215
- interprofessional practice
 - roles/responsibilities, clarity 215
 - teams/teamwork, engagement 215
 - values/ethics 215
- interprofessional team-based care (ACA) 105–106
- Iowa, prescriptive authority for psychologists (RxP) allowance 141
- irritable bowel syndrome (IBS), placebo response 132
- Jim Crow
 - discriminatory practices 54
 - laws 52–53
- Johnson, Lyndon, national healthcare system/changes (advocacy) 101
- Joint Commission on Accreditation of Healthcare Organizations, telemedicine regulations 193
- Karolinska Sleepiness Scale (state sleepiness) 167
- Kennedy, John F., national healthcare system/changes (advocacy) 101
- ketorolac, administration/replacement 129
- laser-evoked potentials (LEPs) studies, placebos (impact) 132
- LGBTQIA, de jure discrimination/de facto discrimination 55
- life-course approach 52, 54
- life crises (coping), social relationships (maintenance) 15
- lifestyle changes, issues 70
- “Living Well and Chronic Illness” (IOM) 3
- living wills 10
- logistical barriers (cancer care) 91
- long sleep, mortality increase (association) 168
- Louisiana, prescriptive authority for psychologists (RxP) allowance 140
- magic, usage 203
- major depressive disorder (MDD), veterans experience 232
- malaria prevention practices, positive impact 96
- maleness, sense 219
- Manhattan Project 245
- marriage, family (association) 254
- masculinizing hormones, side effects 224
- massage, usage 203
- maternal health, community health worker/patient navigation interventions 96–97
- medical conditions, addressing 70
- Medical Outcomes Study 36-Item Short-Form Health Survey (SF-36) 150–151
- medical procedures, intrusiveness 11
- medical psychologist, psychologist prescriber title 140

- medical regimens, adherence (importance) 119
- Medicare reimbursement, forgoing 82
- meditation, usage 201
- “meets the therapist” (doctor-patient relationship step) 136
- mega-team authorship 244–245
- mental health conditions
 - physical health conditions, overlap 38
 - treatment, primary care setting 68
- messenger ribonucleic acid (mRNA) 36
- metabolic health, SDB (relationship) 175, 176
- methane, involvement 36
- methylation chips, construction 37
- Mexican Americans, labor/land exploitation 44
- mHealth Summer Training Institute, team science training experience 248
- midwives 203
- military
 - background information, veterans (health issues) 229
 - prescriptive authority for psychologists, impact 143
- military sexual trauma (MST) 235–236
 - problems 235
- mind (whole person component) 1
- mini-ethnography, practitioner development 203
- minority racial/ethnic populations, SES decrease 51
- minority stress 220
- minority women, health disparities 55
- misgendering 220–221
- mistrust 53
 - cultural mistrust 43–47
- mobilization-minimization hypothesis 258
- molecular biology, focus 35–36
- molecular genetics, perspective 37–38
- Molecular Transducers of Physical Activity in Humans (NIH Common Fund initiative) 31
- Moller, Arlen 248
- mortality (increase), long sleep (association) 168
- motivational interviewing (MI) 71
- motor vehicles
 - accident risk (increase), sleep-disordered breathing (impact) 174–175
 - injuries 62–63
- moxibustion, usage 201
- Multiple Sleep Latency Test (MSLT), usage 166
- μ -opioid receptor
 - agonist, analgesic efficacy (expectancies) 133
 - blockade, effect 128
 - D2/D3 receptors, activation 131*n*
 - opioid binding, occurrence 129–130
 - μ -opioid receptor (OPRM), chronic pain gene 4
- naloxone, impact 128
- Nathan Cummings Foundation 14
- National Academy of Science (NAS)
 - Facilitating Interdisciplinary Science 245–246
 - team science definition 243
- National Cancer Institute, team science funding 246
- National Center for Advancing Translational Sciences (NCATS), goal 249
- national healthcare system/changes, advocacy 101
- National Heart, Lung, and Blood Institute 31
- National Institute of Child Health and Human Development, special initiatives 31
- National Institute of Diabetes and Digestive and Kidney Diseases, special initiatives 31
- National Institute on Alcohol Abuse and Alcoholism, special initiatives 31
- National Institute on Drug Abuse
 - special initiatives 31
 - team science funding 246
- National Institutes of Health (NIH)
 - Brain Research through Advancing Innovative Neurotechnologies (BRAIN) initiative 246
 - extramural research 244
 - Guide for Grants and Contracts 31
 - health psychology research funding 29
 - requests for applications (RFAs) 31
 - special initiatives support 32
- National Palliative Care Registry, staffing study 80
- National Working Conference on Education and Training in Health Psychology (Arden House Conference) 205–206
- Native Americans, genocide 44
- nativism 52
- natural history 127
- natural illnesses 202
- natural world (illness causality category) 200
- Need for a New Medical Model, The* (Engel) 213
- needle phobias (reduction), psychologists (impact) 160
- needs assessment (telepsychology) 190–191
- negative beliefs/feelings, internalization 219
- negative coping 257
- negative eugenics 45
- neuroimaging 73

- neuromatrix theory of pain 2, 4
- New Mexico
 - Medicaid payments, prescribing psychologist acceptance 143–144
 - prescriptive authority for psychologists (RxP) allowance 140
- Nixon, Richard, national healthcare system/changes (advocacy) 101
- nocebo
 - hyperalgesia (mediation), CCK pro-nociceptive system (impact) 129
 - modulation 131*n*
- nonadherence, impact 119
- nonbinary identities, sense 219
- noncommunicable diseases, community health worker/patient navigation interventions 94–96
- nonfatal injuries 62
- nonfatal unintentional injuries 62
- nonpsychiatric physicians (demand reduction), prescribing psychologists (increase/impact) 143
- non-psychologists
 - education, learning environment (enhancement) 214–216
 - healthcare setting training 213
 - psychologists, educator function 214
- non-rapid eye movement (NREM) 165
- Nord-Trøndelag Health Study (HUNT) 172
- Notice of Privacy Protection (NPP) 196
- nutrient content, format (change) 23
- nutrition information
 - availability 23–24
 - truthfulness, government requirement 22
- Nutrition Labeling and Education Act (NLEA), health claims updates 23
- Obama, Barack, national healthcare system/changes (advocacy) 101
- obesity, health disparity factor 44
- Obesity-Related Behavioral Intervention Trials (ORBIT) initiative 31
- obstructive sleep apnea (OSA) 174
 - metabolic disturbances 176
- OEF/OIF veterans, study 231, 235
- Office of Behavioral and Social Sciences Research (OBSSR)
 - establishment 30
 - special initiatives 31
- older adult pedestrians, pedestrian-vehicle collision (injury/death) 63
- oncology, pediatric psychologists (impact) 122
- one-to-one coaching 159
- operating room (OR)
 - psychologist considerations 161–162
 - speed/decisiveness, importance 156
 - surgical hierarchy, respect 157
- osteopathic medicine 1–2
- outpatient palliative care programs, impact 81
- overweight (treatment), apple cider vinegar (usage) 202
- oxytocin
 - trust-inducing properties 136
 - usage 136
- pain. *See* chronic pain
 - behaviors 2
 - biomedical model 2
 - control procedures, nurse/doctor usage 13
 - dimensions 2
 - gate control theory 2
 - management 121
 - multimodal strategy 83
 - neuromatrix theory 2, 4
 - occurrence 120
 - palliative care role 83
 - pediatric psychology, relationship 120
 - placebo responsiveness, dopamine (involvement) 129–130
 - prevention/management, neglect 14
 - relief, production 15
 - sleep, bidirectional relationship 119–120
 - subjective perception 2
 - total pain 15
- palliative care 17
 - ambulatory palliative care programs, focus 81
 - breathlessness 84
 - chronically ill, caring 86
 - communication skills (increase), educational interventions (usage) 85
 - conversations, mental models 85
 - core items, adult clinician performance 83
 - definition 79
 - delivery, models 79–81
 - emergency department (ED), palliative care (providing) 80–81
 - gastrointestinal symptoms 84
 - generalist palliative care, specialist palliative care (contrast) 82–83
 - goals, discussion 84–85
 - growth (US) 79–81
 - home palliative care models, evolution 81
 - hospital-based palliative care consultation 80
 - multimodal strategy 83

- palliative care (*cont'd*)
 - outpatient palliative care programs, impact 81
 - pain 83
 - population, cachexia/anorexia (presence) 84
 - programs, National Palliative Care Registry study 80
 - roles 83–86
 - specialty palliative care services, consultant model 80
 - symptom management 83
 - team, presence (factors) 80
- panic disorder, veterans experience 232
- parasomnias 119
- parent-based interventions 260
 - effectiveness 260–261
- Parkinson's disease
 - patients, placebo treatment (intraoperative single-neuron recording) 131
- Parkinson's disease (placebo responsiveness), dopamine (involvement) 129–130
- patient-centered healthcare homes (PCMHs), impact 107
- patient navigation
 - community health workers, relationship 91
 - development, reason 94–95
 - efficacy, impact 95
 - interventions, conceptualizations 92
 - program, creation 92
- Patient Protection and Affordable Care Act (ACA) 102–103
 - bylaws, “health” (term, addition) 103
 - components/implications 103–108
 - demographic shifts 103–104
 - disease prevention 104
 - health promotion 104
 - health psychology, relationship 101
 - implementation, issues 102
 - technology, impact 104–105
- Patient-Reported Outcomes Measurement Information System Global Health Scale (PROMIS Global Health) 150, 152
- Patient-Reported Outcomes Measurement Information System (PROMIS) Roadmap (NIH initiative) 152
- patients
 - attitudes, importance 47
 - cognitive functioning level, evaluation 159–160
 - IPCBH provider, PCP introduction 67
 - patient-practitioner relationship, augmentation 134
 - satisfaction, perceptions 47
 - social supports, evaluation 159–160
 - stress (surgery) 157–158
- pedestrian injury, risk (impact) 63
- pediatric immunization, community health worker/patient navigation interventions 96–97
- pediatric psychologists
 - activities, summary 115
 - adherence/nonadherence roles 119
 - consultation-liaison (CL) services 118
 - eHealth, usage 123
 - mentorship 123
 - psychosocial treatments 121
- pediatric psychology 115
 - clinical practice/research, future 123–124
 - ethical issues 117–118
 - examples 121–123
 - future 123–124
 - historical foundations 116–117
 - issues 118–121
 - perspectives/systems 117
- pediatric sleep disorders 119–120
- phenotypes, genetic correlations (estimation) 38
- physical development, vulnerability 55
- physical health conditions, mental health conditions (overlap) 38
- physical nociception 2
- physical suffering, study 15
- physician-assisted suicide, legalization (occurrence) 17
- physician orders for life-sustaining treatment (POLST) 10
- physician-patient communication, improvement 13
- physiological tolerance, threshold (excess) 61
- Pittsburgh Sleep Quality Index (global sleep quality) 167
- placebo analgesia
 - blocking, CB1 cannabinoid receptor antagonist (impact) 129
 - D2/D3 receptors, activation 131*n*
 - enhancement 128–129
 - expectations/drugs, additive/synergistically interactive manner (determination) 133
 - prevention, naloxone (impact) 128
 - response (enhancement), oxytocin (usage) 136
- placebo effect 127
 - definition 127–128
 - doctor-patient relationship 134–137
 - experimental separation 134
 - physical/cognitive performance 137

- psychobiological mechanisms 128–132
- psychobiological phenomenon 127–128
- placebo responsiveness
 - dopamine, involvement 129–130
 - genetic variants, association 132
- placebos
 - modulation 131*n*
 - placebo-treatment Parkinsonian patients,
 - intraoperative single-neuron recording 131
 - response, neurobiological mechanisms 130
 - treatments, expectations (absence) 132–134
- polysomnography (PSG), usage 166
- population-based care, IPCBH models (focus) 68
- positive eugenics 45
- positron emission tomography (PET) 73, 74
- posttraumatic stress disorder (PTSD)
 - cardiometabolic conditions, association 38
 - scores, range 76
 - veterans
 - experience 231, 234–235
 - prevalence, increase 235
- prayer, usage 203
- prescriptive authority for psychologists (RxP) 139
 - approval/adoption 140
 - care, access 143–144
 - controversy 141–145
 - effectiveness, studies 142–143
 - ethical implications 145
 - financial/professional incentives 144
 - identity, loss 144–145
 - practices, building 144
 - safety, issue 142–143
 - training
 - contention 141–142
 - differences 141–142
- prescriptive authority movement, history 139
- primary care
 - delivery (improvement), PCMH (impact) 107
 - high-functioning teams, development (student education) 107
- primary care mental health integration (PCMH-I) 67
- primary care settings
 - behavioral conditions, incidence/prevalence (provider education) 107–108
 - pediatric psychologists, work 118–119
 - telepsychology services, increase (anticipation) 194
- professional psychology, term (replacement) 210–211
- proglumide, CCK antagonist (impact) 128–129
- project management tools, usage 247–248
- prolonged exposure (PE), usage 235
- prostacyclin, formation 129
- prostaglandins (PG), synthesis 129
- protective buffering 257
- psychiatric/emotional duress, freedom 17
- psychoeducation, usage 69
- psychological coping mechanisms 203
- psychological principles/theories, knowledge 206
- psychological problems, insomnia (impact) 172
- psychological services, videoconferencing equipment (requirement) 191
- psychologists
 - clinical care, providing 160
 - clinician function 159–160
 - consultant function 160–161
 - educator function 158–159, 214
 - emergency consultation 161–162
 - prescriptive authority 139
 - training initiatives 159
- psychology workforce, impact 109–110
- psychopathology 5
- psychopharmacology, APA task force 140
- Psychopharmacology Demonstration Project 139
 - evaluations 142
 - termination 140
- Psychopharmacology Examination for Psychologists (PEP), APA Practice Organization development 140
- public health outcomes (improvement), regulation (impact) 22–23
- Public Health Service, prescriptive authority for psychologists (RxP) allowance 140
- quality-adjusted life years (QALYs)
 - calculation 150
 - usage 152
- quality care (ACA) 106–107
- quality of care, death recognition 13
- quality of life (QoL) 149
 - definition 149
- Quality of Well-Being Scale Self-Administered (QWB-SA) 150, 152
- racial discrimination 44–45, 52–53
- racial residential discrimination 54
- racism 52

- rapid eye movement (REM) 165
 - stage sleep, discrimination (impact) 53
- “receives the therapy” (doctor-patient relationship step) 136
- redlining, historical practice 54
- regional cerebral blood flow, assessment 131–132
- regulation
 - context 22
 - definition 21
 - examples 23–24
- regulatory science, definition 24–25
- Reiki, usage 203
- remifentanyl, analgesic efficacy (expectancies) 133
- renal disease, symptoms 832
- repetitive transcranial magnetic stimulation (rTMS) 75
- Report to the Nation* (American Cancer Society) 91–92
- requests for applications (RFAs) 31
- residential segregation 53
- resting-state functional connectivity (RSFC)
 - MRI 74
 - patterns 75
 - within-subject RSFC designs, impact 75
- ribonucleic acid (RNA) 36
- rimonabant, impact 129
- road traffic injuries 62–63
- Robert Wood Johnson Foundation
 - health psychology research funding 30
 - special initiatives 31
 - team science funding 246
- Roosevelt, Franklin, national healthcare system/changes (advocacy) 101
- Roosevelt, Theodore, national healthcare system/changes (advocacy) 101
- root doctors, botanicals (usage) 202
- S*-adenosylmethionine, production 38
- salivary oxygen, reduction 129
- Saunders, Cicely 15
- Science of Behavior Change initiative (NIH Common Fund support) 31
- science/scientists, specialization 245
- screening mammography, uptake rate (increase) 94
- secondary insomnia 171
- “seeking relief” (doctor-patient relationship step) 136
- selective serotonin reuptake inhibitors (SSRIs)
 - responders, pretreatment/posttreatment 132
 - usage, double-blind conditions 131–132
- self-control, improvement 120
- self-management 121
 - parental involvement 119
- self-report questionnaires, usage 167
- sensation seeking, temperamental
 - characteristic 63
- sentiment override 259
- serotonin transporter-linked polymorphic region (5-HTTLPR), genotyping 132
- sexism 52
- sexually transmitted infections (STIs), transgender individuals 222
- sexual minorities, de jure discrimination/de facto discrimination 55
- shamanic healing 200
- Short Form-6 Dimension (SF-6D) 151
- short sleep
 - associations 168
 - chronic short sleep, metabolic effects (reversal) 170
 - impact 169
- simulation exercises 159
- sleep
 - actigraphy, usage 166
 - apnea 174
 - severity, measurement 174–175
 - assessment 166–167
 - chronic sleepiness 169
 - definition 165
 - diary, usage 167
 - disruption, experience 169
 - duration, health (relationship) 168–170
 - health, bidirectional relationship 165, 167–176
 - inadequacy, consequences 165–166
 - long sleep, mortality increase (association) 168
 - Multiple Sleep Latency Test (MSLT), usage 166
 - pain, bidirectional relationship 119–120
 - pediatric psychologists, impact 119–120
 - polysomnography (PSG), usage 166
 - protective mechanism, demonstration 170
 - quantity, inappropriateness 167–171
 - self-report questionnaires, usage 167
 - short sleep, associations 168, 169
 - states 165
 - type 2 diabetes mellitus (T2DM), reciprocal relationship 169–170
 - veterans, problems 232–233
- sleep-disordered breathing (SDB) 167, 174–176
 - cardiovascular/metabolic health, relationship 175, 176

- continuous positive airway pressure (CPAP)
 - therapy, usage 175–176
- health, relationship 174–176
- types 174
- smoking
 - health psychology issues 37–38
 - smoking-associated CpG sites 37
 - transgender individuals 222
- social anxiety disorder
 - PET, usage 131–132
 - serotonin transporter-linked polymorphic region (5-HTTLPR), genotyping 132
- social relationships, maintenance 15
- social support, model 257
- social world (illness causality category) 200
- Society of Pediatric Psychology (SPP)
 - founding 116
 - Mission Statement/Vision Statement 116
- sociocultural barriers (cancer care) 91
- socioeconomic immobility 53
- socioeconomic status (SES)
 - decrease (minority racial/ethnic populations) 51
 - diminishing returns hypothesis 51
- socioeconomic status (SES) level
 - health disparity factor 44
 - increase, impact 51
- solution-focused/problem-solving therapy 61
- sorcery 201
- sore throat (treatment), apple cider vinegar (usage) 202
- Southeast Asian groups, illness beliefs 201
- Southern Appalachia, folk medicine healers/practices (usage) 203
- special initiatives, role 30–32
- specialist palliative care, generalist palliative care (contrast) 82–83
- specialization (ACA) 108
- specialty palliative care services, consultant model 80
- spirit (whole person component) 1
- spiritual/existential beliefs 16
- Spring, Bonnie 248
- Standards of Care (SOC) for the Health of Transsexual, Transgender, and Gender Nonconforming People* (WPATH) 223
- Steinhauser, Karen 16
- sterilization laws 45
- STOP-BANG (sleep apnea risk) 167
- stress response 258
- stroke, SDB (relationship) 175
- student exchanges, usage 247–248
- Substance Abuse and Mental Health Services Administration
 - Core Competencies for Integrated Behavioral Health and Primary Care* 215
 - health psychology research funding 29
- substance use disorders (transgender individuals) 221
- suffering
 - economic demands, reality 15
 - physical suffering 15
 - psychological symptoms 15
 - situations/dimensions 15–16
 - spiritual/existential beliefs 16
- suffering, trigger 2
- suicidal ideation, veterans (experience) 232
- suicidality (transgender individuals) 222
- suicide, veterans (problem) 236–237
- supernatural world (illness causality category) 200
- SUPPORT end-of-life care study 13–14
- supportive coping 257
- surgeons
 - action orientation 156
 - board certification 162
 - stress 157–158
- surgery
 - action-oriented structure 156–157
 - health psychologists, role 155
 - psychologists
 - clinician function 159–160
 - consultant function 160–161
 - educator function 158–159
 - training initiatives 159
 - psychology, roles 158–161
- surgical clinical health psychologist, “curbside” consultation 160
- surgical culture, observation (importance) 156–158
- surgical environment, psychologist (functioning) 157
- surgical faculty, psychologist importance (perception) 158
- surgical hierarchy 156–158
 - respect 157
- Sutter Health Program, Advanced Illness Management 81
- symptom management, palliative care role 83
- synthetic DNA, fluorescence 37
- T1 D (endocrine disorder) 122–123
- Tao, concept 201

*Taxonomy for Education and Training in
Professional Psychology Health Service
Specialties* 209

team development sequence 247

team science (TS)

competencies, training 248

cross-institutional teams, impact 247–248

drivers, NAS attribution 245

emergence 244–245

institutional culture, barriers 248–249

interdisciplinary team science

challenges 246–248

initiatives 246

mega-team authorship 244–245

National Academy of Science (NAS)

definition 243

reasons 245–246

science/praxis, definitions 243

support, institutional culture (optimization)
249–250

telebehavioral health 189

teleconferences, usage 247–248

telemedicine, Joint Commission on Accreditation
of Healthcare Organizations
regulations 193

telemental health 189

guidelines, availability 192

telephone assessments, usage 69

telepsychologists, practice 194

telepsychology 189

clients, selection 194–195

clinic, establishment 190–194

coordinator (telefacilitator),
responsibilities 192

ethical/regulatory considerations 192–193

expansion, factors 189–190

needs assessment 190–191

post-session 197

practice 194–197

pre-session preparation 196

protocols 192

referral/scheduling, process 196

reimbursement 193

services

changes 190

increase, anticipation 194

session, clinical encounter (technological
factors) 195

sustainability 193–194

technology selection 191

video quality/camera placement,
importance 1905

televideo

clinic room, setup 191

etiquette, client-provider relationship 195

post-session 197

psychological services, regulation
(limitation) 192–193

services

coverage, Current Procedural Terminology
(CPT) codes (determination) 193

services, quality (improvement) 191

session, elements 196

telomeres, shortening 4

Tempe Summit on Education and Training
in Clinical Health Psychology
206–207, 208

terminal illness

coping frameworks 12

diagnosis, corroboration (state
requirement) 17

terminally ill, sustaining (counterforce) 12

thromboxane (TX), synthesis 129

thymine (T) 35–36

tobacco use (transgender individuals) 222

Toolbox Project (NSF) 248

total pain 15

traditional healers 199

categories 202, 203

traditional healing practices 199

traditional medicine, growth 199

Transdisciplinary Research on Energetics and
Cancer Centers (TREC)

special initiatives 31

team science initiative 246

Transdisciplinary Tobacco Use Research Centers
(TTURC)

co-funding 31

team science 246

trans fats, bans/GRAS list removal 23

transgender health 219

healthcare access 220–221

social context 219–220

transgender identity, mistreatment (fear) 220

transgender individuals

alcohol use disorders 221

cancer 221

cardiovascular disease 222

depression 222

health-related issues 221–223

hormone therapy 223–224

smoking use 222

substance use disorders 221

suicidality 222

- surgical procedures 224
- tobacco use 222
- transition-related health issues 223
- treatments, regulation (absence) 224–225
- weight 222–223
- transgender stigma/consequences, understanding (importance) 220
- transitioning 219
- Translating Initiatives for Depression into Effective Solutions (TIDES) 68–69
 - development 69
- traumatic brain injuries (TBIs)
 - mental health problems, comorbidities 233
 - veterans, experience 233–234
- treatment, patient adherence (importance) 47
- Truman, Harry, national healthcare system/changes (advocacy) 101
- trust
 - behavior (hormonal modulation), oxytocin (usage) 136
 - cultural mistrust 43–47
 - promotion 48
- trustworthy behavior, neural circuitry 136
- tryptophan hydroxylase-2 (TPH2) gene promoter, G-703T polymorphism 132
- tuberculosis
 - community health worker/patient navigation interventions 93–94
 - directly observed treatment (DOT) 93–94
- Tuckman, Bruce 247
- Tuskegee syphilis study 44
- type 2 diabetes mellitus (T2DM)
 - progression, stages 170
 - risk, development 171
 - short sleep, impact 168, 169
 - sleep, reciprocal relationship 169–170
- “Unequal Treatment” (IOM report) 45–46
- unintentional injuries
 - defining 61
 - nonfatal unintentional injuries 62
 - occurrence 62
 - prevention 63–64
 - issue, addressing (difficulty) 64
 - randomness/characteristics 63
 - risk factor 63
 - threat, proportion 62
- United States
 - older population, growth (acceleration) 104
 - palliative care, increase 79–81
 - population, racial/ethnic diversity (increase) 104
 - unnatural illnesses 202
 - US Transgender Survey (USTS) 220
 - Val/Val homozygotes, placebo responses 132
 - ventilation, reduction 129
 - Vermont, folk medicine 202–203
 - veterans
 - abdominal/limb pain 230
 - chronic pain 230–231
 - cognitive processing therapy (CPT), usage 235
 - definition 229–230
 - demographic information 229–230
 - depression 231
 - headaches 230
 - health issues 229, 230
 - improvised explosive devices (IEDS) exposure 233
 - joint/back pain 230
 - military sexual trauma (MST) 235–236
 - OEF/OIF veterans, study 231, 235
 - physical/emotion problems 231
 - posttraumatic stress disorder (PTSD) experience 231, 234–235
 - prevalence, increase 235
 - prolonged exposure (PE), usage 235
 - sleep problems 232–233
 - suicide, problem 236–237
 - traumatic brain injury (TBI) 232–234
 - Veterans Administration (VA) medical centers, palliative care (family member survey) 80
 - Veterans Health Administration IPCBH (VHA IPCBH) programs 68
 - victim, blaming 46
 - videoconferencing
 - pre-session preparation 196
 - security, telepsychology benefits 189
 - services, implementation (factors) 191
 - video quality/camera placement, importance 195
 - virtual clinics, usage 69
 - Virtual Integrated Multisite Patient Aligned Care Team (V-IMPACT) 71
 - vitamin therapy, usage 203
 - voodoo 200, 202
 - warm handoffs 67
 - watchful waiting 69
 - Watson, James 244
 - weathering 52
 - weight (transgender individuals) 222–223
 - Weiss, Stephen M. 205

- well-being, family interaction behavior
 (relationship) 259–260
- White River Model of Colocated Collaborate
 Care 68, 69
 - open-access model 69
- whole person, components 2
- Wilkins, Maurice 244
- Williams, J. Corey 45
- witchcraft 200, 201, 203
- within-subject RSFC designs, impact 75
- World Health Organization Quality of Life
 (WHOQOL) assessments 150, 151
- World Professional Association for Transgender
 Health (WPATH) 223
- mental health assessment,
 - recommendation 224
- Standards of Care (SOC) for the Health of
 Transsexual, Transgender, and Gender
 Nonconforming People* 223
- Wright, Logan 116–117
- yin/yang, forces 201
- young people, unintentional injuries 62
- Young, Thomas 244
- youth
 - health maintenance, medical regimen
 adherence (importance) 119
 - sleep problems, presence 120

WILEY END USER LICENSE AGREEMENT

Go to www.wiley.com/go/eula to access Wiley's ebook EULA.