

The
INTERNATIONAL
ENCYCLOPEDIA
of
DEPRESSION



EDITOR
RICK E. INGRAM

THE INTERNATIONAL ENCYCLOPEDIA OF DEPRESSION

RICK E. INGRAM, PhD

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Rick E. Ingram received his PhD in Clinical Psychology at the University of Kansas. The first part of his career was spent at San Diego State University where he was a professor of psychology and a core member in the joint San Diego State University–University of California at San Diego Doctoral Training Program in clinical psychology. He is currently a professor of psychology at the University of Kansas and serves as the editor of *Cognitive Therapy and Research* and as associate editor for the *Journal of Consulting and Clinical Psychology*. He has over 100 publications and over 175 conference presentations, mainly in the area of depression. He has authored or edited six books, the most recent of which are *Vulnerability to Psychopathology: Risk Across the Lifespan* (R. E. Ingram & J. Price, Eds., 2001) and *Cognitive Vulnerability to Depression* (R. E. Ingram, J. Miranda, & Z. V. Segal, 1998). He is a fellow of Division 12 of the American Psychological Association, and a founding fellow of the Academy of Cognitive Therapy. He is also the recipient of the Distinguished Scientific Award of the American Psychological Association for Early Career Contributions to Psychology, and earlier received the New Researcher Award from the Association for the Advancement of Behavior Therapy.

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CONTRIBUTORS

John R. Z. Abela, PhD

Department of Psychology
McGill University
Montreal, Quebec
Canada

*Attributional Theories of
Depression, Diatheses-
Stress Models of
Depression*

Heather C. Abercrombie, PhD

Department of Psychiatry
University of Wisconsin
School of Medicine and
Public Health
Madison, Wisconsin
*Hypothalamic Pituitary
Adrenal Axis*

Emma K. Adam

Program on Human
Development and Social
Policy
School of Education and
Social Policy
Cells to Society Center,
Institute for Policy
Research
Northwestern University
Evanston, Illinois
Cortisol

Howard Aizenstein, MD, PhD

Western Psychiatric
Institute and Clinic
University of Pittsburgh
School of Medicine
Pittsburgh, Pennsylvania
*Computer Simulation
of Depression and
Depressive Phenomena*

George S. Alexopoulos, MD

Weill Medical College
Cornell University
Ithaca, New York
Director
Weill-Cornell Institute of
Geriatric Psychiatry
White Plains, New York
*Depression-Executive
Dysfunction Syndrome*

Nicholas Allen, PhD

University of Melbourne
Melbourne, Victoria,
Australia
*Childhood Depression:
Family Context*

Adam K. Anderson, PhD

Department of Psychology
University of Toronto
Toronto, Ontario, Canada
Affective Neuroscience

Bernice Andrews, PhD

Department of
Psychology
Royal Holloway
University of London
London, England
Shame

Jules Angst, MD

Zurich University
Psychiatric Hospital
Zurich, Switzerland
*Brief Recurrent
Depression*

Shelli Avenevoli, PhD

Division of Developmental
Translational Research

National Institute of
Mental Health
National Institutes of
Health
Bethesda, Maryland
*Continuity of Depression
Across Development*

R. Michael Bagby, PhD

Center for Addiction and
Mental Health
University of Toronto
Toronto, Ontario, Canada
*Personality Disorders and
Depression*

Michael J. Baime, PhD

Department of Psychology
University of Pennsylvania
Philadelphia, Pennsylvania
Attention

Ross J. Baldessarini, MD

Department of Psychiatry
Harvard Medical School
Boston, Massachusetts
*Bipolar Disorder
Treatment: Lithium*

Harriet A. Ball, MD

Medical Research Council
Social Genetic and
Developmental Psychiatry
Centre
Institute of Psychiatry
King's College
London, England
Twin Studies

Jacques P. Barber, PhD

Center for Psychotherapy
Research

- Department of Psychiatry
University of Pennsylvania
School of Medicine
Philadelphia, Pennsylvania
Psychodynamic Therapy
- Russel A. Barkley**
Medical University of
South Carolina
Charleston, South
Carolina
SUNY Upstate Medical
University
Syracuse, New York
*Attention Deficit
Hyperactivity Disorder*
- Alinne Z. Barrera, PhD**
Department of Psychiatry
University of California at
San Francisco
San Francisco, California
Prevention
- Steven R. H. Beach, PhD**
Department of Psychology
University of Georgia
Athens, Georgia
Marital Therapy
- Christopher G. Beevers, PhD**
Department of Psychology
University of Texas at
Austin
Austin, Texas
*Cognitive Vulnerability:
Genetic Associations*
- German E. Berrios, MD**
Department of Psychiatry
University of Cambridge
Cambridge, England
History of Depression
- Namgyal Bhutia, MD**
Psychiatric Resident
UMDNJ-New Jersey
Medical School
Newark, New Jersey
Electroconvulsive Therapy
- Antonia Bifulco, PhD**
Lifespan Research Group
Royal Holloway,
University of London
London, England
Early Adversity
- Steven L. Bistricky, MA**
Department of Psychology
University of Kansas
Lawrence, Kansas
*Dysfunctional Attitudes
Scale, Minor Depression*
- Rebecca E. Blanton, PhD**
Department of Psychology
University of Southern
California
Los Angeles, California
Hormones
- Sidney J. Blatt, PhD**
Department of Psychology
Yale University
New Haven, Connecticut
*Anaclitic and Introjective
Depression*
- Dan G. Blazer, MD, PhD**
Department of Psychiatry
and Behavioral Sciences
Center for the Study
of Aging and Human
Development
Duke University Medical
Center
Durham, North Carolina
*Postmenopausal
Depression*
- Carol Brayne, PhD**
University of Cambridge
Parkinson's Disease
- Patricia A. Brennan, PhD**
Department of Psychology
Emory University
Atlanta, Georgia
*Family Transmission of
Depression*
- Chris R. Brewin, PhD**
Subdepartment of Clinical
Health Psychology
University College London
London, England
Intrusive Memory
- Tabetha Brockman, MA**
Department of Psychiatry
and Psychology
Mayo Clinic College of
Medicine
Rochester, Minnesota
Smoking
- Caroline B. Browne, PhD**
Department of Psychology
University of North
Carolina at Chapel Hill
Chapel Hill, North
Carolina
Peer Relations
- Lisa D. Butler, PhD**
Department of Psychiatry
and Behavioral Sciences
Stanford University School
of Medicine
Stanford, California
Cancer
- Turhan Canli, PhD**
Department of Psychology
Stony Brook University
Stony Brook, New York
*Genetic Transmission of
Depression*
- Paul Carey, FCPsych, PhD**
Department of Psychiatry
University of Stellenbosch
Stellenbosch, South Africa
*Human Immunodeficiency
Virus*
- Robert M. Carney, PhD**
Department of Psychiatry
Washington University
School of Medicine
St. Louis, Missouri
Heart Disease
- Bernard J. Carroll, MBBS,
PhD, FRCPsych**
Pacific Behavioral
Research Foundation
Carmel, California
*Dexamethasone
Suppression Test*
- Christine Chang-Schneider,
PhD**
Department of Psychology
University of Texas at
Austin
Austin, Texas
Self-Verification
- Iwona Chelminski, PhD**
Department of Psychiatry
and Human Behavior
Brown University School
of Medicine

- Providence, Rhode Island
*The Clinically Useful
Depression Outcome Scale*
- Bruce F. Chorpita, PhD**
Department of Psychology
University of Hawai'i at
Manoa
Manoa, Hawai'i
Internalizing Disorders
- Katherine H. Clemans, MA**
Department of Psychology
University of Florida
Gainesville, Florida
Childhood Depression
- Kimberly A. Coffey, MA**
Department of Psychology
University of North
Carolina at Chapel Hill
Chapel Hill, North
Carolina
*Positive Emotion
Dysregulation*
- Sheldon Cohen, PhD**
Department of Psychology
Carnegie Mellon
University
Pittsburgh, Pennsylvania
Social Support
- Jennifer Connolly, PhD**
Department of Psychology
York University
Toronto, Ontario, Canada
Romantic Relationships
- Jennifer K. Connor-Smith, PhD**
Department of Psychology
Oregon State University
Corvallis, Oregon
Stress and Coping
- Yeates Conwell, MD**
Center for the Study and
Prevention of Suicide
University of Rochester
School of Medicine
Rochester, New York
Suicide in the Elderly
- Rena Cooper-Kazaz, MD**
Biological Psychiatry
Laboratory
Department of Psychiatry
Hadassah-Hebrew
University Medical Center
- Jerusalem, Israel
Thyroid Function
- E. Jane Costello, PhD**
Department of Psychology
Duke University
Durham, North Carolina
*Age of Onset of
Depression*
- James C. Coyne, PhD**
Abramson Cancer Center
University of Pennsylvania
Philadelphia, Pennsylvania
*Marital Functioning and
Depression*
- Jorden A. Cummings, MA**
Department of Psychology
University of Delaware
Newark, Delaware
*Cognitive Behavioral
Therapy*
- Jill M. Cyranowski, PhD**
Department of Psychiatry
Department of Psychology
University of Pittsburgh
School of Medicine and
Medical Center
Pittsburgh, Pennsylvania
*Medical Conditions and
Depression*
- Boldizsár Czéh, MD**
Clinical Neurobiology
Laboratory
German Primate Center
Göttingen, Germany
Hippocampus
- Tim Dalgleish, PhD**
Emotion Research Group
Cognition and Brain
Sciences Unit
Medical Research Council
Cambridge, England
Differential Activation
- Collin L. Davidson, MA**
Oklahoma State
University
Stillwater, Oklahoma
Stress Generation
- Betsy Davis, PhD**
Oregon Research Institute
Eugene, Oregon
- Childhood Depression:
Family Context*
- Asli Demirtas-Tatlidede, MD**
Berenson-Allen Center
for Noninvasive Brain
Stimulation
Beth Israel Deaconess
Medical Center
Harvard Medical School
Boston, Massachusetts
*Transcranial Magnetic
Stimulation*
- Elizabeth DeOreo, MD**
Department of Psychiatry
and Behavioral Sciences
Emory University School
of Medicine
Atlanta, Georgia
*Hypothalamic-Pituitary-
Thyroid Axis*
- Richard A. Depue, PhD**
Department of Human
Development
Cornell University
Ithaca, New York
Dopaminergic Systems
- Mary Amanda Dew, PhD**
Department of Psychiatry
Department of Psychology
Department of
Epidemiology
University of Pittsburgh
School of Medicine and
Medical Center
Pittsburgh, Pennsylvania
*Medical Conditions and
Depression*
- Guy Diamond, PhD**
Children's Hospital
of Philadelphia and
University of Pennsylvania
Philadelphia, Pennsylvania
*Family and Parent-Child
Therapy*
- Sona Dimidjian, PhD**
Department of Psychology
University of Colorado
Boulder, Colorado
*Behavioral Models of
Depression*

- Danielle S. Duggan, PhD**
 Department of Psychiatry
 University of Oxford
 Warneford Hospital
 Oxford, England
Suicidal Cognition
- Todd W. Dunn, MA**
 Department of Psychiatry
 Southwestern Medical
 Center
 University of Texas
 Dallas, Texas
Psychosocial Functioning
- Yulia Chentsova Dutton, PhD**
 Department of Psychology
 Georgetown University
 Washington, D.C.
Culture and Depression
- Kari M. Eddington, PhD**
 Department of Psychology
 University of North
 Carolina at Greensboro
 Greensboro, North
 Carolina
*Neuroimaging and
 Psychosocial Treatments
 for Depression*
- Anne Farmer, MD**
 Medical Research Council
 Social, Genetic, and
 Developmental Psychiatry
 Centre
 Institute of Psychiatry
 King's College
 London, England
Genetics of Depression
- Frank D. Fincham, PhD**
 Department of Psychology
 Florida State University
 Tallahassee, Florida
Marital Therapy
- Philip A. Fisher, PhD**
 Oregon Social Learning
 Center and Center for
 Research to Practice
 Eugene, Oregon
Maltreatment
- Srnka J. Flegar, MD**
 Department of Psychiatry
 and Mental Health
 Observatory
 Groote Schuur Hospital
 University of Cape Town
 Cape Town, South Africa
*Dermatology and
 Depression*
- Gordon L. Flett, PhD**
 Department of Psychology
 York University
 Toronto, Ontario, Canada
Perfectionism
- Erika E. Forbes, PhD**
 Department of Psychiatry
 University of Pittsburgh
 Pittsburgh, Pennsylvania
Neural Systems of Reward
- Joseph C. Franklin, MA**
 Department of Psychology
 University of North
 Carolina at Chapel Hill
 Chapel Hill, North
 Carolina
Peer Relations
- Barbara L. Fredrickson, PhD**
 University of North
 Carolina at Chapel Hill
 Chapel Hill, North
 Carolina
*Positive Emotion
 Dysregulation*
- Kenneth E. Freedland, PhD**
 Department of Psychiatry
 Washington University
 School of Medicine
 St. Louis, Missouri
Heart Disease
- Eberhard Fuchs, MD**
 Clinical Neurobiology
 Laboratory
 German Primate Center
 DFG Research Center
 Molecular Physiology of
 the Brain
 University of Göttingen
 Department of Neurology
 Medical School
 University of Göttingen
 Göttingen, Germany
Hippocampus
- Daniel Fulford, MA**
 Department of Psychology
 University of Miami
 Coral Gables, Florida
Bipolar Disorders
- Melinda A. Gaddy, BA**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
*Anger, Endogenous and
 Reactive Depression,
 Global Burden of
 Depression*
- Krauz Ganadjian, MD**
 Resident Physician
 Department of Psychiatry
 University of California at
 San Diego
 San Diego, California
Bereavement
- Robert W. Garland, PhD**
 Private Practice
 San Jose, California
Cancer
- Genevieve M. Garratt, PhD**
 Sacramento, California
Cognitive Meditation
- Brandon E. Gibb, PhD**
 Department of Psychology
 Binghamton University
 (SUNY)
 Binghamton, New York
*Developmental
 Antecedents of
 Vulnerability*
- Paul Gilbert, PhD, FBPsS**
 Mental Health Research
 Unit
 Kingsway Hospital
 Kingsway, Derby, England
Evolution
- Omri Gillath, PhD**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
Attachment
- Jane Gillham, PhD**
 Swarthmore College
 Swarthmore,
 Pennsylvania
 University of Pennsylvania
 Pittsburgh, Pennsylvania
*Family and Parent-Child
 Therapy*

- Michael J. Gitlin, MD**
Department of Psychiatry
Geffen School of Medicine
at UCLA
Los Angeles, California
*Monoamine Oxidase
Inhibitors*
- Layne A. Goble, PhD**
Psychology Service
Veterans Administration
Connecticut Healthcare
System
Yale University
New Haven, Connecticut
Chronic Pain
- Sherryl H. Goodman, PhD**
Department of Psychology
Emory University
Atlanta, Georgia
Mothers and Depression
- Araceli Gonzalez, MA**
Department of Psychology
San Diego State University
University of California
San Diego
San Diego, California
*Childhood Depression:
Treatment*
- Ian H. Gotlib, PhD**
Department of Psychology
Stanford University
Palo Alto, California
Functional Neuroimaging
- Julia A. Graber, PhD**
Department of Psychology
University of Florida
Gainesville, Florida
Childhood Depression
- John D. Guerry, MA**
Department of Psychology
University of North
Carolina at Chapel Hill
Chapel Hill, North
Carolina
Peer Relations
- J. Paul Hamilton, PhD**
Department of Psychology
Stanford University
Palo Alto, California
Functional Neuroimaging
- Nancy A. Hamilton, PhD**
Department of Psychology
University of Kansas
Lawrence, Kansas
Insomnia
- Benjamin L. Hankin, PhD**
Department of Psychology
University of Denver
*Attributional Theories of
Depression, Diatheses-
Stress Models of
Depression*
- Kate L. Harkness, PhD**
Department of Psychology
Queen's University
Kingston, Ontario,
Canada
Stressful Life Events
- Robert G. Harrington, PhD**
Department of
Psychology and Research
in Education
University of Kansas
Lawrence, Kansas
Externalizing Disorders
- Tirril Harris, MD**
Department of Health and
Population Research
King's College
London, England
Loss
- Lauren C. Haubert, PhD**
Department of Psychology
University of Calgary
Calgary, Alberta, Canada
Behavioral Observations
- Adele M. Hayes, PhD**
Department of Psychology
University of Delaware
Newark, Delaware
*Cognitive Behavioral
Therapy*
- Nicole Heilbron, MA**
Department of Psychology
University of North
Carolina at Chapel Hill
Chapel Hill, North
Carolina
Peer Relations
- Dirk Hermans, PhD**
University of Leuven
Leuven, Belgium
Self-Esteem
- Laura Hernangómez, PhD**
School of Psychology
Complutense University
Madrid, Spain
*Automatic and Controlled
Processing in Depression*
- Paula T. Hertel, PhD**
Department of Psychology
Trinity University
San Antonio, Texas
Memory Process
- Paul L. Hewitt, PhD**
Department of Psychology
University of British
Columbia
Vancouver, British
Columbia, Canada
Perfectionism
- Jacqueline Hoare, MBChB,
DMH, MRCPsych, FCPsych**
HIV/Neuropsychiatry
Program
Groote Schuur Hospital
University of Cape Town
Cape Town, South Africa
*Human Immunodeficiency
Virus*
- Jill M. Hooley, PhD**
Department of Psychology
Harvard University
Cambridge, Massachusetts
Expressed Emotion
- Jeffrey Horenstein, MA**
Department of Psychology
Carnegie Mellon
University
Pittsburgh, Pennsylvania
Social Support
- Steven S. Iardi, PhD**
Department of
Psychology
University of Kansas
Lawrence, Kansas
*Therapeutic Lifestyle
Change*

- Rick E. Ingram, PhD**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
Aaron Beck, Automatic Thoughts, Causality, Classification of Depression, Cognitive Distortion, Cognitive Reactivity, Cognitive Theories of Depression, Cognitive Vulnerability, Comorbidity, Depressive Realism, Depressogenesis, Gender Differences, High-Risk Research Paradigm, Internal Working Models, Interpersonal Model of Depression, Risk, Scar Hypothesis, Schemas, Self-Focused Attention, Symptoms of Depression, Vulnerability
- Lianna S. Ishihara-Paul, PhD**
 GlaxoSmithKline
 Pharmaceuticals
 WorldWide Epidemiology
 Harlow, Essex
 United Kingdom
Parkinson's Disease
- Robin B. Jarrett, PhD**
 Department of Psychiatry
 Southwestern Medical Center
 University of Texas
 Dallas, Texas
Psychosocial Functioning
- Amishi P. Jha, PhD**
 Department of Psychology
 University of Pennsylvania
 Pittsburgh, Pennsylvania
Attention
- Sheri L. Johnson, PhD**
 Department of Psychology
 University of Miami
 Coral Gables, Florida
Bipolar Disorders
- Thomas E. Joiner, Jr., PhD**
 Department of Psychology
 Florida State University
 Tallahassee, Florida
Suicide Theories
- John A. Joska, MBChB, MMed (Psych), FCPsych (SA)**
 HIV/Neuropsychiatry Program
 Groote Schuur Hospital
 University of Cape Town
 Cape Town, South Africa
Human Immunodeficiency Virus
- Cynthia Karlson, MA**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
Insomnia
- Leslie Karwoski, PhD**
 Evidence-Based Treatment Centers of Seattle
 Seattle, Washington
Eating Disorders, Exercise, and Depression
- Paul Keedwell, BSc, MB, ChB, MRCPsych**
 Department of Psychological Medicine
 Cardiff University
 Heath Park, Cardiff, Wales
 Honorary Lecturer
 Institute of Psychiatry
 Decrespigny Park
 London, England
Brain Circuitry
- Charles Kellner, MD**
 Department of Psychiatry
 UMDNJ-New Jersey Medical School
 Newark, New Jersey
Electroconvulsive Therapy
- Robert D. Kerns, PhD**
 Psychology Services
 Veterans Administration
 Connecticut Healthcare System
 Yale University
 New Haven, Connecticut
Chronic Pain
- Ronald C. Kessler, PhD**
 Department of Health Care Policy
 Harvard Medical School
 Boston, Massachusetts
Epidemiology
- Cara J. Kiff, MA**
 Department of Psychology
 University of Washington
 Washington
Temperament
- Hyoun K. Kim, PhD**
 Oregon Social Learning Center and Center for Research to Practice
 Eugene, Oregon
Maltreatment
- Cheryl A. King, PhD, ABPP**
 Departments of Psychiatry and Psychology
 University of Michigan
 Depression Center
 University of Michigan
 Ann Arbor, Michigan
Suicide in Youths
- Jason Klein, BA**
 Department of Psychiatry
 University of Kansas
 Lawrence, Kansas
Minor Depression
- John P. Kline, PhD**
 Department of Psychiatry
 University of South Alabama
 Mobile, Alabama
Psychophysiology of Depression
- Maria Kovacs, PhD**
 Department of Psychiatry
 University of Pittsburgh School of Medicine
 Pittsburgh, Pennsylvania
Children's Depression Inventory
- Robert F. Krueger, PhD**
 Department of Psychology
 University of Minnesota
 Twin Cities
 Minneapolis and St. Paul, Minnesota
Categorical and Dimensional Models of Depression
- Benji T. Kurian, MD, MPH**
 Department of Psychiatry
 Southwestern Medical Center

- University of Texas
Dallas, Texas
*Combined
Psychological and
Psychopharmacological
Treatment*
- Cecile D. Ladouceur, PhD**
Department of Psychiatry
University of Pittsburgh
Pittsburgh, Pennsylvania
*Brain Function in
Depressed Children and
Adolescents*
- Christine L. Larson, PhD**
Department of Psychology
University of Wisconsin–
Milwaukee
Milwaukee, Wisconsin
*Hemispheric
Lateralization*
- K. A. Lehman, PhD**
Department of Psychology
University of Kansas
Lawrence, Kansas
Omega-3 Fatty Acids
- Liliana J. Lengua, PhD**
Department of
Psychology
University of Washington
Seattle, Washington
Temperament
- Bernard Lerer, MD**
Biological Psychiatry
Laboratory
Department of Psychiatry
Hadassah-Hebrew
University Medical Center
Jerusalem, Israel
Thyroid Function
- Kenneth N. Levy, PhD**
Pennsylvania State
University
State College,
Pennsylvania
*Psychodynamic Model of
Depression*
- Peter M. Lewinsohn, PhD**
Oregon Research Institute
Eugene, Oregon
*Behavioral Models of
Depression*
- Alison R. Lewis, MA**
Northwestern University
Evanston, Illinois
Anxiety
- Cara C. Lewis, MS**
Department of Psychology
University of Oregon
Eugene, Oregon
Adolescent Depression
- Tzuri Lifschytz, PhD**
Biological Psychiatry
Laboratory
Department of Psychiatry
Hadassah-Hebrew
University Medical Center
Jerusalem, Israel
Thyroid Function
- Carissa A. Low, MA**
University of California at
Los Angeles
Los Angeles, California
Pittsburgh, Pennsylvania
Cytokines
- David D. Luxton, PhD**
Department of Psychology
University of Kansas
Lawrence, Kansas
*Insomnia, Posttraumatic
Stress Disorder*
- Sarah D. Lynne, MA**
Department of Psychology
University of Florida
Gainesville, Florida
Childhood Depression
- Danielle J. Maack, MA**
Department of Psychology
University of Wyoming
Laramie, Wyoming
Course of Depression
- Christopher R. Martell, PhD,
ABPP**
University of Washington
Seattle, Washington
*Behavioral Models of
Depression*
- Eric J. Mash, PhD**
Department of
Psychology
University of Calgary
Calgary, Alberta, Canada
Behavioral Observations
- James P. McCullough, Jr.,
PhD**
Department of Psychology
Virginia Commonwealth
University
Richmond, Virginia
*Cognitive Behavioral
Analysis System of
Psychotherapy*
- Danyale McCurdy, MA**
University of Kansas
Lawrence, Kansas
*Eating Disorders, Exercise
and Depression*
- Joseph B. McGlinchey, PhD**
Department of Psychiatry
and Human Behavior
Brown University School
of Medicine
Providence, Rhode Island
*Clinically Useful
Depression Outcome
Scale*
- Peter McGuffin, PhD**
Social Genetic and
Developmental Psychiatry
Centre
Institute of Psychiatry
Medical Research Council
King's College
London, England
Twin Studies
- Caroline McIsaac, MA**
Department of
Psychology
York University
Toronto, Ontario, Canada
Romantic Relationships
- John R. McQuaid, PhD**
Psychology Services
Veterans Administration
San Diego Healthcare
System
University of California
San Diego, California
Stress Assessment
- Tiffany Meites**
Department of Psychology
University of Kansas
Lawrence, Kansas
*Beck Depression
Inventory, Recurrence*

- Gerald I. Metalsky**
 Department of Psychology
 Lawrence University
 Appleton, Wisconsin
Hopelessness
- David J. Miklowitz**
 Department of Psychology
 University of Colorado at
 Boulder
 Boulder, Colorado
*Bipolar Disorder
 Treatment: Psychotherapy*
- Joshua D. Miller**
 University of Georgia
 Athens, Georgia
*Personality Disorders and
 Depression*
- Bethany H. Morris, MA**
 Department of Psychology
 University of South Florida
 Tampa, Florida
*Behavioral Activation
 System, Behavioral
 Inhibition System*
- Michelle Moulds, PhD**
 School of Psychology
 University of New South
 Wales
 Sydney, New South Wales,
 Australia
Differential Activation
- Ricardo F. Muñoz, PhD**
 Department of Psychiatry
 University of California at
 San Francisco
 San Francisco, California
Prevention
- Christy Nelson, MA**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
Insomnia
- Charles B. Nemeroff, MD,
 PhD**
 Department of Psychiatry
 and Behavioral Sciences
 Emory University School
 of Medicine
 Atlanta, Georgia
*Hypothalamic-Pituitary-
 Thyroid Axis*
- Arthur M. Nezu, PhD**
 Department of Psychology
 Drexel University
 Philadelphia, Pennsylvania
*Assessment of Depression,
 Problem-Solving Therapy*
- Christine Maguth Nezu, PhD**
 Department of Psychology
 Drexel University
 Philadelphia, Pennsylvania
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 New Haven, Connecticut
Rumination
- K. Daniel O'Leary, PhD**
 Department of Psychology
 Stony Brook University
 Stony Brook, New York
*Cognitive Behavioral
 Couples Therapy*
- Roisin O'Mara, MS**
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 Ann Arbor, Michigan
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- Gordon Parker, MD**
 Black Dog Institute
 Prince of Wales Hospital
 University of New South
 Wales
 Randwick, New South
 Wales, Australia
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- Alvaro Pascual-Leone, MD,
 PhD**
 Berenson-Allen Center
 for Noninvasive Brain
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 Beth Israel Deaconess
 Medical Center
 Harvard Medical School
 Boston, Massachusetts
*Transcranial Magnetic
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- Christi A. Patten, PhD**
 Department of Psychiatry
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- Mayo Clinic College of
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 Rochester, Minnesota
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- Suzanna L. Penningroth, PhD**
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 University of Wyoming
 Laramie, Wyoming
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- Carolyn M. Pepper, PhD**
 Department of Psychology
 University of Wyoming
 Laramie, Wyoming
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- Lukas Pezawas, MD**
 Division of Biological
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 Medical University of
 Vienna
 Vienna, Austria
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- Vicky Phares, PhD**
 Department of Psychology
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 Florida
 Tampa, Florida
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- Paul A. Pilkonis, PhD**
 Department of
 Psychiatry
 Department of Psychology
 University of Pittsburgh
 School of Medicine and
 Medical Center
 Pittsburgh, Pennsylvania
*Medical Conditions and
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- Bruce G. Pollock, MD**
 Department of Psychiatry
 University of Toronto
 Toronto, Ontario,
 Canada
Geriatric Depression
- Robert M. Post, MD**
 George Washington
 University School of
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 Washington, D.C.
 Penn State University
 College of Medicine
 Hershey, Pennsylvania

- Bipolar Collaborative
Network
Bethesda, Maryland
Kindling
- Mitchell J. Prinstein, PhD**
Department of Psychology
University of North
Carolina at Chapel Hill
Chapel Hill, North
Carolina
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- Rakesh Reddy, MD**
Department of Psychiatry
Vanderbilt University
School of Medicine
Nashville, Tennessee
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- Lynn Rehm, PhD**
Department of Psychology
University of Houston
Houston, Texas
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- Uzma S. Rehman, PhD**
Department of Psychology
University of Waterloo
Waterloo, Ontario,
Canada
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- Karen Reivich, PhD**
Department of Psychology
University of Pennsylvania
Philadelphia, Pennsylvania
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- John E. Roberts, PhD**
Department of Psychology
State University of New
York at Buffalo
Buffalo, New York
Self-Esteem
- Kathryn A. Roecklein, PhD**
Psychology Services
Jackson Veterans Affairs
Medical Center
University of Mississippi
Medical Center
Jackson, Mississippi
*Seasonal Affective
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- Kelly J. Rohan, PhD**
Department of Psychology
University of Vermont
Burlington, Vermont
*Seasonal Affective
Disorder*
- Paul Rohde, PhD**
Oregon Research Institute
Eugene, Oregon
*Adolescent Depression:
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- Paul D. Rokke, PhD**
Department of Psychology
North Dakota State
University
Fargo, North Dakota
Self-Efficacy
- Jonathan Rottenberg, PhD**
Department of Psychology
University of South
Florida
Tampa, Florida
*Behavioral Activation
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- M. David Rudd, PhD**
Department of Psychology
Texas Tech University
Lubbock, Texas
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- Natalie Sachs-Ericsson, PhD**
Department of Psychology
Florida State University
Tallahassee, Florida
*Postmenopausal
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- Brenda Sampat, PhD**
Department of Psychology
University of Kansas
Lawrence, Kansas
Reassurance-Seeking
- Monica Z. Scalco, MD**
Department of Psychiatry
University of Toronto
Baycrest Centre for
Geriatric Care
Toronto, Ontario, Canada
Geriatric Depression
- Christine D. Scher, PhD**
Department of Psychology
California State University
at Fullerton
Fullerton, California
Parenting
- Walter D. Scott, PhD**
Department of Psychology
University of Wyoming
Laramie, Wyoming
Self-Regulation
- Soraya Seedat, MBChB,
FCPsych (SA), PhD, MRC**
Unit on Anxiety and Stress
Disorders
Department of Psychiatry
University of Stellenbosch
Stellenbosch, South Africa
*Human Immunodeficiency
Virus*
- Zindel V. Segal, PhD, CPsych**
Center for Addiction and
Mental Health
University of Toronto
Toronto, Ontario, Canada
*Mindfulness-Based
Cognitive Therapy*
- Emre Selcuk, MA**
Department of Psychology
University of Kansas
Lawrence, Kansas
Attachment
- Brian A. Sharpless, PhD**
Center for Psychotherapy
Research
Department of Psychiatry
University of
Pennsylvania School of
Medicine
Philadelphia, Pennsylvania
Psychodynamic Therapy
- Lisa Sheeber, PhD**
Oregon Research Institute
Eugene, Oregon
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- Richard C. Shelton, MD**
Department of Psychiatry
Vanderbilt University
School of Medicine
Nashville, Tennessee
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- Greg Siegle, PhD**
 University of Pittsburgh
 School of Medicine
 Western Psychiatric
 Institute and Clinic
 Pittsburgh, Pennsylvania
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 and Depressive
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- Anne D. Simons, PhD**
 Department of Psychology
 University of Oregon
 Eugene, Oregon
Adolescent Depression
- Lisa M. Sontag, MS**
 Department of Psychology
 University of Florida
 Gainesville, Florida
Childhood Depression
- Susan C. South, PhD**
 Department of Psychology
 University of Minnesota,
 Minneapolis, Minnesota
*Categorical and
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- Kartik K. Sreenivasan, PhD**
 Department of Psychology
 University of Pennsylvania
 Pittsburgh, Pennsylvania
Attention
- Nicole K. Starace, MA**
 University of Hawai'i at
 Manoa
 Manoa, Hawai'i
Internalizing Disorders
- Dana Steidtmann, PhD**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
*Attributional Style,
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- Dan J. Stein, MD, PhD**
 Department of Psychiatry
 and Mental Health
 University of Cape Town
 Cape Town, South Africa
*Human Immunodeficiency
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- Laurence D. Steinberg, PhD**
 Department of Psychology
 Temple University
 Philadelphia, Pennsylvania
*Continuity of Depression
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- Cinnamon Stetler, PhD**
 Department of Psychology
 Furman University
 Greenville, South Carolina
Immune System
- Natalie R. Stevens, MA**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
*Insomnia, Postpartum
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- Daniel M. Stout, PhD**
 Department of Psychology
 North Dakota State
 University
 Fargo, North Dakota
Self-Efficacy
- Timothy J. Strauman, PhD**
 Department of Psychology
 and Neuro Science
 Duke University
 Durham, North Carolina
*Neuroimaging and
 Psychosocial Treatments
 for Depression*
- Natalie N. Stroupe, MA**
 Department of Psychology
 University of Kansas
 Lawrence, Kansas
Circadian Rhythms
- William B. Swann, Jr.**
 Department of Psychology
 University of Texas at
 Austin
 Austin, Texas
Self-Verification
- Eva Szigethy, MD, PhD**
 Psychiatry and Pediatrics
 University of Pittsburgh
 School of Medicine
 Department of
 Gastroenterology
 Children's Hospital of
 Pittsburgh
 Pittsburgh, Pennsylvania
Cytokines
- June Price Tangney, PhD**
 Department of Psychology
 George Mason University
 Fairfax, Virginia
Guilt
- Susan R. Tate, MA**
 Psychology Review
 Veterans Administration
 San Diego Healthcare
 System
 University of California at
 San Diego
 San Diego, California
Stress Assessment
- Michael Terman, PhD**
 Professor, Department of
 Psychiatry
 Columbia University
 Medical Center
 New York, New York
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- Julian F. Thayer, PhD**
 Department of Psychology
 The Ohio State University
 Columbus Ohio
Heart Rate Variability
- Caitlin Thompson, PhD**
 Center for the Study and
 Prevention Suicide
 University of Rochester
 Rochester, New York
Suicide in the Elderly
- Leonardo Tondo, MD**
 University of Cagliari
 Cagliari, Italy
*Bipolar Disorder
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- Leandro Torres, PhD**
 Department of Psychiatry
 University of California at
 San Francisco
 San Francisco, California
Prevention
- Madhukar H. Trivedi, MD**
 Department of Psychiatry
 Southwestern Medical
 Center
 University of Texas
 Dallas, Texas

- Combined Psychological and Psychopharmacological Treatment*
- Jeanne Tsai, PhD**
Department of Psychology
Stanford University
Palo Alto, California
Culture and Depression
- Rudolf Uher, MD**
Medical Research Council
Social, Genetic, and
Developmental Psychiatry
Centre
Institute of Psychiatry
King's College
London, England
Genetics of Depression
- Amanda A. Uliaszek, MA**
Department of Psychology
Northwestern University
Evanston, Illinois
Anxiety
- Kimberly A. Van Orden, MA**
Department of
Psychology
Florida State University
Tallahassee, Florida
Suicide Theories
- Carmelo Vázquez, PhD**
School of Psychology
Complutense University
Madrid, Spain
*Automatic and
Controlled Processing
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- Bavanisha Vythilingum,
MBCChB, FCPsych**
Groote Schuur Hospital
University of Cape Town
Cape Town, South Africa
*Dermatology and
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- Karen Dineen Wagner, MD,
PhD**
Department of Psychiatry
and Behavioral Sciences
University of Texas
Medical Branch
Galveston, Texas
- Childhood Depression:
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- Jerome C. Wakefield, PhD,
DSW**
Department of Psychology
New York University
New York, New York
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- Rachel H. Wasserman, MA**
Department of Psychology
Pennsylvania State
University
State College,
Pennsylvania
*Psychodynamic Model of
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- David Watson, PhD**
Department of
Psychology
University of Iowa
Iowa City, Iowa
*Positive and Negative
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- V. Robin Weersing, PhD**
Department of
Psychology
San Diego State University
University of California at
San Diego
San Diego, California
*Childhood Depression:
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- Daniel R. Weinberger, MD**
Genes, Cognition, and
Psychosis Program
National Institutes of
Health
Bethesda, Maryland
Molecular Genetics
- Myrna Weissman, PhD**
College of Physicians and
Surgeons
Mailman School of Public
Health
Columbia University
New York State
Psychiatric Institute
New York, New York
*Interpersonal
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- Adrian Wells**
Academic Division of
Clinical Psychology
University of Manchester
Manchester, England
Metacognition
- Tony T. Wells, MA**
Department of
Psychology
University of Texas at
Austin
Austin, Texas
*Cognitive Vulnerability:
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- Thomas A. Widiger, PhD**
Department of
Psychology
University of Kentucky
Lexington, Kentucky
*Diagnostic and Statistical
Manual of Mental
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- Kay Wilhelm, AM, MB, BS,
MD, FRANZCP**
School of Psychiatry,
Faculty of Medicine
Black Dog Institute
University of New South
Wales
Randwick, New South
Wales, Australia
Psychiatry
St. Vincent's Hospital
Darlinghurst, New South
Wales, Australia
Serotonin
- J. Mark G. Williams, PhD**
Department of Psychiatry
University of Oxford
Warneford Hospital
Oxford, England
Suicidal Cognition
- Paul Willner, DSc, FBPsS**
Department of Psychology
Swansea University
Swansea, Wales
Dopamine
- LaRicka R. Wingate, PhD**
Department of Psychology
Oklahoma State University
Stillwater, Oklahoma
Stress Generation

Eddie J. Wright, MA

Department of Psychology
University of Kansas
Lawrence, Kansas
*Adjustment Disorder,
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Diane Young, PhD

Department of Psychiatry
and Human Behavior
Brown University School
of Medicine
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*The Clinically Useful
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Department of Psychiatry
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Providence, Rhode Island
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Richard E. Zinbarg, PhD

Department of Psychology
Northwestern University
Evanston, Illinois
Assessment of Depression

Sidney Zisook, MD

Department of Psychiatry
University of California at
San Diego
San Diego, California
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David C. Zuroff, PhD

Department of Psychology
McGill University
Montreal, Quebec,
Canada
*Anaclitic and Introjective
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PREFACE

Depression touches virtually all of us. Millions of people worldwide experience clinical depression and many millions more experience subclinical depressive states. But the effects of depression reach well beyond those who are afflicted; people who are depressed have families and, to one degree or another, depression also takes a toll on the mothers and fathers, brothers and sisters, and sons and daughters of the depressed.

To mental health professionals, there is no more central concern than depression. Indeed, it is the rare psychologist, psychiatrist, or social worker who does not assess, treat, or counsel patients with primary or secondary depression. Mental health professionals, however, do not just diagnose and treat depression; numerous investigators have made depression the main focus of their research. In fact, few research programs are untouched by consideration of depression, either in conjunction with efforts to understand other mental health issues (e.g., anxiety research, treatment efficacy research), or in efforts to understand physical health (e.g., cardiovascular disorders, chronic pain, addictions). Not surprisingly, students in all of these areas have a keen interest in understanding depression.

The intense interest in depression is warranted. Depression is currently considered the second-most disabling disorder in the world and is predicted to be the most disabling disorder within the next two decades. Depression impairs functioning in all areas of life, from marital functioning to more general interpersonal functioning, and from occupation functioning to academic functioning. Depression can also have deleterious effects on physical health, and in the most extreme form is associated with earlier mortality compared to those who are not depressed, either from suicide or from damaged health. To state that depression is an enormous public health problem is something of an understatement.

Over the last few decades, knowledge of depression has grown rapidly. Although a number of antecedents to this explosion in depression theory and research could be identified, several developments in various scientific disciplines are key. From psychiatry, the introduction and now widespread use of antidepressant medications increased not only attempts to understand the efficacy of medication, but has also led to better knowledge of the underlying neurochemistry of depression. Developments in biology and genetics gave rise to a new understanding of the pathophysiology and heredity of depression, while innovations by cognitive researchers have led to neuroimaging advances that can provide a picture of brain functioning in depression. Finally, the development of cognitive therapy of depression revolutionized the psychological study of depression.

The wealth of information now available on depression is encapsulated in this encyclopedia. Experts from around the globe have contributed on everything concerning depression, from adolescent depression to HIV to vulnerability. In summarizing the vast amount of information on depression, the goal of the *International Encyclopedia of Depression* is to be an

important resource for the public in general, and more specifically for clinicians, researchers, students, and patients and their families.

A number of people deserve acknowledgment and thanks. The encyclopedia would not have been possible without the help of an internationally esteemed editorial board composed of Chris Brewin, Ian Gotlib, Steve Ilardi, Robin Jarrett, Thomas Joiner, Scott Monroe, Susan Nolen-Hoeksema, Zindel Segal, Greg Siegle, Dan Stein, and Karen Rudolph. Their insights and suggestions went beyond superb. A special thanks also goes to Phil Laughlin at Springer Publishing, who initially approached me about the idea of doing this encyclopedia. Phil has been supportive and helpful at every step in the process. It has been a pleasure to work with him. Finally, I want to acknowledge the love and support of my family, Nancy Hamilton, and Suzanna Ingram, who graciously understood the long days and nights necessary to finish this encyclopedia. I am forever in their debt.

RICK E. INGRAM
LAWRENCE, KANSAS

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Aaron Beck

Aaron T. Beck is the founder of cognitive therapy, arguably the most widely practiced psychotherapy in the world. Many psychologists and psychiatrists argue that he is the most influential figure ever in the field of depression. John Rush, a well-known depression researcher and professor of psychiatry at the University of Texas Southwestern Medical School, is quoted in the *Philadelphia Inquirer* as saying, “His work changed the paradigm—how we do things, how we think about things. . . . Somebody like him comes along every 50, 100 years.”

Beck was born in 1921 in Rhode Island and received his undergraduate degree from Brown University and his MD from Yale University. He was trained as a neurologist but began to pursue a career in psychiatry, partly in response to the enormous mental health needs of veterans returning from World War II. Like most psychiatrists of the time, Beck pursued psychoanalysis as a way to treat patients. Dissatisfied by the lack of efficacy of traditional psychoanalysis, Beck began to consider alternative ways of conceptualizing psychological disorders, eventually recognizing the importance of the cognitive construction that individuals place on life events. This recognition led to the development of a cognitive theory of depression, which suggested that events were not responsible for the emotional turmoil that leads to depression as much as the way that individuals interpret these events.¹ This idea now reflects the core foundation for the

understanding of not just depression, but of other psychopathological states as well.

Beck’s 1967 book proposed the first modern cognitive theory of depression, and his 1987 book, coauthored with Rush, Shaw, and Emery, introduced cognitive therapy to a wide audience. Both books are considered landmarks, and the 1987 book remains the predominant manual today for the conduct of cognitive therapy.

Beck has published 18 books (and counting) and hundreds of journal articles. He founded *Cognitive Therapy and Research*, a thriving scientific journal, and the Beck Institute for Cognitive Therapy and Research. He is the recipient of numerous awards, including awards by the American Psychiatric Association and the American Psychological Association; he is the only psychiatrist ever to have received awards from both associations. In 2006 he was awarded the prestigious Albert Lasker Award for Clinical Medical Research, considered by many to be the “American Nobel Prize.” In 2007 he was under consideration for the Nobel Prize.

Beck is currently a professor emeritus of psychiatry at the University of Pennsylvania and continues to write on cognitive theory and therapy.

RICK E. INGRAM

See also

Automatic Thoughts
Cognitive Behavioral Therapy
Cognitive Theories of Depression

Note

1. Around the same time, Albert Ellis (1913–2007) also developed cognitive ideas about emotional distress and a system of therapy known as rational emotive therapy. Ellis's contributions are recognized as extremely important, but because these ideas were not specific to depression, and because most cognitive therapy practiced today is inspired by Beck's work, most depression researchers credit Beck with the worldwide prominence of both cognitive theories of depression and cognitive treatment of depression.

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Adjustment Disorder

Adjustment disorder is characterized by a psychological response to an identifiable stressor or stressors that result in clinically significant distress or functional impairment (American Psychiatric Association [APA], 2000). Though this diagnosis is common, its validity has been questioned and research is lacking. Adjustment disorder was initially conceptualized as a transient personality disorder in the first edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-I; APA, 1952)* and a transient situational disturbance in the second edition. With the publication of the *DSM-III (APA, 1980)* came the term *adjustment disorder*, which was refined in subsequent editions.

Clinical Features

Adjustment disorder is a residual diagnostic category for stress-related emotional and behavioral disturbances that do not meet criteria for more specific disorders (APA, 2000). The *DSM-IV-TR* provides six subtypes: depressed mood, mixed anxiety and depressed mood, and mixed disturbance of emotions and conduct are relevant to depressive presentations. The remaining subtypes include anxiety, disturbance of conduct, and unspecified (APA, 2000). There is a dearth of

research demonstrating the validity and clinical utility of these subtypes (Casey, 2001).

In contrast to posttraumatic stress disorder and acute stress disorder, the precipitating stressor in adjustment disorder can be of any magnitude (i.e., it does not have to be traumatic). Impaired occupational or academic performance and changes in social relationships are common manifestations of adjustment disorder (APA, 2000). Research suggests that individuals with the condition are younger and more likely to be impulsive or to have a personality disorder than those with certain other psychiatric illnesses (Jones, Yates, Williams, Zhou, & Hardman, 1999). While the disorder is associated with an increased risk of suicide behaviors, this risk appears to be substantially lower than that of other major psychiatric illnesses, such as major depressive disorder (Casey, 2001). Furthermore, suicidal ideation typically resolves rapidly following the dissolution of the stressful event (Casey, 2001). Adjustment disorder is further associated with substance misuse and somatic complaints (APA, 2000; Greenberg, Rosenfeld, & Ortega, 1995).

Adjustment disorders that develop into more specific psychiatric disorders are reclassified as such. For example, if the symptoms of an individual with adjustment disorder with depressed mood crystallize into a major depressive episode, then he or she would be diagnosed with the appropriate mood disorder (e.g., major depressive disorder). While depressive episodes often emerge from stressful life events, adjustment disorder is generally less severe than depressive illnesses (Casey, 2001). There is some evidence suggesting that adjustment disorder with depressed mood is a distinct condition from more specific depressive disorders (Jones et al., 1999). Considering the severity of adjustment presentations that warrant psychological or psychiatric attention (e.g., the presence of suicidal behaviors), however, this distinction may not be clinically useful (Casey, 2001). Furthermore, the lack of specific symptom profiles for adjustment disorder can make distinguishing it from normal stress reactions and problems of living difficult. The

individual's cultural context is useful when clarifying such presentations.

Prevalence

Adjustment disorder is quite common, particularly in primary care and general medical settings (Casey, 2001). As the disorder can complicate medical illnesses (APA, 2000), addressing such issues is important. In clinical settings, adult women are twice as likely to be diagnosed with the disorder as their male counterparts, while it is equally distributed among male and female children and adolescents (APA, 2000). Jones and colleagues (1999) found that female outpatients were more likely to be diagnosed with a major depressive disorder or dysthymic disorder than adjustment disorder with depressed mood or mixed anxiety and depressed mood (73% vs. 57%). Prevalence rates vary depending on the population examined and the assessment methods employed. Adjustment disorder has been diagnosed in 10% to 30% of outpatients in mental health settings and up to 12% in general hospital inpatients referred for a psychological consultation (APA, 2000). A study concerning psychiatric inpatients found that 7.1% of the adults and 34.4% of the adolescents had an adjustment disorder diagnosis (Greenberg et al., 1995). Prevalence rates for community samples of children, adolescents, and the elderly range from 2% to 8% (APA, 2000). Despite these rates, most large-scale epidemiological studies of psychiatric disorders have neglected adjustment disorder (Casey, 2001).

Course

The diagnostic criteria for adjustment disorder state that the disturbance must occur within 3 months of the onset of the stressor and not persist longer than 6 months following its conclusion (APA, 2000). The diagnostic label, however, may be maintained beyond 6 months if the stressor is chronic or has enduring consequences. Research

suggests a good prognosis for adjustment disorder. By definition, symptoms typically resolve with the passage of time, but severe symptoms (e.g., the individual is acutely suicidal) may require intervention. While the persistence of adjustment disorder or its development into more severe conditions may be more common in children and adolescents, this may be due to comorbid disorders or the possibility that the initial presentation represented a subclinical manifestation of the more severe condition (APA, 2000).

Andreasen and Hoenk (1982) observed individuals with adjustment disorder and found that 79% of the adults and 57% of the adolescents were well at 5-year follow-up, with 8% and 13%, respectively having an intervening problem. While most adults who remained ill developed major depressive disorder or alcoholism, adolescent illnesses included schizophrenia, schizoaffective disorder, major depression, bipolar disorder, antisocial personality disorder, and substance abuse. The chronicity of the disorder and the presence of behavioral symptoms were the strongest predictors of major pathology in the adolescent participants at follow-up (Andreasen & Hoenk, 1982). Another study found that both adult and adolescent inpatients had shorter initial hospitalizations but presented with more suicidal behaviors than comparison psychiatric inpatients (Greenberg et al., 1995). Although depressed mood and mixed disturbance of emotions and conduct were the most common subtypes applied at admission, they did not predict length of the stay or rehospitalization. Forty percent of the inpatients did not maintain a diagnosis of adjustment disorder at discharge (Greenberg et al., 1995).

EDDIE J. WRIGHT

See also

Classification of Depression
Diagnostic and Statistical Manual of
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Adolescent Depression

Adolescence is characterized by change. Indeed change during this stage of life is so ubiquitous, occurring across almost all possible domains from physical to interpersonal, that the word *adolescence* is almost synonymous with change. Many of these changes can be quite positive and usher in exciting opportunities for growth and development. Unfortunately, some of the changes in this developmental period can be quite negative and open the door for undesirable outcomes and life course. One such negative change is the dramatic increase in the probability of developing clinical depression, a psychological disorder characterized by low mood (or irritability) accompanied by a variable set of other symptoms (such as changes in appetite and sleep, loss of concentration, guilt, and suicidality) that persists for at least 2 weeks and impairs functioning. While estimates of *childhood* depression are quite low, with only 1% to 3% of children under the age of 12 experiencing an episode of depression, estimates of adolescent depression are quite high, with approximately 20% of teens experiencing an episode of major depression by age 18. These percentages are actually much higher when including adolescents who exhibit significant symptoms of depression that

fall short of meeting the duration, frequency, or severity criteria for a *DSM* diagnosis of depression.

The likelihood of experiencing depression in adolescence is hardly inconsequential. While there is no opportune time to experience depression, it may be particularly disadvantageous during adolescence, a time that calls for the negotiation of critical life role transitions related to educational attainment, entry into the workforce, family and peer relationships, and romantic partnership. Depression can arrest these processes in their tracks and initiate a negative trajectory from which it can be difficult to recover. Longitudinal studies suggest that adolescent depression is associated with a long-term course characterized by recurrent depressions and adverse outcomes across a number of domains including other Axis I disorders (especially substance use but also anxiety and eating disorders), poor academic performance, pregnancy, problematic peer and family relations, and suicide.

The surge in depression in adolescence is especially notable for girls and leads to the well-known gender difference in adult depression. Epidemiology studies have reported female:male lifetime risk ratios ranging from 1.7:1 to 2.4:1, and such ratios have been found not just in the United States but in numerous other countries as well. The emergence of this difference begins sometime between the ages of 11 and 13 and is relatively firmly established by 15 years of age. Therefore, any model of adolescent depression has to be able to explain the spike in rates of depression for boys and girls, and the much higher rate of depression in girls (Hyde, Marzulis, & Abramson, 2008).

Prevailing models of depression are cast within variants of a diathesis-stress framework, a perspective that allows for a number of different vulnerabilities (e.g., neurobiologic, genetic, cognitive, and environmental) to interact with discrete or chronic life stressors (including those that occur early in life, e.g., abuse, as well as those that occur later, e.g., school transitions). The appeal of diathesis-stress models lies in their ability

to unite two obvious classes of influence on psychopathology—individual vulnerability and environmental adversity—and their ability to explain why some, but not all, people become depressed in the face of the same environmental events or in the presence of the same vulnerability. The fundamental premise of a diathesis-stress model is that both the diathesis (also interchangeably referred to as vulnerability) and stress are necessary for the development of disorder; neither one alone is sufficient (Monroe & Simons, 1991). Contemporary models also recognize that vulnerabilities, life stress, and depression are not necessarily neatly independent. Rather, the nature of the interrelationships among these variables is likely reciprocal and dynamic over time. For example, early stress and adversity may result in subsequent vulnerability. Further, depressive symptoms themselves may generate negative life events (e.g., irritability may lead to family conflict) and/or may lead to the development of vulnerabilities (Hammen, 2005). In other words, stressors can lead to vulnerabilities and vulnerabilities can lead to stressors over time to produce depression.

We focus particularly on research that recognizes and includes the unique developmental features of adolescence (e.g., brain development, peer and family context) rather than simply applying a downward extension of adult models of depression. This reflects accumulating research that suggests there are important differences between adolescent and adult depression. Indeed, the prevailing diagnostic system recognizes that the symptom profile for depressed teens may be different than for depressed adults, by allowing irritability rather than sadness as the cardinal symptom. Adolescents with depression are also more likely than adults to have a comorbid disorder, especially anxiety, and are more likely to present with so-called atypical depression, characterized by increases, rather than decreases, in sleeping and eating, and by somatic symptoms. Interestingly, depressed adolescents are more likely to continue to be reactive to the social environment but are also more likely than depressed

adults to have suicidal ideation. In addition, adolescent depression is typically the first depression, whereas adult depression is often a recurrence of depression. This latter distinction is important to consider, as it has been observed that predictors of first onsets and later episodes often appear to be quite different. Finally, results from randomized clinical trials of different treatments for adolescent depression also indicate differences between adolescent and adult depression in terms of treatment response (Treatment for Adolescents With Depression Study Team, 2004).

To understand the etiology of adolescent depression, the following three questions are considered: (a) what are the developmental changes of adolescence that may confer vulnerability to depression; (b) what is the nature of the life stress that interacts with these vulnerabilities to produce depression; and (c) how might these stressors and vulnerabilities reciprocally influence one another? As we address these questions here, it is important to keep in mind not only the transactional nature of these relationships, but also the notion of equifinality, that is, the idea that many different diathesis-stress pathways may result in the same outcome (i.e., depression).

Puberty

The fact that the increase in depression coincides with the onset of puberty has led to the speculation that hormonal changes are involved. However, studies reveal only very weak associations between gonadal hormone changes and negative affect. Research has also examined the neuropeptide oxytocin, which is regulated by the female gonadal hormones, estrogen and progesterone, and consequently rises in adolescence. Oxytocin plays an important role in affiliative behavior and may explain the well-known increase in desire for interpersonal connection among adolescent girls. This desire for affiliation, however, may come to constitute a vulnerability for depression. For example, female teens may be sensitized to the disruption of relationship bonds, a class of negative events that has been shown to precede depression in

teens (Monroe, Rohde, Seeley, & Lewinsohn, 1999) and is probably the most common trigger for suicide in teens.

Others have examined pubertal timing and found early-maturing girls have significantly elevated rates of major depression compared to girls who mature in synchrony with their peers. Interestingly, pubertal timing does not seem to make a difference for boys in relation to depression. Pubertal status (i.e., stage of development within puberty) has also been examined as a risk factor for depression, particularly for girls, with findings that rates of depression rise after reaching Tanner Stage III. It is important to note, however, that while pubertal status has been shown to predict depression in Caucasian girls, it does not predict depression in African American or Hispanic girls (Hayward, Gotlib, Schraedley, & Litt, 1999). This suggests that one must look at other factors operating in an adolescent's life to understand the effects of puberty and its possible relationship to depression (e.g., meaning of, and evaluation of, puberty-linked changes; skills required to deal with sexual attention).

Brain Development

Puberty-related physical changes are readily visible. Other biologic changes in brain structure and function, while less observable, may be more important for understanding adolescent depression. It has been stated that "puberty starts in the brain" (Davey, Yucel, & Allen, 2008), when the hypothalamus begins to release gonadotrophin-releasing hormone and continues with important changes, particularly in the prefrontal cortex. These changes include the selective elimination of unused synapses and the increase in myelination, resulting in a decrease in gray matter and an increase in white matter, thereby making the brain overall more efficient. This maturation of the prefrontal cortex is reflected in significant changes in cognitive functioning—the ability to think in more logical and abstract terms, to reflect on one's own thoughts, to understand the concept of extended time, and to anticipate the future. Davey and colleagues (2008)

argue that these emerging cognitive skills can double as vulnerabilities for an adolescent, as they also enable rumination, the generation of causal explanations (attributional style) for negative events, and frustration when anticipated rewards and goals are not forthcoming.

Genetic Factors

The literature suggests that postpubertal depression has a significant heritable component, with novel genetic influences emerging during this stage of development. Of note, one of the strongest predictors of childhood or adolescent depression is having one parent with a diagnosis of depression (Beardslee, Versage, & Giastone, 1998). Familial aggregation of depression has been examined through both top-down and bottom-up methodologies. The former method has found that children with a depressed parent have a 5:1 odds ratio of risk relative to controls and are approximately three times more likely to experience depression within their lifetime (Kaminski & Garber, 2002). The latter method of analysis has found elevated rates of depression in first-degree relatives of depressed children. However, regardless of this notable familial component, approximately one-third to one-half of teens develop depression in the *absence* of any familial history of the disorder. Another line of research that directly involves genetics has focused on a specific polymorphism, the short allele of the serotonin transporter gene (5-HTTLP-R). Partially replicating work done by Caspi and colleagues (2003) with young adults, Eley and colleagues (2004) found that this gene was a marker for the development of depression for adolescent girls, but not boys, who had experienced a recent negative life event.

Cognitive Factors

Cognitive factors have received considerable attention as risk factors for depression. A long list of cognitive factors, including attributional or inferential style, perfectionism,

self-criticism, rumination, dysfunctional attitudes, and self-schema have all received at least partial support as conferring vulnerability to the development of depression (Abela & Hankin, 2008). There is also some suggestion of a gender difference in at least some of these cognitive factors (e.g., rumination), which might help to explain the higher rates of depression in girls. It is important to note that these cognitive factors most likely follow developmental trajectories and are likely to change in the weight they carry in the development of depression. In other words, the influence of cognitive factors on depression may change as individuals age. For example, it has been found that negative attributional style interacts with life events to predict depression in middle schoolers (sixth to eighth graders), but not in younger children (third to fifth graders). Although subject to consideration of research attention, cognitive factors as diatheses for depression have yet to be firmly established; this may be in part due to pooling different age groups, as well as to inconsistencies in the measurement of these variables.

Life Stress

All diathesis-stress models conceptualize stress as playing an important role in the development of depression. In adult samples overall, it has been shown that most episodes of major depression are preceded by negative life events. Interestingly, the converse is not true; the majority of people do *not* develop depression after a negative life event (Hammen, 2005). However, although stress is considered important in the development of adolescent depression, the research on adolescents in relation to life stress and depression has lagged far behind that of adults. There continues to be a wide range of definitions, conceptualizations, and assessment approaches to stress employed in research investigating this developmental period. Therefore, of the three major domains of relevance—vulnerabilities, stressors, and depression—the least is known about the role of stress.

For the purposes of this chapter, the definition offered by Grant, Compas, Thurm, McMahon, and Gibson (2004) is adopted: “*environmental* events or chronic conditions that objectively threaten the physical and/or psychological health or well being of individuals of a particular age in a particular society” (p. 449). This definition argues against the view of stress as being only what is *perceived* as distressing, taxing, or beyond one’s coping capabilities. Rather, perceptions or appraisals of events and responses to events are seen as moderators of the impact of stress and/or mediators of the relationship between stressors and depression. This view suggests that interviews that can provide relatively objective assessments of what stressors have occurred, when they occurred, and how threatening they were, given the autobiographical circumstances of the adolescents, are preferable to self-report checklists. A review of the different studies purporting to examine the role of stress in teen depression, however, reveals that 45% of the studies constructed their own measures of stress, and fewer than 2% employed context-based interviews (Grant et al., 2004). While acknowledging the labor-intensive nature of administering and rating these interviews, lack of widespread use has likely prevented the acquisition of new knowledge about this important construct.

Environmental events may be especially potent in adolescence, given the pervasive social changes and role transitions that transpire during this time period. Family dynamics change as teens strive for more autonomy and the importance of peer groups becomes paramount. Not surprisingly, much attention has been given to interpersonal relationships and events such as family conflicts, peer rejection, bullying, relational aggression, and relationship breakups. In addition to specific events, the *themes* of events have been considered and have been shown to be important. In particular, the theme of loss, be it the loss of a person through death or the loss of a so-called cherished ideal (e.g., ideal of parental unity and intact family lost when parents divorce) or goal (e.g., school failure)

appears to be especially salient. The number and severity of events, as well as whether the stressor is seen as dependent on the individual's own actions (e.g., a girl is dumped by her peer group for betraying confidences) or independent of the individual's behavior (e.g., a girl's grandmother dies from cancer), have been shown to be associated with increased risk for depression. In general, greater and more severe events, especially dependent events, carry greater risk for subsequent depression.

Events occurring early in life, such as child abuse, maltreatment, and neglect, deserve special mention. Brown, Cohen, Johnson, and Smailes (1999) discovered a three- to fourfold increase in the likelihood of developing adolescent depression in teens who had experienced child abuse. In particular, sexual abuse carries with it the most significant risk of depression and suicide, independent of the contextual risks that the literature has identified to accompany abuse (e.g., low parental involvement, low parental warmth, low family income). The increased risk for depression is especially notable in females: in a mixed adolescent and young adult sample, the odds ratio for depression in females who had experienced childhood sexual abuse was 3:8 (Molnar, Bukar, & Kessler, 2001). In addition to increasing risk for later depression, early adversity also appears to increase risk for later adverse events and stressors, especially interpersonal difficulties. Early adversity, too, may result in greater psychobiological sensitivity to later stressors. The pathways by which early adversity translates into stress sensitization, subsequent stressors, and depression are unknown at this time but are the focus of considerable current research. Our understanding of the pathways to depression in adolescence is still incomplete. However, the lines of research highlighted in this chapter are quite promising. A diathesis-stress perspective offers a potential fruitful framework to view the dynamic interrelationships among genetics, puberty, brain changes, cognitive factors, and stressors.

See also

Adolescent Depression: Treatment
Brain Function in Depressed Children and Adolescents
Childhood Depression
Childhood Depression: Family Context
Childhood Depression: Treatment
Childhood Depression: Treatment With Pharmacotherapy
Children's Depression Inventory
Family and Parent-Child Therapy
Family Transmission of Depression
Fathers and Depression
Mothers and Depression

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Adolescent Depression: Treatment

Given the high prevalence, negative sequelae, and significant public health burden of adolescent depression, a number of interventions designed to treat this condition have been developed and evaluated in randomized controlled trials. Current treatments for depressed adolescents include various psychosocial interventions, pharmacotherapy, and combination treatments (medication plus psychotherapy).

Psychosocial Treatments

Several randomized controlled studies have examined the efficacy of a variety of individual and group-based psychosocial interventions for depressed adolescents. Based on the available research, certain forms of psychotherapy appear to be an appropriate initial treatment recommendation for adolescent depression.

Cognitive behavioral therapy (CBT) focuses on the role of one's thoughts and actions in becoming and remaining depressed. CBT has been evaluated in the largest number of trials to date and has been shown to be superior to wait-list control, and generally

more efficacious than alternative psychosocial treatments. Brent and colleagues (1997) contrasted individual CBT, systemic behavior family therapy, and individual nondirective supportive therapy for adolescent depression. Over the course of 12 to 16 weeks of acute treatment, response rates were significantly higher for CBT (60%) compared to either active comparison condition (39% for supportive therapy and 38% for family therapy), although treatment differences faded during the 2-year follow-up period. In the United Kingdom, an initial study of brief CBT (averaging six sessions) did not demonstrate superiority to an active comparison (nonfocused intervention), although a subsequent modification of brief CBT was superior to a different active comparison (relaxation training).

Peter Lewinsohn and colleagues found that the group-administered Adolescent Coping With Depression Course (CWD-A), a 7-week, twice-weekly intervention, was superior to a wait-list control condition. Clarke, Rohde, Lewinsohn, Hops, and Seeley (1999) conducted a larger replication of this study ($N = 96$) with treatment extended to 8 weeks and found similar results. In both trials, recovery rates for two forms of the CWD-A (one with just adolescents and the second consisting of an identical group for adolescents with a separate group for parents) were superior to wait-list, with nonsignificant differences between active treatments (e.g., 67% of treated adolescents no longer met criteria at posttreatment vs. 48% in wait-list in the second trial). More recent research with the CWD-A has included one of the first randomized controlled trials of a psychosocial intervention for depressed adolescents with current comorbidity (conduct disorder in that study) and the incremental benefit of augmenting standard care in a health maintenance organization (HMO) with the CWD-A. Although this group CBT resulted in greater reductions in depression measures compared to an active control condition, it did not confer a significant added benefit over usual HMO care.

These studies show that, for the treatment of depressed adolescents, CBT was superior

to passive control or comparison conditions, and superior acutely to some but not all active comparison conditions. Follow-up assessments indicate that the relative superiority of CBT compared to other active interventions was attributable to more rapid effects, as other treatments “caught up” with CBT during the follow-up period. Although initial meta-analyses reported large effect sizes for CBT across studies (e.g., 0.97–1.27), the most recent meta-analysis (Weisz, McCarty, & Valeri, 2006) was far more sobering. Although CBT for depression continued to show a significant benefit for young people, the effect sizes were significantly smaller than those reported in earlier studies (mean $ES = .34$), and CBT was not significantly more effective than other psychotherapeutic approaches.

Current research in CBT includes applications of the recent reconceptualization of behavioral activation (BA) for the treatment of depressed adolescents. This approach focuses on the specific role of avoidance in depression, relying heavily on an examination of the consequences of depressotypic behaviors, particularly those that serve to avoid imminent distress at the cost of blocking access to future positive reinforcement.

Another psychosocial intervention, interpersonal psychotherapy for adolescents (IPT-A), which was developed by Laura Mufson and colleagues, is based on the theory that interpersonal conflicts or transitions maintain depression. IPT-A has been evaluated in an initial pilot study looking at pre-postchange, a randomized controlled trial comparing it to clinical monitoring, and an effectiveness study comparing IPT-A to treatment as usual (TAU) in school-based mental health clinics (Mufson et al., 2004), finding that depressed adolescents receiving IPT-A had significantly greater symptom reduction and improvement in overall functioning, compared to those receiving TAU. Most recently, IPT-A has been modified to be delivered in a group format, but this version of the intervention has not yet been empirically validated. Another version of IPT for adolescents has been compared to CBT and

wait-list control in Puerto Rican adolescents with major depression. The two active interventions were found to be comparable and superior to wait-list control in reducing depressive symptoms.

In addition to these individual and group-based psychosocial interventions, Guy Diamond and colleagues have developed and begun evaluation of an attachment-based family therapy for adolescent depression. This family-based intervention appears promising and suggests that forms of family therapy warrant further attention for the treatment of depressed adolescents.

Pharmacotherapy

The rationale for pharmacological treatments of depression in adolescents is based on data suggesting the continuity of adolescent mood disorders with depressive disorders in adults and the responsiveness of depressive disorders in adults to a wide range of antidepressant medications. While earlier studies evaluating the efficacy of tricyclic antidepressants provided no support for their use as a first-line treatment, recent findings for the serotonin-specific reuptake inhibitors (SSRIs) are more promising. A variety of medications (paroxetine, sertraline, citalopram) have been found to have at least some degree of efficacy for depression, although only fluoxetine has been approved by the U.S. Food and Drug Administration as both safe and effective for minors with depression, based on data from a single-site NIMH-funded trial (Emslie et al., 1997) and a large, multisite, industry-sponsored trial. Research by Emslie and colleagues also suggests that continued fluoxetine treatment may be effective in preventing depression recurrence in child and adolescent patients who initially responded to acute medication treatment, although no other published controlled studies are available evaluating the safety and efficacy of antidepressants beyond short-term acute treatment. The Treatment for Adolescents With Depression Study (TADS), described below, recently provided very strong support for the effectiveness of

fluoxetine (Treatment for Adolescents With Depression Study Team [TADST], 2004).

Significant controversy has arisen regarding the safety of antidepressant therapy in adolescents, and close monitoring of young patients treated with antidepressant medications has been strongly suggested by both regulatory agencies and professional organizations. In conclusion, the current evidence-based treatment guidelines support the careful use of antidepressants in children and adolescents with significant mood disorders, although it is clear that a minority of patients suffer either nuisance or intolerable side effects that limit the utility or acceptability of pharmacotherapy.

Combination Therapies

The simultaneous emergence of (a) research suggesting that even our most empirically supported psychotherapies achieve smaller treatment effects than had previously been found, and (b) concerns regarding small, but real, increases in suicidality in adolescents treated with antidepressants has led to some confusion and concern regarding the recommended treatment for depressed adolescents. The combination of pharmacotherapy and psychotherapy may represent a choice that addresses the safety and efficacy concerns raised by using either intervention modality alone.

TADS was the first published study to look at combination of pharmacotherapy and psychotherapy as compared to monotherapies for adolescent depression, and this treatment package was found to be superior when considering both costs and benefits. The TADS project compared individual CBT, fluoxetine, combination CBT-fluoxetine, and a pill placebo with clinical management in 439 adolescents with major depression. TADS consisted of a 12-week acute treatment phase (after which, adolescents receiving pills only were unblinded), 6 weeks of graduated maintenance treatment, 18 weeks of maintenance treatment, and a one-year open follow-up. Results from the acute phase of therapy (TADST, 2004) used the Children's Depression Rating Scale-Revised as the primary

outcome and found a significant advantage for combination treatment compared to pill placebo ($p = .001$), which was not present for either fluoxetine ($p = .10$) or CBT ($p = .40$) monotherapies. Combined treatment also was superior to fluoxetine ($p = .02$) and CBT ($p = .01$); in addition, fluoxetine was superior to CBT ($p = .01$). Using a dichotomous measure of recovery (Clinical Global Impression Improvement score), rates of response were 71% for combination treatment, 61% for fluoxetine, 43% for CBT, and 35% for pill placebo. Regarding patient safety, the rates of harm-related adverse events were 12% for fluoxetine, 8% for combination therapy, 4% for CBT, and 5% for pill placebo.

Although the acute phase results in TADS were disappointing for CBT monotherapy, data from the Week 36 follow-up suggest that recovery rates for CBT monotherapy caught up to the recovery rates for fluoxetine and combination treatments, suggesting that CBT requires more time to be effective. Rates of response at Week 36 were 86% for combination therapy and 81% for both fluoxetine and CBT monotherapies. In summary, the TADS project found that treatment with either fluoxetine monotherapy or in combination with CBT accelerated the response for depression adolescents, and the addition of CBT enhanced the safety of pharmacotherapy. Balancing the benefits and risks of various interventions, the combination treatment appeared superior to either monotherapy as a treatment for adolescents with moderate to severe depression.

The other large trial of combination therapy, the Treatment of SSRI-Resistant Depression in Adolescents (TORDIA), was a randomized controlled trial of a clinical sample of 334 adolescents (ages 12–18) with a primary diagnosis of major depression who had not responded to an initial SSRI treatment. Participants were randomized to 12 weeks of (a) a second, different SSRI (paroxetine, citalopram, or fluoxetine); (b) a different SSRI plus individual CBT; (c) venlafaxine; or (d) venlafaxine plus CBT. At the end of 12 weeks, the combination of CBT plus either medication resulted in a higher response

rate than either medication monotherapy (55% vs. 40%, $p = .009$). Differences across the four treatment conditions on continuous measures of depression change, suicidal ideation, and harm-related events, however, were nonsignificant.

Other, smaller studies comparing combination treatments versus monotherapy approaches have not found significant differences. Greg Clarke and colleagues (1999) found that the addition of a brief CBT intervention (mean of five sessions) as an adjunct to SSRI treatment delivered in an HMO failed to improve outcomes for depressed adolescents, although this may have been because the group randomized to CBT received a lower average medication dose. Glenn Melvin and colleagues found no significant benefit for combination therapy compared to either CBT or pharmacotherapy monotherapy for the treatment of mild-to-moderate depression in adolescents. In that study, youths receiving CBT monotherapy had a superior response compared to those receiving pharmacotherapy monotherapy, although this may have been due to a relatively low dosage of sertraline. Ian Goodyer and colleagues recently reported results from a British study comparing treatment with a combination CBT and fluoxetine to fluoxetine only in a study of depressed adolescents. This study failed to replicate findings from the TADS and TORDIA trials that superior outcomes are obtained from combination treatment. One possible explanation for the nonsignificant results could be that the fluoxetine monotherapy protocol in the Goodyer study actually included a number of psychosocial ingredients (e.g., problem solving, focus on family and peer relationships), which suggests that it may have been more similar to combination treatment than to the medication monotherapy protocols evaluated in TADS or TORDIA.

Conclusions

Depression in adolescents is a serious public health concern, with the costs including significant lifetime morbidity and even mortality. Fortunately, significant progress over the past

20 years has been made in the development and evaluation of evidence-based interventions for this population of vulnerable youths. Research studies are now available that support the use of both psychosocial and pharmacological treatments, alone or in combination, for adolescents with depressive illness. This developing field, however, has not been without controversy. Even the best-supported psychotherapy interventions have not consistently fared well in comparison to other active interventions. Medication treatment studies have not been universally positive, and in some studies, SSRIs have been associated with elevated rates of serious adverse events. From a risk-benefit perspective, findings from the most well-powered trials currently argue for the adoption of a combination of SSRI medication and CBT as the best-practice treatment of adolescent depression. Future research, however, needs to address whether the positive effects of combination treatment are specific to CBT or could be obtained with other, more broadly available forms of psychotherapy, and the optimal method of disseminating best-practice interventions to depressed adolescents being treated in community settings.

PAUL ROHDE

See also

- Adolescent Depression
- Brain Function in Depressed Children and Adolescents
- Childhood Depression: Family Context
- Childhood Depression: Treatment
- Childhood Depression: Treatment With Pharmacotherapy
- Children's Depression Inventory
- Family and Parent-Child Therapy
- Family Transmission of Depression
- Fathers and Depression
- Mothers and Depression

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Affective Neuroscience

The term *emotion* most commonly denotes thoughts and feelings that combine to generate a specific experiential state. With the advent of modern functional neuroimaging, it is now possible to examine neural activity associated with the generation of emotional experience. The importance of such studies is, on the one hand, to uncover the functional neuroanatomy of emotions, that is, to associate emotional experiential states to specific brain substrates—mapping function to structure. In addition, affective neuroscience

allows an addressing of the fundamental representational structures on which emotional experience depends. This latter approach can provide neural constraints on the traditional theoretical models used to examine and understand emotional experience.

Structure of Emotions: Theory

For better or worse, affective neuroscience research in humans is dominated by competing models of the structure of emotions originating from behavioral investigations (Murphy, Nimmo-Smith, & Lawrence, 2003). Basic emotions models argue all humans are evolutionarily endowed with a limited number of discrete emotions that are specific adaptations for coping with particular situations (e.g., anger, fear, sadness). Each basic emotion is postulated to have a distinct facial expression and pattern of peripheral physiological response, as well as a dedicated central nervous system representation. The varieties of emotional experience can then be understood as the recruitment of brain regions supporting these distinct emotion programs, as well as their combination to promote complex emotional states. This conception of emotion space has some direct support from studies looking at stimulus triggers with emotional value, such as social signals of emotional states. For example, the amygdala has been associated with comprehension of fear expressions, and the right anterior insula-caudate disgust (Murphy et al., 2003).

Dimensional models, on the other hand, maintain that distinct emotions are combinations of lower-order underlying dimensions. These dimensions can be related to hedonics (positive or negative valence, either as bipolar or separate unipolar dimensions), activation or arousal (low vs. high, tense vs. energetic), action tendencies (appetitive vs. defensive, approach and withdrawal), or peripheral nervous system representation and regulation (sympathetic vs. parasympathetic). Distinct emotions that have unique verbal labels and experiential correlates would then be subserved by a smaller number of common

neural systems that represent the dimensionality of all emotional experience. This conception of emotion space has some direct support from studies looking at experiential states, such as finding that distinct regions of the orbitofrontal cortices are related to the subjective pleasantness and unpleasantness of sensory experience (Anderson et al., 2003).

Comparing and Contrasting Basic and Dimensional Models

The direct mapping of emotional functions to brain structures, although intuitively appealing, has many complexities. To illustrate some of these, I focus below on one of the brain structures most consistently associated with emotional functioning—the amygdala, a complex of multiple subnuclei that in non-human animals has been shown to play diverse roles. Numerous neuroimaging studies have shown amygdala activity varies with emotional experience in both healthy and affect-disordered populations, suggesting a central role in emotional phenomenology. However, the precise contributions of the amygdala remain a matter of ongoing study. It has been argued that the amygdala may play a specific role in fear and anxiety, supporting a modular basic emotions model for emotional experience. The amygdala has also been characterized as supporting a more dimensional role, related to overall negative affectivity, or more generally related to the intensity or arousing and/or activating nature of emotional experience (Anderson et al., 2003; Murphy et al., 2003). The difficulty in separating these models of amygdala function is highlighted by the fact that fearful or anxiety-evoking stimuli result in greater negative valence and experiential and sympathetic arousal, as well as many other variables, and as such amygdala recruitment can reflect any of these correlated experiential dimensions.

Studies in healthy samples that have matched emotional valence and intensity have shown the amygdala responds equally to carefully titrated pleasant and unpleasant

experiences and correlates with the intensity of experience, consistent with a more primitive arousal response (Anderson et al., 2003). However, this arousal dimension may be tuned through normative or pathological experience to represent enduring traits, whereby the amygdala is hyperresponsive to fear or negative affect, or even positive affect. Manipulations of present valence focus (to consider the positive or negative values of objects) have been shown to tune the amygdala to be responsive to more short term negatively or positively valenced states (Cunningham, Van Bavel, & Johnsen, 2008). This online attentional shaping of amygdala response may be interpreted as reflecting some intrinsic valence dimensional representation, rather than a valence tuning of a more primitive amygdala arousal orienting response.

In contrast with the amygdala, the orbitofrontal cortices (OFCs) appear to more clearly support some form of pleasant and unpleasant valence dimensions (Anderson et al., 2003; Rolls, 2000). The OFCs are highly plastic and represent the dynamics of affective value, being less a representation of the stimulus and more an internal representation of changing values to the perceiver (Rolls, 2000)—a prerequisite for ascribing a valenced phenomenological state to brain substrates. In particular, the more medial portions of the OFCs have been associated with pleasurable experiences, from basic pleasant smells and tastes, to abstract monetary reward, to looking at attractive individuals. Whereas, more lateral OFC regions have been associated with negative affective experience. It has been argued on the basis of nonhuman primate anatomical connectivity that the medial and lateral OFCs represent largely independent networks with greater intra- than interregional connectivity (Rolls, 2000). Corroborating functional imaging findings suggests that valence is not neurally coded along a single bipolar dimension but is subserved by separate valence axes in the medial and lateral OFCs (Anderson et al., 2003).

This medial-lateral pattern, however, has not always been replicated using more complex stimuli that may engage more sophisticated

cognitive and emotional appraisal processes. Indeed, meta-analyses collapsing across stimulus types do not clearly neurally demarcate such valence axes (Murphy et al., 2003). It may be that the brain does not support amodal valence dimensions, being sensory or stimulus specific, making the neural representation of emotional experience much higher in dimensionality and complexity than previously thought. In addition, emotional valence may better characterize linguistic conceptions of emotional states rather than their underlying neural bases. Emotions of similar negative valence such as anger and disgust may evoke different action tendencies (approach vs. withdrawal) and autonomic response (sympathetic vs. parasympathetic activation).

Neuropsychological Studies

Functional imaging research can demonstrate correlations between regional activity and emotional experience; however, these findings are often misinterpreted to reflect a causal relationship. Neural activity may represent any of diverse but correlated processes associated with emotions. Thus, neuropsychological studies remain critical to the study of affective neuroscience. For example, considering the mounting neuroimaging evidence associating the amygdala with emotional experience, there is little direct evidence of disordered emotional experience following amygdala damage. Patients with amygdala lesions provide reports of basic and valenced states of similar magnitude, frequency, and underlying covariance structure as healthy controls and, when asked to relive emotions, display remarkably intact emotional expressions including fear (see Anderson & Phelps, 2002). By contrast, more evidence supports the amygdala's role in altering information processing associated with emotional experience, including the emotional enhancement of mnemonic and attentional processes.

Self and Emotion

As the concept of “self” is central to human emotions, increased interest in the neural

foundations of selfhood have important implications for affective neuroscience. Altered cortical midline activation, particularly in the ventromedial prefrontal cortices (VMPFCs), has been associated with judgments of self-relevance as well as appreciation of emotional valence (Phan et al., 2004), ranging from simple sensory to more complex and abstract events. As this region receives connections from all exteroceptive and interoceptive modalities, it has been viewed as a polymodal convergence zone (Rolls, 2000), supporting the integration of external and internal stimuli with judgments about their value to the self. These VMPFC representations are in contrast to lateral prefrontal regions that may support a more self-detached and objective analysis of events. Another important aspect of the self is how the afferents from the body are represented in the brain. Structural and functional imaging research has linked right anterior insular volume and activity with increased sensitivity to visceral awareness (Critchley, Wiens, Rothstein, Ohman, & Dolan, 2004). In conjunction with the somatosensory cortex, the right mid and anterior insula enable a cortical representation of feedback regarding the exteroceptive somatic and interoceptive physiologic condition of the body. The future of affective neuroscience is to more fully and precisely understand how these bodily and conceptual bases of self interact, giving rise to complex neural states that support emotional experience.

ADAM K. ANDERSON

See also

- Amygdala
- Behavioral Activation System
- Behavioral Inhibition System
- Brain Circuitry
- Hemispheric Lateralization
- Hypothalamic-Pituitary-Adrenal Axis
- Hypothalamic-Pituitary-Thyroid Axis
- Positive Emotion Dysregulation

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Age of Onset of Depression

There are several questions about when depression first occurs whose answer requires different research designs; for several, the research has still to be done. Here we review what is known about four key questions about the onset of depression: when do depressive disorders first appear; at what age are people most likely to experience the onset of depression; what factors are associated with early onset of depression; and does early onset predict a worse course of illness?

When Do Depressive Disorders First Appear?

The idea that children could suffer from depressive disorders is fairly recent. Until the 1970s it was believed that children were cognitively unable to experience many of the core symptoms of depression, such as hopelessness or feelings of worthlessness. Beginning in the 1970s, empirical studies began to establish that children and adults report very similar depressive symptoms. Recently,

research on children as young as 2 years old has shown that mothers report similar syndromes even in the preschool years (Angold, Egger, & Carter, 2007). Epidemiological studies in the past 2 decades have shown that the population prevalence of depression is low before puberty but rises to adult levels in adolescent girls (Angold, Worthman, & Costello, 2003). From adolescence on, prevalence rates are in the same range as those seen in studies of adults.

But are child- and adult-onset depressions cases of the same disorder? Even when the same diagnostic criteria are used, might these symptoms reflect different neurobiological processes, just as symptoms such as fever or fatigue can be epiphenomena of many different underlying disorders? Some have argued there is too little evidence to conclude that childhood and adult depression are the same disorder, and much neurobiological evidence that they are different. One of the most important differences is response to tricyclic antidepressant medication. Unlike adults, children respond to tricyclics no better than to placebos (Geller, Reising, Leonard, Riddle, & Walsh, 1999). On the other hand, depression is common in the adult relatives of children who develop depression (Warner, Weissman, Mufson, & Wickramaratne, 1999), and childhood depression is a strong predictor of adult depression (Reinherz, Paradis, Giaconia, Stashwick, & Fitzmaurice, 2003). In short, it makes sense to treat childhood, adolescent, and adult depression as members of the same family until proven different.

At What Age Are People Most Likely to Experience the Onset of Depression?

Cases of depression may be diagnosable as early as 2 years of age, but the peak age at onset is somewhat later, especially for girls. Almost all epidemiological studies find that the prevalence rate of depression is considerably higher in adolescence than in childhood, which suggests that many adolescent cases are

first-onset cases. In studies of adult samples that ask about age at onset of first episode, in people with a lifetime history of depression, the median age tends to be around 30, with an interquartile range of about 25 years. This means that, assuming everyone lives to age 75, half of those who ever had one or more major depressive episodes would have had their first episode by age 30, and half of these would have had their first episode by age 18 (Kessler, Berglund, Demler, Jin, & Walters, 2005).

A problem with these estimates is that they are based on retrospective recall of the onset of depression by people of all ages, including elderly people, who may have forgotten how early their problems began. The recent National Comorbidity Survey–Replication estimates that around 20% of an adult sample aged 18 and older had suffered from depression at some time in their lives (Kessler et al., 2005). Several studies of samples aged younger than 20 report similar rates. For example, in one birth cohort followed to age 32, over 50% of women and 30% of men had experienced at least one episode of depression (Moffitt et al., 2007). In another prospective longitudinal study, 11% of young people had experienced at least one episode by age 21. Reports such as this suggest that the evidence of older people may overestimate the peak age at which depression begins.

What Factors Are Associated With Early Onset of Depression?

The factor most widely publicized as predicting early onset of depression is date of birth. There is a widespread belief that, at least in the second half of the 20th century, there has been a dramatic fall in the age of onset of depression, accompanied by an increase in prevalence. However, a review of more than 30 studies published since the 1970s shows no evidence for either increased prevalence or earlier onset (Costello, Erkanli, & Angold, 2006).

There is more reliable evidence that comorbidity with other psychiatric disorders

affects, and is affected by, the onset of depression. In the Great Smoky Mountains Study (Costello, Angold, & Sweeney, 1999), the mean age of onset of depression, in those who developed the full disorder by age 16, was 13.5 years (*SD* 2.1 years). The mean age at onset was a full year earlier (12.8, *SD* 2.4) in those who had a history of anxiety disorder than in those who did not (13.8, *SD* 1.8). It was also earlier in girls, but not boys, with oppositional disorder. Conversely, the mean age at onset of oppositional disorder and separation anxiety disorder were later in youths with a history of depression than in others with the disorders, while the onset of generalized anxiety disorder and attention deficit hyperactivity disorder were earlier in those with depression than in others with the disorders. In summary, the onset of depression may bring forward the emergence of some other psychiatric disorders.

Although gender is well known to increase the risk of onset of adolescent depression (Angold et al., 2003), the same study showed no difference in mean age at onset of depression by age 21. Family history of depression has been linked to earlier onset, however.

Does Early Onset Predict a Worse Course of Illness?

We lack the life-course longitudinal studies that would answer this question properly. There is evidence that, like adult-onset depression, early depression is likely to be recurrent, although the National Comorbidity Survey data suggest that, in both adolescents and adults, the excess of women with depression applies only to first episodes; thereafter the risk of recurrence is the same for both sexes (Kessler, 2003).

Conclusions

This brief survey of the literature on age of onset of depression concludes that (a) depression, as defined in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (American Psychiatric Association,

1994), can be diagnosed in children as young as 2; (b) there are many similarities, although some differences, when childhood- and adult-onset depressions are compared; (c) at least a quarter of people who experience depression during their lifetime had their first episode in childhood or adolescence; (d) the evidence on whether more people are having early-onset depression is equivocal; (e) the presence of other psychiatric disorders may increase the risk of early-onset depression; (f) early onset is more common in children from families with a history of depression; (g) childhood-onset depression is as likely as later-onset depression to be recurrent. Early-onset depression is a serious public health problem, not something that children will “grow out of.”

E. JANE COSTELLO

See also

Adolescent Depression
Childhood Depression
Course of Depression
Epidemiology

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Amygdala

Location and Structure

The amygdala (Latin for “almond”) is an oblong limbic brain region located in the medial temporal lobe of reptiles and mammals. The human amygdala is conventionally thought of as being bounded posteriorly by the alveus of the hippocampus, anteriorly by the horn of the lateral ventricle, inferiorly by white matter, and superiorly by the subarachnoid space. It is bounded laterally by periamygdaloid cortex adjacent to white matter and medially by periamygdaloid cortex adjacent to the subarachnoid space.

The amygdala represents a collection of nuclei with different functions and structural connectivity to different regions of the brain. Its original definition incorporated what is currently termed the basolateral complex of the amygdala, including the lateral, basal, and accessory basal nuclei. Subsequently, additional adjacent but structurally and functionally heterogeneous nuclei, including the central, medial, and cortical nuclei, have been included. Increasingly, a notion of the amygdala

as extending even more superiorly toward the rostral extent of the bed nucleus of the stria terminalis has been embraced, having often been described the “extended amygdala.”

Function and Connectivity

The amygdala, as a whole, is generally regarded as being important to recognizing emotional aspects of information and generating emotional reactions (for reviews, see Phelps, 2006; Zald, 2003). The different subnuclei of the amygdala appear to subserve different aspects of this overall functionality. The basolateral region is often considered the so-called input region of the amygdala, which, given incoming connections from sensory regions such as the thalamus, regions associated with memory retrieval such as the hippocampus, and higher associations in medial cortical regions, computes the salience, threat, or reward value of the stimulus. Outgoing connections to regions such as the orbitofrontal cortex, hippocampus, and striatum are hypothesized to influence subsequent cognitive consequences (e.g., memory formation) or behaviors (e.g., approach/avoidance) based on these computations. The basolateral amygdala appears to disinhibit neurons in the central nucleus via intra-amygdala intercalated cell masses. The central nucleus is often conceived of as the primary output region of the amygdala, involved in generating emotional reactions such as autonomic and cognitive aspects of fear and anxiety, emotion-relevant attentional allocation, and so forth, through connections to a variety of cortical and subcortical regions, including regions associated with autonomic regulation, such as the hypothalamus, nucleus ambiguous (which is involved in cardiac regulation), and facial motor nucleus. Thus, in anatomic studies, the amygdala is implicated as one of the most widely connected brain structures, having influences on, or being influenced by, nearly every functionally important region of the mammalian brain.

The amygdala has specifically been shown to react to low-level perceptual features associated with threatening or

environmentally salient information, including low-spatial-frequency stimuli, the whites of eyes, and masked threat-related stimuli presented too quickly for conscious awareness. It also responds to higher-level emotional stimuli such as emotional pictures, films, personally relevant words, and expected monetary loss. The amygdala appears to activate proportionally to the intensity and arousal-content of emotional stimuli and as a function of prestimulus sad-mood priming. Thus, the amygdala has been implicated in a wide variety of human behaviors involved in emotional information processing, including emotion recognition, generation, emotional memory, emotional learning, the experience of sadness, and rumination.

Inhibition of the amygdala by the cortex has been described as a mechanism for emotion regulation and has been observed through both animal studies and human neuroimaging. In particular, inhibitory connections from the ventromedial prefrontal cortex (specifically the peri- and subgenual regions of the cingulate cortex) and orbital prefrontal cortex have been hypothesized to serve this function (for reviews, see M. L. Phillips, Ladouceur, & Drevets, 2008).

Functional Role of the Amygdala in Depression

Tonic Activity

Given the centrality of the amygdala in emotional reactivity, a strong role for this structure in the pathology of depression has long been hypothesized and observed (for reviews, see Davidson, Jackson, & Kalin, 2000; W. C. Drevets, 1999; W. C. Drevets, Price, & Furey, 2008; Mayberg, 2003; M. L. Phillips, Drevets, Rauch, & Lane, 2003; Whalen, Shin, Somerville, McLean, & Kim, 2002). As the amygdala is involved in detection of emotional features of information, observed increased tonic amygdala activity in unipolar depression is hypothesized to maintain negative affect, increase detection

of emotional features in the environment, and create a predisposition toward affectively charged information processing. This activity is observed not only during waking but also in sleep. In support of the functional importance of tonic amygdala activity in depression, the severity of depression has been associated with tonic amygdala activity. Activity has been related to changes in the neurochemical milieu of the amygdala in depression (for review see W. C. Drevets, Price, & Furey, 2008); decreased serotonin binding and decreased mu-opioid binding potential during sad mood have been specifically implicated.

Reactivity

Increased amygdala reactivity is consistently observed in depression, to low-level stimuli such as faces, particularly those that are remembered, and to unattended emotional stimuli, suggesting that biases in amygdala reactivity are associated with early, automatic processing (for review consistent with this perspective, see Leppanen, 2006). Increased and sustained amygdala activity are often observed in depression in response to higher-level emotional stimuli such as negative words and thoughts, even during attempts at regulation of sad feelings. Increased and sustained amygdala reactivity have been associated with rumination and depressive severity. Increased activity of the extended amygdala during anticipation of aversive events in depressed individuals further attests to the potential role of this structure in the phenomenology of cognitive biases such as anticipation of negative outcomes. Amygdala reactivity in depression appears to be modulated by genes associated with serotonin function.

Functional Connectivity

Increasingly, examinations of amygdala function in depression acknowledge the importance of the greater network of brain function involved in the disorder. Thus,

data suggest that amygdala activity across the hemispheres is more tightly coupled in depression, but that functional connectivity assessed via covariation with prefrontal regulatory regions is decreased in depressed individuals, particularly during mood regulation, though these effects may differ as a function of genetics and life stress.

Heterogeneity

Other research suggests considerable functional heterogeneity in the role of the amygdala in depression. For example, initial studies suggest that amygdala reactivity to emotional stimuli appears to be decreased in depressed postpartum women and children, and decreased in vulnerable youths by the presence of a task compared to passive viewing. As depressed children display increased amygdala activity for emotional information they remember, a general pattern of avoidance in childhood depression may be present. Thus, further investigation into the functional heterogeneity and causes of variation in amygdala function in depression may be necessary. Additionally similar patterns of increased amygdala activity and reactivity have been observed in a host of other related disorders such as social anxiety disorder, suggesting a potential lack of specificity for observed effects, though the presence of comorbid depression appears to be associated with decreased amygdala activity in posttraumatic stress disorder.

Structure

The functional importance of the amygdala in depression is echoed by data suggesting that amygdala shape and volume are also disrupted. The direction of effects in these studies is mixed, though, with studies showing increased, decreased, or no differences in amygdala volume in depression. Heterogeneity in the ways amygdala volumes are quantified (e.g., what boundaries are used for tracing the amygdala on an MRI) has been

shown to moderate detection of group differences and may thus contribute to the lack of consistent results. Limited data suggest that decreased amygdala volumes are related to increased activity in response to emotional information but decreased biases toward remembering negative information in depression.

Involvement in Treatment

Significant evidence suggests a role for the amygdala in recovery from depression in evidence-based treatments such as cognitive therapy and antidepressant medications (for review, see DeRubeis, Siegle, & Hollon, 2008). Recovery is associated with reductions of tonic amygdala activity, increased functional coupling with prefrontal regions, and decreased reactivity to low-level stimuli, suggesting that amygdala function may be an index of the depressive state. Yet, other data suggest that upon induction of a depressive state, amygdala activity in formally depressed individuals serves roles seen in depression, such as increasing a predisposition to remember negative information, consistent with a diathesis-stress interpretation of the role of the amygdala. Consistent with this idea, depletion of serotonin in vulnerable individuals is also associated with increased amygdala activity in response to emotional stimuli. Furthermore, targeted interventions such as stimulation of subgenual cingulate regions with strong connectivity to the amygdala appear to better target depressive symptomatology than dorsal cingulate regions without this connectivity. Yet, the causal role of the amygdala in either the phenomenology or treatment of depression may be questioned in a report of a patient who had depressed mood and responded to antidepressant medications despite amygdala lesions.

The extent to which structural volume abnormalities resolve with treatment for depression is unclear and may be dependent on the modality. For example, an early study suggested that volumes normalize

with electroconvulsive therapy, but other data have not found volumetric changes in recovered individuals.

Increased amygdala activity in response to cognitive and emotional stimuli appears to positively predict recovery in depressed individuals for treatments that target emotional functioning such as cognitive therapy, as well as interventions known to affect limbic function partial sleep deprivation, antidepressant medications, and unselected treatments.

GREG SIEGLE

See also

Affective Neuroscience
Behavioral Activation System
Behavioral Inhibition System
Brain Circuitry
Functional Neuroimaging
Hemispheric Lateralization
Hippocampus
Hypothalamic-Pituitary-Adrenal Axis
Hypothalamic-Pituitary-Thyroid Axis

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Anaclitic and Introjective Depression

Several investigators have noted marked limitations in studying depression from the categorical symptom-based approach articulated in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; 1994)* and have instead focused on differences in the experiences that lead to depression—a more person-centered rather than a disease- or symptom-centered approach.

Symptoms of depression are quite heterogeneous and include psychological symptoms (e.g., mood change and pervasive loss of energy and interest) and somatic or neurovegetative symptoms (e.g., loss of sleep and appetite). This heterogeneity of symptoms has made it difficult to differentiate meaningful subtypes of depression in both clinical and nonclinical samples. Several research teams (Arieti & Bemporad, 1978; Beck, 1983; Blatt, 1974, 2004) over the past three decades, however, have differentiated subtypes of depression based not on symptoms but on differences in personality style and the life experiences that can lead to depression. These research teams have

differentiated two major types of depression: a depression initiated by disruptions of interpersonal relationships such as interpersonal loss and feelings of abandonment and loneliness (an anaclitic or dependent [e.g., Blatt, 1974], sociotropic [Beck, 1983], or dominant other [e.g., Arieti & Bemporad, 1978] depression) from a depression initiated by disruptions of self-esteem, achievement, identity (an introjective or self-critical [e.g., Blatt, 1974; autonomous [Beck, 1983], or dominant goal [e.g., Arieti & Bemporad, 1978] depression).

Extensive research with both clinical and nonclinical samples (see summaries in Blatt, 2004) demonstrates that these two types of depression—a depression focused on interpersonal issues and one on issues of self-esteem and self-worth—derive from very different early life experiences, are precipitated by different types of current life events or stressors, have different clinical expression, and are responsive to different types of therapeutic intervention. An anaclitic-dependent, sociotropic, or a dominant-other type of depression that derives primarily from neglectful and depriving parenting is precipitated primarily by interpersonal stressors (e.g., experiences of loss and abandonment); is expressed in lethargy, feelings of helplessness and loneliness, somatic concerns and can involve suicidal gestures; and is responsive primarily to supportive types of therapeutic intervention. An introjective-self-critical, autonomous or a dominant-goal type of depression, in contrast, that derives primarily from harsh, punitive, judgmental parenting; is precipitated primarily by experiences of failure and criticism; is expressed in feelings of hopelessness, failure, intense self-criticism, and can involve serious suicidal risk; and is responsive primarily to more intensive, long-term, interpretive types of therapeutic interventions.

Evaluation of therapeutic response in long-term intensive treatment, in both inpatient and outpatient settings, indicates that anaclitic and introjective patients are differentially responsive to supportive-expressive

psychotherapy and more intensive psychoanalysis (Blatt & Shahar, 2004) and change in different ways—in ways consistent with their basic personality organization (Blatt & Ford, 1994). While introjective patients are more responsive than anaclitic patients to intensive long-term treatment, they are significantly less responsive to brief manual-directed psychotherapy and antidepressive medication (e.g., Blatt & Zuroff, 2005).

Thus, a person-centered developmental approach to depression has been valuable in establishing a meaningful clinical distinction that has led to fuller understanding of some of the etiological factors that contribute to depression, as well as some of the processes involved in effective therapeutic intervention.

Extensive research has also been devoted to describing individual differences in vulnerability to the two forms of depression. Dependency is a personality dimension that confers vulnerability to anaclitic depression; self-criticism is a stable personality dimension that confers vulnerability to introjective depression (Blatt, 2004). The most widely used measure of dependency and self-criticism is the Depressive Experiences Questionnaire (Blatt, D'Afflitti, & Quinlan, 1979), which has been translated into over 15 languages spoken in North America, South America, Europe, the Middle East, and Asia.

Dependent and self-critical people differ in terms of their fundamental motivations, cognitions about themselves and others, patterns of interpersonal relationships, and affective experiences (Blatt, 2004). Individuals with high levels of dependency have a greater number of interpersonal goals involving affiliation and intimacy and fewer achievement and individualistic goals (Mongrain & Zuroff, 1995); dependent individuals' needs for nurturance and affection come at the expense of their own individuation. Individuals with high levels of self-criticism, in contrast, endorse a greater number of achievement goals, more self-presentation strivings, and fewer interpersonal strivings (Mongrain & Zuroff, 1995), indicating a relative disinterest in affiliative and warm exchanges with others.

Highly dependent individuals perceive themselves as weak and vulnerable, and needing the support and guidance of others. At the same time, they lack confidence in the emotional availability of others, and fear abandonment and the loss of supportive relationships. Self-critical individuals report negative representations of self and others, including parents, friends, and romantic partners. They are afraid of being rejected, are not comfortable with closeness, and do not feel they can rely on their partners (Zuroff & Fitzpatrick, 1995). Their negative models of self and others leave them distrustful, guarded, and avoidant of intimacy, seeking to protect themselves against anticipated rejection and criticism.

High dependency is associated with a mixed pattern of positive and negative interpersonal experiences. Highly dependent individuals describe themselves as experiencing more love and intimacy and having greater social support (Priel & Shahar, 2000), but not as more satisfied with their interactions or relationships (Zuroff, Stotland, Sweetman, Craig, & Koestner, 1995). The interpersonal experiences associated with self-criticism are consistently negative, with impairments reported in peer relationships, romantic relationships, child-rearing, and therapeutic relationships. Associated with these impairments are low levels of social support and high levels of stress (Dunkley, Zuroff, & Blankstein, 2003).

The characteristic emotional tones of individuals with high scores on dependency and self-criticism also differ (Zuroff et al., 1995). Self-criticism is associated with consistently high levels of negative emotions and consistently low levels of positive emotions. Dependency is also associated with high levels of negative emotions but is unrelated to levels of positive emotions, suggesting that positive emotions may fluctuate from low to high in those with high levels of dependency. In summary, highly dependent individuals value and seek relatedness, fear its loss, and, perhaps because of their fluctuating relationships, experience considerable variations in their emotional states. Self-critical individuals

seek achievement and perfection rather than relatedness, fear being hurt by others, and, perhaps because of their impaired relatedness with others, experience little positive affect and much negative affect.

These findings about the nature of two types of depressive experiences and the characteristics of dependent and self-critical individuals suggest that other forms of psychopathology beyond depression, especially the personality disorders, may be more fully understood from a more person-centered approach in which psychopathology is understood as exaggerated distorted preoccupation, at different developmental levels, with issues of interpersonal relatedness or issues of self-definition. Thus, a person-centered approach would facilitate further understanding of the complex links between depression and personality disorders.

SIDNEY J. BLATT AND DAVID C. ZUROFF

See also

Attachment
Cognitive Theories of Depression
Cognitive Vulnerability
Internal Working Models
Psychodynamic Model of Depression
Psychodynamic Therapy

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Anger

The frequency with which anger is experienced in adult depression may not be adequately emphasized in current diagnostic criteria. In the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*; American Psychiatric Association [APA], (1994), sadness or “depressed mood” is the only affective symptom explicitly included in the diagnostic criteria for major depressive episode in adults. The subsequent revised edition (*DSM-IV-TR*; APA, 2000) acknowledges that irritable mood may contribute to a diagnosis of depression in children and appear alongside atypical features in adults. However, a substantial number of individuals presenting with major depressive disorder present with clinically relevant levels of anger.

Anger and the Nosology of Depression

Depressive syndromes characterized by anger or hostility are commonly highlighted in subtyping approaches to the classification of depression. Sometime ago, for instance, Paykel (1971) conducted a cluster analysis of both inpatients and outpatients and found

four subgroupings of depression: psychotic, anxious, hostile, and young mildly depressed individuals with personality pathology. Similarly, a cluster analysis by Overall, Hollister, Johnson, and Pennington (1965) yielded three subcategories of major depressive disorder, labeled the hostile, anxious, and retarded depression types.

In 1990, Fava, Anderson, and Rosenbaum (1990) first introduced the concept of “anger attacks,” in patients diagnosed with major depressive disorder. These anger attacks are similar to panic attacks in their temporal course and autonomic activation, but without the intense fear and anxiety that accompanies panic. These attacks are not viewed as ego-syntonic, rather, they are considered uncharacteristic by the person having the attacks. Several studies have been undertaken since 1990 to explore major depressive disorder with anger attacks in terms of response to antidepressant medications, physiological correlates, and clinical presentation.

Some researchers have suggested that anger in the context of unipolar major depressive disorder signifies a bipolar spectrum mood disturbance (e.g., Benazzi & Akiskal, 2005). In patients diagnosed with major depressive disorder, anger is related to markers of bipolar disorders, including younger age of onset, bipolar family history, higher rates of atypical depressive features, and hypomanic symptoms. There is also evidence, however, that depression with anger may be more closely aligned with unipolar depression. For example, antidepressant medications have been found to reduce anger and hostility, and individuals with major depressive disorder plus anger seem no more likely than their counterparts without anger attacks to experience a switch to bipolar I or II following treatment with antidepressant medications.

Demographic, Personality, Cognitive, and Biological Correlates

Prevalence estimates of clinically relevant levels of anger and hostility in samples of

depressed individuals have ranged from nearly one-fifth to over one-third. Anger and hostility appear to be clinically relevant in a large portion of both inpatients and outpatients with major depressive disorder. Patients with major depressive disorder plus anger do not seem to differ in age compared to those with major depressive disorder but without anger attacks. Evidence suggests, however, that those experiencing significant irritability or hostility in the context of depressive episodes tend to be younger than those not experiencing irritability or hostility.

Findings regarding gender differences in hostile, angry, and irritable expressions of depression have been mixed. Where gender differences on a particular anger-related construct are found in some research, other research does not find these differences. Despite mixed findings regarding the relationship of gender to anger in depression, some have suggested that males tend to exhibit a unique depressive syndrome marked by irritability, anger, and low impulse control.

Little research has examined the personality and cognitive correlates of anger in the context of major depressive disorder. The presence of anger attacks in major depressive disorder may be indicative of Cluster B and/or C Axis II pathology, and hostility is associated with low agreeableness and conscientiousness. With regards to cognitive correlates, depression has been associated more strongly with cognitive as opposed to behavioral expressions of hostility. For example, depression with hostility may be associated with external and specific attributions for negative events.

Whereas research exploring the personality and cognitive correlates of anger in depression is sparse, studies examining the biological correlates have been many. For instance, research suggests that major depressive disorder plus anger is associated with hypofunctionality of the ventromedial prefrontal cortex and insufficient inhibition of amygdalar activity in anger inducing situations. In addition, dysregulated neurotransmitter functioning is associated with anger in depression. Diminished serotonergic activity

has been linked to stable hostility and anger attacks, elevated dopamine neurotransmission has been associated with anger attacks, and low levels of GABA have been linked to overt hostility in depression. Finally, a genetic contribution to anger in depression is likely. Research has suggested links between anger in depression and polymorphisms in genes implicated in serotonergic functioning, hypothalamic-pituitary-adrenal axis functioning, and emotional reactivity (Wasserman, Geijer, Sokolowski, Rozanov, & Wasserman, 2007).

Clinical Presentation and Treatment Response

In general, hostility in the context of major depressive disorder seems to carry negative prognostic value, and is associated with greater time spent ill. Compared with less hostile forms, those experiencing major depressive disorder and notable hostility may experience greater obsessive worry, paranoia, psychotic symptoms, and/or appetite increase but also may experience fewer somatic symptoms. Although anger seems to exhibit a positive correlation with depressive symptom severity, research evidence is mixed regarding anger as a predictor of suicidality.

Researchers have yet to explore the relative efficacy of any forms of psychotherapy for depression with and without anger. Several studies, however, have explored response to pharmacological treatments. Research suggests that monoamine oxidase inhibitors are more effective than placebo in reducing hostility in hostile depressed patients, whereas evidence for the efficacy of tricyclics in these patients is mixed. The treatment of depressed outpatients with the serotonin reuptake inhibitors (SSRIs) has resulted in decreased hostility as well as improvement in depressive symptoms. Treatment with SSRIs also seems effective in reducing anger attacks, but it is unclear to what extent they are effective in reducing depressive symptoms in those with major depressive disorder plus anger. Finally, hostile-depressed nonresponders to

antidepressant medication may respond when switched to treatment with mood stabilizers, but it is unclear whether mood stabilizers in combination with antidepressants would be beneficial in treating major depressive disorder with anger.

Conclusions

Anger in the context of depression seems to be an important construct in need of further exploration by researchers. Significant anger and/or hostility is experienced by about one-third of individuals presenting with a major depressive disorder. Although depression with anger seems to be related to certain personality features, biological correlates, and clinical features, it is not yet clear whether depression with anger or major depressive disorder plus anger would be better classified as a distinct subtype of major depressive disorder. Treatment research related to depression with anger is sparse, especially with regards to psychotherapy, but available evidence suggests some efficacy of certain antidepressant medications for reducing depressive symptoms and anger in those with angry or hostile depression.

It would be helpful for standard assessments for unipolar depression to also assess symptoms such as hostility and anger. However, there is little agreement in the literature about how to best define and measure these constructs. *Anger*, *hostility*, and *irritability* are used interchangeably by some to describe the same phenomena, whereas others reserve *anger* to indicate the affective construct that is distinct from, although related to, the cognitive construct of hostility and the behavioral construct of aggression. The lack of clarity in definitions of and instruments used to measure these constructs has caused confusion regarding the degree to which descriptions of “hostile” and “angry” depression overlap, and represents a significant roadblock to advancing our understanding of how anger and hostility affect clinical presentation and outcomes in depression with anger.

See also

Comorbidity

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Anxiety

Though anxiety and depression are distinguishable, as we discuss in more detail below, they overlap to a considerable degree and share important features. Thus, no consideration of depression would be complete without also addressing anxiety disorders and the relationship between depression and the anxiety disorders. With an estimated 29% of people suffering from an anxiety disorder at some point in their lives, the anxiety disorders as a group are the single most common form of psychopathology within the general population. There are several different anxiety disorders, and what unites these problems is the central role of anxiety in each of them. In addition, they tend to share some common features, often involving some form of avoidance and a tendency to selectively focus on threatening information. What distinguishes the various anxiety disorders from one another

is the focus of anxiety involved in each (Barlow, 2002).

Panic disorder (PD) is characterized by recurrent unexpected panic attacks, discrete periods in which intense fear is accompanied by strong symptoms of physical arousal, and a fear of future panic attacks. A common complication of PD is agoraphobic avoidance of situations in which it might be difficult to escape or receive help should a panic attack occur.

Specific phobias involve circumscribed fears of particular objects or situations often leading to avoidance behavior. Subtypes include animal type, natural environment type, blood-injection injury type, situational type, and other type.

Social phobia involves anxiety about being evaluated in social or performance situations. Concerns may be confined to particular performance or social situations (such as public speaking) or may be triggered by many types of social interaction.

Obsessive-compulsive disorder (OCD) is characterized by repetitive and intrusive thoughts or images that provoke anxiety and/or the compulsive performance of overt or covert rituals that serve to neutralize anxiety or distress.

Posttraumatic stress disorder (PTSD) involves anxiety about a particular past event that provoked intense fear, horror, or helplessness. This anxiety is manifested by reexperiencing the traumatic event, avoidance of stimuli associated with the trauma, and hypervigilance.

Separation anxiety disorder (SAD) is a childhood disorder that involves excessive fear or anxiety concerning separation from the home or an attachment figure.

Generalized anxiety disorder (GAD) involves experiencing excessive and uncontrollable worry or anxiety about a number of different issues. This worry is also typically accompanied by several other symptoms of anxiety such as insomnia, muscle tension, and feeling restless, keyed up, or on edge.

Most anxiety disorders are more prevalent among women, although OCD is equally prevalent among men and women

(American Psychiatric Association [APA], 2000). In general, anxiety disorders tend to onset relatively early in life, typically manifesting before late adolescence (APA, 2000). PD is unusual in that it most frequently begins in early adulthood, although panic attacks often onset by midadolescence. PTSD may occur at any age, including in childhood, following the occurrence of a trauma.

In adults, untreated anxiety disorders tend to be relatively chronic, with only 12% to 30% of adults exhibiting spontaneous remission. Rates of recovery are somewhat higher in adolescents, and the majority of youth anxiety disorders remit by early adulthood. However, early anxiety disorders, particularly those that develop in late adolescence, are a major risk factor for the development of anxiety disorders in adulthood (Pine, Cohen, Gurley, Brook, & Ma, 1998). Many disorders, such as PD, social phobia, GAD, and OCD, frequently exhibit waxing and waning symptom courses in which the exacerbation of symptoms may relate to stress (*DSM-IV-TR*; APA, 2000).

Comorbidity Between Anxiety and Depressive Disorders: Overlapping and Distinctive Features

Comorbidity at the individual level refers to the co-occurrence of disorders; comorbidity at the group level refers to the co-occurrence of disorders across individuals at a rate that is greater than what would be expected by chance alone. Countless studies attest to the high frequency of comorbidity between depressive and anxiety disorders (see Mineka, Watson, & Clark, 1998). That is, research suggests that the frequent co-occurrence of depressive and anxiety disorders cannot be completely explained by the chance pairings of several highly prevalent disorders. Instead, the relationships between depressive and anxiety disorders are more nuanced. It has been consistently shown that anxiety disorders

frequently precede depressive onset, but that depression rarely precedes anxiety. The complexity of the relationship between depressive and anxiety disorders has led many researchers to conclude that the presence of a common diathesis for both types of disorders best explains this relationship. This diathesis has been described in both phenotypic and genotypic analyses of depression and anxiety, briefly reviewed below.

At the phenotypic level, it is thought that depression and anxiety disorders consist of a common core of negative affect, along with factors that are more specific to each disorder. For example, a low positive affect or anhedonia factor is relatively specific to depression, while physiological hyperarousal is relatively specific to panic attacks (Mineka et al., 1998; Zinbarg et al., 1994). The common core of negative affect is believed to account for the high rates of comorbidity of anxiety disorders and depression. In contrast, the anhedonia specific to depression and the various factors specific to different types of anxiety may account for the fact that anxiety disorders and depression can be reliably diagnosed and distinguished from each despite their high comorbidity. Interestingly, it appears that one of the anxiety disorders, GAD, is more highly comorbid with major depressive episodes and dysthymia than it tends to be with the other anxiety disorders (Krueger, 1999).

Behavioral genetic research has yielded moderately consistent results, suggesting a genotypic model of depression and anxiety that is closely related to the phenotypic model described above. Results suggest that the genetic vulnerability for depression and anxiety consists of two separate, but related, factors. One of these genetic factors appears to be a vulnerability for major depression and GAD, which are genetically indistinguishable from one another. This genetic diathesis has been shown to be closely linked to neuroticism, the personality trait thought to make one vulnerable to negative affect—the common phenotypic core of depression and anxiety. PD, agoraphobia, and social phobia are related to this factor as

well but are even more strongly related to a second genetic factor. This second genetic factor also underlies specific phobias, which are largely unrelated to the first genetic factor, and it may best represent a vulnerability to the somatic symptoms of panic attacks (Kendler et al., 1995). The evidence suggesting genetic indistinguishability of GAD and depression, together with the phenotypic evidence showing that GAD is even more highly comorbid with depression than it is with the other anxiety disorders, may lead to a reorganization of our current diagnostic system. In this reorganization GAD would be grouped together with the major depressive episodes and dysthymia rather than with the anxiety disorders.

Treatment of Anxiety Disorders

Earlier we stated that untreated anxiety disorders in adults tend to be relatively chronic. Fortunately, the anxiety disorders are among the most treatable of all the psychiatric disorders (Barlow, 2002). The two forms of treatment for anxiety disorders that have the greatest amount of empirical evidence to support them are cognitive behavior therapy and medications.

There are three primary cognitive behavior therapy techniques for anxiety that are often combined into a comprehensive treatment package: cognitive restructuring, relaxation exercises, and exposure therapy. Cognitive restructuring consists of teaching the patient to (a) better identify thoughts, beliefs, and/or images that might be contributing to excessive anxiety, (b) challenge these thoughts with existing evidence and more balanced thoughts, and (c) subject these thoughts to empirical tests. Relaxation exercises include progressive muscle relaxation, diaphragmatic breathing, guided imagery, and more recently, mindfulness meditation exercises. While there are several varieties of exposure therapy, all involve intentionally confronting anxiety-provoking triggers and situations and replacing avoidance behavior with approach behavior. Imaginal exposure involves

confronting anxiety triggers in imagination, whereas in vivo exposure involves actual confrontation with anxiety triggers. Interoceptive exposure involves intentionally bringing on the sensations of anxiety, which can trigger further anxiety or fear, and is often used in the treatment of PD. Whereas cognitive behavior therapy does not always produce what might be considered a complete cure, the vast majority of people who complete a cognitive behavior therapy program for anxiety disorders experience substantial improvement.

Recent evidence has begun to suggest that the anxious patient's marital and marriage-like relationships and relationships with other close family members predict the patient's response to individual cognitive behavior therapy (e.g., Zinbarg, Lee, & Yoon, 2007). In particular, hostility expressed toward the patient by relatives or partners predicts higher rates of treatment dropout and poorer treatment outcome. In contrast, nonhostile criticism appears to predict better outcome. These results suggest that some form of family therapy may augment the effectiveness of treatment for at least some cases of anxiety disorders. Indeed, whereas the effects of family therapy remain largely untested in anxiety disorders, some forms of involvement of intimate partners in treatment for PD with agoraphobia has led to superior outcomes (Daiuto, Baucom, Epstein, & Dutton, 1998).

The most commonly prescribed medications for anxiety disorders currently include the selective serotonergic reuptake inhibitors and the benzodiazepines. Medications prescribed for anxiety are effective for many people and in most cases will begin to act more quickly than cognitive behavior therapy. Unfortunately, however, these medications sometimes have unpleasant side effects, and relapse rates are very high if the medications are withdrawn before the patient has had a course of cognitive behavior therapy. Moreover, cognitive behavior therapy is typically effective even in the absence of medications. Thus, many psychologists

consider cognitive behavior therapy the first-line treatment for most cases of anxiety disorders, while seeing medications as particularly useful when no trained cognitive behavior therapists are available, or when the patient does not have the time necessary to devote to cognitive behavior therapy or has not responded to a trial of cognitive behavior therapy.

RICHARD E. ZINBARG, AMANDA A. ULIASZEK,
AND ALISON R. LEWIS

See also

Comorbidity
Posttraumatic Stress Disorder

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Assessment of Depression

During the past century, close to 300 measures of depression have been developed. Collectively, they address one or more of the following assessment goals: screening, diagnosis and classification, and measuring symptom severity (Nezu, Nezu, McClure, & Zwick, 2002). Most are self-report inventories or questionnaires, whereas others require trained raters or clinicians to administer, score, and interpret. Some purport to be general measures of depression (i.e., all adults), whereas others focus on a particular population as demarcated by age (e.g., older adults), concomitant diagnosis (e.g., medical patients), or ethnic or cultural background (e.g., Latino adults).

Screening

In clinical settings (e.g., primary care settings), screening helps to answer the question of whether further assessment is warranted (e.g., “Is this person clinically depressed or suffering from a sleep disorder?”). For research purposes, screening helps to determine whether a given individual meets initial inclusion criteria (e.g., diagnosis of major depressive disorder) for potential participation in an investigation. Often cutoff scores have been derived to help make such decisions. Because of the need to be brief in nature, depression screening measures usually are self-report inventories, such as the Center for Epidemiological Studies Depression Scale and the Patient Health Questionnaire.

Diagnosis and Classification

An instrument designed specifically to help derive a diagnosis needs to contain content that corresponds to the criteria required by a formal diagnostic system, such as the various versions of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association [APA], 2000). It should also demonstrate good interrater agreement when administered by two or more independent evaluators assessing the same individual.

Further, such a measure should lead to an accurate differential diagnosis; that is, it should correctly denote not only what diagnosis a person qualifies for, but also, which diagnosis he or she does *not* qualify for (e.g., bipolar disease), especially when there is symptom overlap across diagnoses. An example of a clinician-rated diagnostic assessment procedure is the Structured Clinical Interview for DSM Disorders.

Assessing Symptom Severity

Measuring the severity of depressive symptoms is probably the most frequent assessment goal in both clinical and research settings. Such measures generally include a listing of symptoms thought to be important characteristics of depression across behavior (e.g., sleep patterns), affect (e.g., sadness), cognition (e.g., thoughts of suicide), and motivation (e.g., loss of pleasure) and require a rating of the presence or severity of such symptoms. To be useful in providing such information, measures of symptom severity especially need to demonstrate strong reliability over time. Common depression measures used for this purpose include both self-report questionnaires, such as the Beck Depression Inventory and the Zung Self-Rating Depression Scale, and scales administered by trained clinicians or raters, such as the Hamilton Rating Scale for Depression. Note that measures used for screening purposes often are additionally administered to obtain a rating of depressive symptom severity.

General Versus “Specific” Depression Measures

The vast majority of depression measures have been developed with the notion that they are applicable across multiple populations. However, as research continues to document the differences in etiology, topography, duration, and pathways that exist as a function of multiple individual difference characteristics, researchers have realized the need to develop measures of depression for

specific populations (Nezu, Nezu, Friedman, & Lee, 2009).

Age Differences

One patient variable of consequence is age. For example, not only would adult measures of depression be potentially inappropriate for children due to language differences, but they would also be inappropriate with regard to the potential divarications in the overall constellation of symptoms and their behavioral expression. As such, a variety of depression measures have been specifically developed for children and adolescents, such as the Children’s Depression Inventory. On the other end of the spectrum, depression measures have been specifically developed for older persons, such as the Geriatric Depression Scale.

Concomitant Diagnoses

A further potential individual-difference variable that can impact the accuracy of depression assessment is the presence of additional psychiatric or medical diagnoses. In certain cases, these concomitant problems can limit the validity of depression measures that do not take these disorders into account. An example includes the Calgary Depression Scale for Schizophrenia, a clinician-rated protocol that was developed in response to the observation that other assessment instruments for depression did not accurately represent depressive symptoms or syndromes in persons with schizophrenia. The Hospital Anxiety and Depression Scale was developed to provide a more valid assessment of these two types of distress constructs in order to reduce potential bias by not including items that address physical symptoms (e.g., sleep or eating problems). Other examples of measures specifically developed or validated to assess depression in various medical populations include the Depression in the Medically Ill Scale and the Cardiac Depression Scale.

Because patients with bipolar disorder are frequently misdiagnosed with major depression if seen during a depressive episode,

researchers have developed specific assessment procedures to differentiate between these diagnostic entities. Examples include the Screening Assessment of Depression–Polarity, a clinician-administered instrument, and the Mood Disorder Questionnaire, a self-report inventory.

Cultural Differences

Of particular importance regarding interindividual differences is the influence of differing ethnic and cultural backgrounds. Sensitivity to this concern first involves ensuring that a depression measure has been competently back-translated into a foreign language when used with a non-English-speaking sample. In addition, for a depression measure to be valid within a given sample, research needs to demonstrate that it actually addresses constructs that have meaning within that culture of interest. Whereas some similarities exist in the expression of depression across various cultures, differences occur with regard to predominate symptom features. In the United States, depression is often detected by sad mood or decreased interest in activities, whereas predominant depression features in Latino and Mediterranean cultures include headaches and “nerves,” fatigue and “imbalance” among Asian cultures, and “problems of the heart” in Middle Eastern countries (APA, 2000). Many measures of depression that were initially developed in Western cultures have, in fact, been demonstrated to be applicable across a variety of cultures and have been translated into numerous languages, such as the Beck Depression Inventory, the Patient Health Questionnaire, and the Zung Self-Rating Depression Scale. However, depression measures have also been specifically developed for non-Western cultures, including the Vietnamese Depression Scale and the Chinese Depressive Symptom Scale.

Future Directions

It is important to note that during the past several decades, the actual definition of

depression has changed, as documented across the various *Diagnostic and Statistical Manuals for Mental Disorders*. As such, an important future concern involves the need for current measures of depression to be revised or new measures to be developed in order for them to be consistent with such definitional changes (Nezu et al., 2009).

In addition, future research should focus on improving the assessment of depression among (a) differing ethnic populations, (b) medical populations experiencing symptoms that overlap with depression, (c) individuals residing in rural areas, (d) persons in lower socioeconomic levels and poor literacy rates, (e) the elderly (especially those experiencing cognitive difficulties), and (f) the disabled.

ARTHUR M. NEZU AND CHRISTINE MAGUTH NEZU

See also

Beck Depression Inventory
Clinically Useful Depression
Outcome Scale
Diagnostic and Statistical Manual of
Mental Disorders

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Attachment

In the third volume of his *Attachment and Loss* trilogy, Bowlby (1980) wrote about the way attachment insecurities may contribute to later development of depression. Bowlby argued that individuals who experience

negative interactions with their attachment figures (close relationship partners—usually primary caregivers during childhood—who provide protection, comfort, and support), are more likely to develop psychopathologies in general and depression in particular.

According to attachment theory, human beings are equipped with an innate attachment behavioral system, which regulates proximity-seeking behaviors. When encountering threats or stressors, infants are motivated to restore their sense of security by seeking proximity to their attachment figures. Over time, the attachment figures and the interactions with them are internalized as mental representations or working models. These mental representations include knowledge about the self, the relationship partners, and the social world.

A sensitive and responsive parenting style is likely to lead individuals to internalize positive representations of the self (as worthy of love and support) and others (as dependable and likely to provide love and support). Individuals holding such representations are thought to have a secure attachment style. An inconsistent or rejecting parenting style, on the other hand, is likely to lead individuals to internalize negative representations of the self (as not worthy of love) and others (rejecting, neglecting, unreliable, and not helpful). Individuals holding such representations are said to have an insecure attachment style. Once formed, these attachment styles are thought to be relatively stable and tend to influence people's cognitions, emotions, and behaviors.

Individual differences in adult attachment are most commonly conceptualized in terms of two dimensions of insecurity. The first dimension, attachment avoidance, results from encountering consistent rejections from attachment figures. It is characterized by a strong preference for self-reliance, reluctance to get close or show emotions to relationship partners, as well as discomfort with letting others depend on oneself. Avoidantly attached people tend to downplay their emotions in an attempt to deactivate their attachment system.

The second dimension, attachment anxiety, is thought to result from encountering inconsistent and intrusive caregiving behaviors. It is characterized by a strong desire for closeness to—and protection from—relationship partners, and a hypervigilance toward cues of partner rejection or unavailability. Anxiously attached people tend to ruminate on negative experiences, be preoccupied with negative thoughts and emotions, and present themselves as helpless and needy.

Currently, there are two main approaches to measure individual differences in adult attachment styles. One approach, mainly used by social psychologists, is based on self-reports. As research on attachment progressed, self-report measures evolved from assessing attachment in terms of three or four types (e.g., secure, anxious, dismissing-avoidant, and fearful-avoidant) to assessing it along the above-mentioned anxiety and avoidance dimensions. The other approach, mainly used by clinical and developmental psychologists, is based on interviews. The most widely used of these measures is the Adult Attachment Interview (AAI). In the AAI, adults are asked to describe their parents and the relationships they had with them during childhood. The responses are scored primarily in terms of discourse coherence rather than the content. The coherency of the overall responses is assumed to reflect the interviewee's "state of mind with respect to attachment" and is used to assign the interviewee into one of the three major "state of mind" categories (autonomous—corresponding to a secure style, preoccupied—corresponding to an anxious style, and dismissing—corresponding to an avoidant style).

To date, hundreds of studies have shown that individual differences in attachment style, measured either via self-reports or interviews, are correlated with relationship satisfaction, well-being, forms of coping with stress and regulating affect, and mental health. One of the central findings coming out of this literature is that attachment security provides a resilience resource that reduces the likelihood to develop psychological disorders.

Early Attachment Experiences and Later Vulnerability to Depression

Bowlby (1980) suggested that depression may result from a failure to form secure, supporting bonds with one's primary caregivers. This lack of secure bonds might be either due to the actual loss of a caregiver (because of death or prolonged separation) or due to rejection or inconsistent care from a caregiver. Individuals who experienced such events (e.g., prolonged separation or rejection), are more likely to form negative perceptions of the self, which include feelings of being abandoned, unwanted, unlovable, and unable to form and maintain affectional bonds. As a result, these individuals are at a higher risk to develop depression.

Studies that tested the effects of caregiving quality or separation during early childhood on later depression provided empirical support for Bowlby's ideas. For example, individuals who lost one or both of their parents in childhood (due to either separation or death) were found to be more likely to show depressive symptoms in adulthood as compared to individuals who did not experience such loss. Likewise, individuals who reported receiving insensitive caregiving from their attachment figures in childhood were more likely to show depression in adulthood. Beyond this correlational evidence, there is also experimental evidence showing that individuals who reported low-quality maternal caregiving were more likely to show an attention bias toward negative stimuli (which can be interpreted as an index of vulnerability to depression), as compared to individuals who reported high-quality maternal caregiving.

Individual Differences in Adult Attachment Style and Depression

Cross-Sectional Studies

Numerous studies focusing on individual differences in adult attachment style have found positive associations between attachment insecurities and depressive symptoms. In a recent comprehensive review, Mikulincer and

Shaver (2007) identified more than a hundred studies investigating this association in nonclinical samples, with most of them using self-reports to assess attachment style. Regardless of the attachment measure used, these studies consistently showed that anxious attachment was positively associated with depression in nonclinical samples. Results were more mixed with respect to avoidant attachment. Whereas some researchers reported positive associations between avoidance and depression, others reported null findings.

Mikulincer and Shaver (2007) noted that although anxiety seems to be more strongly associated with depression than avoidance, this discrepancy is less pronounced when researchers examined how anxiety and avoidance relates to different facets of depression. Thus, anxiously attached individuals' chronic preoccupation with emotional closeness and reassurance-seeking from relationship partners make them more vulnerable to interpersonal facets of depression, such as being overly dependent and lacking autonomy. Conversely, avoidant individuals' excessive preference for self-reliance makes them more vulnerable to intrapersonal facets of depression, such as perfectionism or self-criticism. Indeed, research has shown that attachment anxiety was positively correlated with dependency, concern about what others think, and pleasing others; whereas attachment avoidance was positively correlated with perfectionism, need for control, and defensive separation.

Studies conducted with clinically depressed individuals found similar positive associations between insecure attachment and depression. Using self-report measures, several studies found that fearful-avoidant individuals (those who are high on both attachment anxiety and avoidance) were more likely to suffer from major depression. Findings based on the AAI were less consistent. Whereas some researchers found individuals diagnosed with depression to be more often classified as having a dismissing state of mind, other researchers found that these individuals were actually more likely to be classified as having a preoccupied state of mind.

Various explanations were suggested to account for these conflicting findings. One of these explanations is related to the studies' inclusion criteria for the depressed sample. Dozier, Stovall-McClough, and Albus (2008) noted that studies excluding individuals with comorbid internalizing symptoms (such as symptoms of borderline personality disorder) found dismissing state of mind to be associated with depression. In contrast, studies excluding individuals with comorbid externalizing symptoms (such as symptoms of conduct disorder) found preoccupied state of mind to be associated with depression. Another explanation is the depression subtype. Research has shown that patients with bipolar disorder were more likely to be classified as dismissing as compared to patients with major depressive disorder or dysthymia.

Longitudinal Studies

In line with cross-sectional findings, longitudinal investigations also confirmed the hypothesis that insecure attachment predicts depressive symptoms. Using self-report measures, numerous researchers reported that during college years (a time of transitioning to young adulthood for many people), attachment insecurity prospectively predicts depressive symptoms assessed 6 weeks to 2 years later. Longitudinal studies conducted with participants experiencing other important life transitions also found a positive correlation of attachment insecurity and level of depressive symptoms. For example, women's attachment anxiety assessed prenatally was found to predict elevated postpartum depressive symptoms. Moreover, one's partner's attachment style was also found to affect one's own levels of depression. For example, husbands' attachment security predicted reduction in wives' depressive symptoms over a 6-month period; whereas husbands' avoidant attachment was found to be positively associated with the persistence of wives' depressive symptoms.

Direction of the Relationship

Although it is theoretically more plausible to expect that attachment insecurity leads

to depression, it may also be the case that depression heightens attachment insecurity. Findings from recent experimental studies have favored the former hypothesis over the latter one. In one of these studies, participants were primed with either the phrase "Mommy and I are one" (a prime which might create a sense of closeness to an attachment figure) or an attachment-unrelated control phrase, and then their depressive symptoms were assessed using a self-report measure. The negative correlation between attachment security and depressive symptoms was found to be stronger among participants primed with the attachment-related phrase as compared to those primed with the control phrase. This finding provides some support for the hypothesis that a sense of security lowers depressive symptoms. One explanation for this finding can be that the security prime strengthens the association circuits in memory related to maternal closeness and security, and weakens the association circuits related to negative self views.

A different way to examine the direction of the link between attachment and depression is by investigating whether experimentally increasing depressive mood would lower the sense of attachment security. In studies using this strategy, participants were exposed to either a depressive, neutral, or happy mood induction, and then their attachment styles were assessed. Results of these studies revealed that there were no self-reported or interview-based attachment style differences between the different mood induction conditions (depressed vs. happy or neutral). Taken together, these studies provide two preliminary conclusions: (a) There might be a causal link between attachment insecurity and depression; and (b) the direction of the link seems to be such that changes in attachment insecurity are likely to bring changes in depressive symptoms. Before a more decisive conclusion could be drawn, however, more empirical evidence showing that elevation of the sense of attachment security can reduce depressive symptoms is needed.

Process Models

Recently, researchers have started to investigate the process underlying the relationship between attachment insecurities and depression. This line of research identified many mediating factors. Among cognitive factors, low self-esteem, dysfunctional attitudes about one's self-worth, low self-reinforcement (ability to value, encourage, and support oneself), and maladaptive perfectionism were found to mediate the relationship between both types of insecure attachment and depressive symptoms. When looking separately at each of the insecurities, changes in self-efficacy beliefs, self-concealment (predisposition to conceal intimate and negative personal information), and self-splitting (inability to integrate different images of oneself) were found to mediate only the relationship between attachment anxiety and depressive symptoms, whereas incoherency and low emotional intensity of autobiographical memories were found to mediate the relationship between attachment avoidance and depressive symptoms.

Among interpersonal factors, negative events in interactions with close others (family members, peers, and romantic partners), and inability to meet autonomy and relatedness needs were found to mediate the association between both types of attachment insecurities and depressive symptoms. Loneliness and need for reassurance were found to mediate the association between attachment anxiety and depressive symptoms, whereas discomfort with self-disclosure was found to mediate the association between attachment avoidance and depressive symptoms. Similarly, different emotion regulation strategies were found to mediate the links between each of the attachment insecurities and depressive symptoms. Thus, emotional reactivity (a strategy characterized by hypersensitivity to stimuli in the environment) was found to mediate the association between attachment anxiety and depressive symptoms, whereas emotional cutoff (a strategy characterized by distancing from others in times of intense emotional experiences) was found to mediate the association

between attachment avoidance and depressive symptoms.

Unfavorable contextual factors were also found to moderate the association between attachment insecurities and depressive symptoms. Insecure people, who usually lack psychological resources to cope effectively with stress, would be expected to be more likely to develop depression when they face socioeconomic, environmental, or interpersonal stressors. In line with this reasoning, anxious women who experienced stressful life events were found to show higher levels of depressive symptoms as compared to anxious women who did not experience such stress.

Finally, dyadic factors may interact with attachment security to affect depressive symptoms. Thus, not only that one's attachment style may affect his or her partner's levels of depression (as mentioned above), but dyadic factors may moderate this link. For example, it was found in married couples that spouses' attachment insecurity was related to lower depressive symptoms when marital satisfaction was high than when it was low. Similarly, husbands' high social support and low anger weakened the effects of wives' attachment anxiety on their own postpartum depressive symptoms. When examining husbands' depressive symptoms, it was found that husbands' perceptions of their wives as unresponsive decreased the husbands' sense of attachment security, which, in turn, increased their depressive symptoms. Taken together, these findings indicate that having a secure and supporting partner mitigates the effects of having an insecure attachment style on depression; whereas having an insecure and unresponsive partner exacerbates these effects.

Attachment and Response to Treatment of Depression

Individual differences in attachment style were also found to be associated with responses to therapy aiming to alleviate depression. Studies conducted with people who participated in therapy programs for

major depression found that fearful avoidance was negatively associated with remission and positively associated with time to stabilization (time to consistently obtain low-depressive symptom scores) among remitted individuals.

EMRE SELCUK AND OMRI GILLATH

See also

Anaclitic and Introjective Depression
Early Adversity
Internal Working Models
Maltreatment

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Attention

Distractions constantly challenge our ability to stay on task—a “New E-mail Message” note appears on your computer screen as you attempt to work; a ringing cell phone distracts your driving. The ability to achieve and maintain goal-focused behavior in the face of distraction is critical for surviving and thriving in our world. This highlights the importance of attention, which is the ability to select what is most relevant for current task goals. Attention developed to help the brain solve a computational problem of information overload. For example, during perception of natural scenes, there is a multitude of incoming sensory input, which cannot all be fully analyzed by a limited-capacity perceptual system within the human brain. Under these circumstances, attention serves to restrict sensory processing

in favor of the most relevant subset of items in order to ensure that the behavior of the organism is guided by the most relevant information.

Attention is a multidimensional system known to be dysfunctional in depression (Ingram, 1990). Therefore, understanding the computational structure and neuroanatomical basis of attention is a crucial step in treating attentional dysfunction associated with depression. Here, we review what is known about attention from this body of research and introduce our work investigating the influence of mindfulness training on the attention system. Our initial studies suggest that mindfulness training may improve attention by improving the ability to select information. We explore the hypothesis that these attentional effects may contribute to the efficacy of mindfulness-based clinical interventions in the treatment of depression relapse.

The Human Attention System

Attention comprises three functionally and neuroanatomically distinct cognitive networks. These networks carry out the operations of alerting, orienting, and conflict monitoring. Alerting consists of achieving and maintaining a vigilant or alert state of preparedness; orienting restricts processing to the subset of inputs that are relevant for the current task goals; and conflict monitoring prioritizes among competing tasks and resolves conflict between goals and performance. Two basic paradigms have been used to investigate attentional subsystems: the attentional spatial cuing paradigm and the flanker paradigm (see Fan, McCandliss, Sommer, Raz, & Posner, 2002, for an overview).

Attentional spatial cuing paradigms provide a means to behaviorally index attentional alerting and orienting. In this paradigm, participants sit at a computer and perform a visual computer task similar to a simple video game. They are to attempt to detect a target that is presented after either informative or neutral spatial cues. Informative cues provide spatial information

regarding the target location with high probability. Neutral cues signal the imminent appearance of a target but provide no spatial information regarding its location. Neutral cues confer an attentional advantage when compared to no-cue trials. This advantage in performance is thought to be due to *alerting*. The neutral, so-called warning cue increases arousal signifying that a target is forthcoming. Comparisons of performance on trials with informative cues versus neutral cues assess orienting. Performance is typically fastest and most accurate for targets whose location had been correctly predicted by an informative cue (valid cue) and slowest and least accurate for targets whose location had been incorrectly predicted by the cue (invalid cue). The advantage in performance for valid relative to neutral cues, referred to as the validity effect, is due to orienting of spatial attention as directed by the cue prior to the target's appearance. The disadvantage in performance for invalid relative to neutral cues, referred to as the invalidity effect, is due to the cost of recovery of attention after orienting to the wrong location. The validity and invalidity effects have been used to further characterize the orienting subsystem as comprising attentional engagement at the cue location, disengagement when the cue location is misleading, and moving of attentional focus (often referred to as a spotlight) to the appropriate location after disengagement.

Flanker paradigms provide a means to behaviorally index conflict monitoring by selectively manipulating the presence or absence of response competition while keeping other task demands constant. In this simple visual computer task, a target is to be identified by a two-alternative forced-choice method (e.g., determine if the arrow "<" is left or right facing). The target is surrounded by task-irrelevant flankers that are either of the same response category (<<<) as the target or of another response category (>>>). Responses in trials in which the flanking stimuli indicate a different response than the central stimulus (incongruent condition) are significantly slower than those in trials in which all stimuli indicate the same response (congruent

condition). Longer response times are attributed to the need for greater conflict resolution and monitoring during incongruent relative to congruent trials.

Recently, the Attention Network Test (ANT) has been devised to identify behavioral and neural indices of alerting, orienting, and conflict monitoring during a single task (Fan et al., 2002). The task manipulates attentional cuing (valid, neutral, and no-cues) as well as the type of target (congruent or incongruent flanker). Alerting is indexed by subtracting performance measures on neutral cue trials from no-cue trials. Orienting is indexed by subtracting performance measures on spatial cue trials from neutral cue trials. Conflict monitoring is indexed by subtracting performance measures on congruent from incongruent target trials. Our studies of the effects of meditation training on attention use this task (see below).

Attention With Mindfulness Training

While the attention system developed over evolutionary history as a means of solving information overload from the external environment, mindfulness or meditation practices may have developed over human history as a means of solving the overload suffered by the fragile internal environment of our mental landscape as it becomes easily flooded by sensations, thoughts, emotions, and memories.

Numerous meditation texts distinguish between two disparate forms of attention, described as *concentrative* and *receptive*, that can be trained (see Lutz, Slagter, Dunne, & Davidson, 2008, for a review). One meditation practice used to cultivate concentrative attention begins with instructions to maintain attention on the breath. If attention wanders from the breath, it is to be gently returned. Another meditation practice involves instruction to experience the present moment without orienting, directing, or limiting attention in any way. Practitioners are to be receptive to any stimulus (e.g., sounds, lights, tactile sensations,

thoughts, memories, emotions) that engages attention while keeping awareness neutral and unreactive. This exercise is used to cultivate receptive attention. Thus, while attention to the present moment of experience is a critical aspect of meditation instruction, in general, the particular way in which this instruction guides practitioners to attend can differ across meditation exercises.

There are two dimensions along which meditation texts distinguish concentrative and receptive attention: (a) the aperture of attentional focus and (b) the intensity of attentional focus. Concentrative practices direct subjects to hold a very narrow focus on the contents of attention. Examples across meditation traditions include a repeated sound, an imagined image, or a specific body sensation such as the breath. These types of practices emphasize the narrowing of attention and are sometimes described as single-pointed practices, in that attention is to be focused very closely on a specific focus or a single point. Receptive practices, in contrast, direct subjects to hold a very broad aperture of attentional focus. For example, instruction may direct one to attend to “all sounds” without specifying the particular sound on which attention should be focused. The concept of an attentional aperture is akin to cognitive psychological discussion of the attentional “zoom lens” in which the spatial extent of attention is thought to be modulated intentionally (see Eriksen & St. James, 1986). The second dimension of attention described in mindfulness texts is the intensity of the attentional focus. Some traditions encourage a more intense focus on the object of the meditation, while others emphasize an effortless engagement of attention. Typically, concentrative practices have a narrow attentional aperture and a higher intensity of focus. Concentrative training is associated with facilitated ease of attentional engagement. In contrast, receptive attention has the features of having a broad attentional aperture, akin to keeping attention ready for some unspecified event, and having low intensity of focus, which results in a less-intense engagement and greater ease of

disengagement from a stimulus. Importantly, each of these dimensions is considered to be along a continuum.

In our recent work (Jha, Krompinger, & Baime, 2007) we examined the influence of meditation training on the functioning of specific attentional subsystems. Participants receiving meditation training in the form of participation in an 8-week mindfulness-based stress reduction (MBSR) course or a 1-month meditation retreat (attended by experienced meditators) performed the Attention Network Test before and after training. Their performance was contrasted with the performance of control subjects who were also tested at two time points. We investigated two main hypotheses: (a) We hypothesized that prior experience with, and training in, concentrative meditation techniques would correspond to greater efficiency in the functioning of voluntary top-down attentional selection. Positive support for this hypothesis was observed on two counts. First, retreat participants, who were experienced with concentrative meditation prior to training, demonstrated better conflict monitoring (reduced flanker interference), compared to meditation-naïve subjects. Second, after meditation training, MBSR participants improved in their orienting performance relative to control participants. Since both conflict monitoring and orienting are forms of voluntary attentional selection, our results suggest that concentrative meditation may indeed alter functioning of voluntary response- and input-level selection processes. (b) We hypothesized that prior experience with concentrative meditation may allow for the emergence of receptive attention after training. Receptive attention corresponds to attentional readiness and alerting. We found that after training, not only did retreat participants differ in their alerting performance compared to control and MBSR participants, but the magnitude of their alerting scores after training was correlated with prior meditation experience. Greater experience corresponded with reduced alerting scores, indicating that attention was in a more ready state when no warning about target onset

was provided. Thus, our results suggest that meditation training improves performance on specific conditions of the attention network test.

Attention, Mindfulness Training, and Depression

In recent years, a dysregulation of the attention system has been increasingly identified as a hallmark of clinical depression. Specifically, the attentional focus of depressed individuals tends to be inordinately self-related, and this self-focus may play a role in prolonging depressive symptoms such as rumination (Ingram, 1990). Rumination is also strongly associated with depression (Nolen-Hoeksema, 2000) and mindfulness trains individuals to refocus their attention in a manner that may counteract the tendency toward rumination (Ramel, Goldin, Carmona, & McQuaid, 2004). A related finding is that patients with depression appear to focus their attention on items in the environment with a negative emotional valence. These pieces of evidence have resulted in a focused effort to treat symptoms of depression with techniques designed to improve components of the attention system.

Mindfulness-based cognitive therapy (MBCT) has already been shown to reduce relapse and recurrence of depressive episodes in certain depressed populations (for a review see, Ma & Teasdale, 2004, and Segal, this volume). MBCT combines techniques from MSBR courses along with elements of cognitive behavioral therapy. As reviewed above, mindfulness-based techniques are known to modulate different aspects of the attention system, suggesting that the therapeutic effects of MBCT may arise from its ability to improve attention so that it is directed analytically at the self to improve awareness of negative thinking patterns. Future research on the interface of attention, mindfulness-training, and depression will better reveal the correspondence between changes in attentional engagement and disengagement tied to specific mindfulness-based techniques

that might result in the best clinical outcomes.

AMISHI P. JHA, MICHAEL J. BAIME, AND
KARTIK K. SREENIVASAN

See also

Memory Processes
Mindfulness-Based Cognitive
Therapy
Rumination
Self-Focused Attention

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Attention Deficit Hyperactivity Disorder

Children, adolescents, and adults diagnosed with attention deficit hyperactivity disorder (ADHD) have considerably higher rates of comorbidity with certain other psychiatric

disorders than would be expected from the base rates of those disorders in the population at large (Barkley, Murphy, & Fisher, 2008). At least 80% or more of ADHD cases have at least one other disorder, and 50% or more may have two other disorders (Barkley et al., 2008). Major depression is among those disorders having some relationship to ADHD, though the relative association of these disorders varies as a function of whether samples are children or adults and whether they are ascertained through clinic referrals or epidemiological samples.

Studies of Children

Symptoms of depression are often elevated among clinical samples of children with ADHD (Jensen, Martin, & Cantwell, 1997), with the highest levels occurring among those children having comorbid aggression (or oppositional defiant disorder/conduct disorder; ODD/CD). Symptoms reflecting low self-esteem, however, are chiefly associated with aggressiveness and particularly depression in ADHD samples and are otherwise not especially problematic when ADHD is found alone (Bussing, Zima, & Perwien, 2000).

A review of this association found between 15% and 75% of ADHD cases had depression as a comorbidity (Spencer, Wilens, Biederman, Wozniak, & Crawford, 2000). However, most studies reported rates of 9% to 32% of children with ADHD having major depressive disorder (Biederman, Newcorn, & Sprich, 1991). Up to 20% of children with ADHD seen in a pediatrics clinic and up to 38% of those seen in a psychiatric clinic may have comorbid major depressive disorder (Spencer et al., 2000). Pffnner and colleagues (1999) studied clinic-referred boys with ADHD and a control group referred to the same outpatient clinic but not diagnosed as ADHD. They found that while just 5% of ADHD cases had depression alone (vs. 11% of boys without ADHD), another 21% had depression with an anxiety disorder (vs. 15% of the psychiatric control group). All of this suggests a clear risk of depression or frank

major depressive disorder in 25% to 30% of children with ADHD.

Wilens et al. (2002) reported that dysthymia occurred in 5% of both preschool- and school-age groups, while major depressive disorder was diagnosed in nearly half of their samples, 42% and 47%, respectively—risks higher than found by most other researchers. Biederman, Mick, and Faraone (1998) have argued that this association reflects an overlap of two clinical disorders and that the depression evident in ADHD is not just a reflection of demoralization over failures in major life activities. Yet early longitudinal studies of children with ADHD followed to adulthood did not report significantly elevated rates of depression or other mood disorders (e.g., Spencer et al., 2000). A more recent follow-up study (Fischer, Barkley, Smallish, & Fletcher, 2002), in contrast, found that 27% of these children had major depressive disorder by young adulthood. This risk had dropped substantially by the age 27 follow-up, however, and its presence was mediated by CD in adolescence and young adulthood and the persistence of ADHD to age 27 (Barkley et al., 2008). However, Peterson, Pine, Cohen, and Brook (2001) found that ADHD was consistently related to depression across four follow-up periods from childhood to young adulthood.

The inverse relationship of ADHD risk in cases of depression in children is far less studied. But the weight of the available evidence suggests some elevated risk of ADHD among youths diagnosed with depression (Spencer et al., 2000). Early data suggest, however, that rates of ADHD were not significantly elevated, but levels of other disruptive behavior, such as oppositional and conduct problems were so.

Large studies of community samples can shed further light on the existence and nature of this comorbid relationship in children. In their meta-analysis of such studies, Angold, Costello, and Erkanli (1999) reported a median odds ratio of 5.5 for the comorbidity of ADHD and major depressive disorder, ranging from 3.5 to 8.4 and being significantly

greater than that seen between ADHD and anxiety disorders, noted above. Undoubtedly, then, ADHD and major depressive disorder show a greater level of association than expected by chance alone. But depression is also strongly associated with ODD/CD and anxiety, raising the possibility that it is the presence of one of these latter disorders that mediates the relationship between ADHD and major depressive disorder, as was suggested in the follow-up study by Fischer and colleagues (2002), where lifetime CD predicted occurrence of major depressive disorder. This was also evident in evidence provided by Angold and colleagues (1999), where the association of ADHD with depression was greatly reduced when controlling for comorbidity of ADHD with ODD/CD and with anxiety. In other words, the relationship of ADHD and depression may be an epiphenomenon (Angold et al., 1999) that arises only because of the association of ADHD with ODD/CD and ADHD with anxiety—absent these other two disorders, ADHD may not have an association with depression.

The comorbidity of depression along with ADHD is often associated with a poorer outcome than either disorder alone (Spencer et al., 2000). This comorbidity is also a marker for a history of greater family and personal stress, and greater parental symptoms of depression and mood disorders (see Jensen et al., 1997, and Spencer et al., 2000, for reviews). Though not well established, this group of comorbid children may respond better to antidepressants than do those ADHD children without comorbidity for internalizing symptoms (Jensen et al., 1997). Unlike anxiety disorders, major depression does demonstrate a familial linkage with ADHD such that risk for one disorder in a child predisposes to risk for the other disorder not only in these children but also among biological family members of the comorbid children (Biederman, Faraone, & Lapey, 1992). Thus, ADHD and major depressive disorder may share underlying familial etiological factors (Spencer et al., 2000). As noted above, though, CD is also elevated among these comorbid children and among their family members and could, in

part, explain the link of ADHD with major depressive disorder. Obviously the jury is not in yet on the reason why ADHD and depression share such an elevated comorbidity, but the overlap of both with CD provides one possible explanation.

Studies of Adults

Approximately 16% to 31% of adults meeting ADHD diagnostic criteria also have major depressive disorder (Barkley, Murphy, & Kwasnik, 1996). Indeed, one study of Norwegian adults with ADHD reported a lifetime prevalence of 53% and current prevalence of 9% for major depression (Torgersen, Gjervan, & Rasmussen, 2006), although its sample appeared to have quite severe ADHD, among other disorders. Dysthymia, a milder form of depression, has been reported to occur in 19% to 37% of clinic-referred adults diagnosed with ADHD (Murphy, Barkley, & Bush, 2002). Even so, a few studies comparing clinic-referred adults with ADHD to adults seen at the same clinic without ADHD did not find a higher incidence of major depression among the ADHD group (Barkley et al., 2008), questioning whether the link of ADHD and depression is partly an artifact of clinic-referral status. But most studies did find elevated ratings of depressive symptoms or even diagnosable dysthymia in ADHD adults relative to clinic and community control groups nonetheless, even if frank major depressive disorder was not present (Barkley et al., 2008). Thus, not only is the risk of major depression elevated in adults with ADHD, but this is even more so with its milder variant of dysthymia. Such an association of ADHD with risk for depressive symptoms and disorders was recently found in two large epidemiological studies of general population samples of adults, suggesting that there is true comorbidity between ADHD and these disorders (Kessler et al., 2006; Secnik, Swensen, & Lage, 2005). Rucklidge and Kaplan (1997) reported one of the few studies of women with ADHD and found them to report more symptoms of depression, anxiety, stress, and

low self-esteem, and a more external locus of control than did women in the control group. But psychiatric diagnoses were not reported in this study, making it difficult to compare to earlier research using such diagnoses.

In a study of parents of ADHD children who also have ADHD, Minde and colleagues (2003) did not find a greater prevalence of major depression relative to a control group of parents (15% vs. 8%). The study, however, used small samples, limiting its representation of parents with ADHD and its statistical power to detect group differences. It also did not find elevated rates of antisocial personality disorder, which might be a potential moderator between ADHD and depression. In contrast, the much larger study of parents with ADHD who also had children with ADHD by McGough et al. (2005) did find greater mood disorders than in their comparison group.

There is a significant elevation in risk for suicidal thinking in children with ADHD, particularly during high school (see Barkley et al., 2008, for a review). But ADHD is especially associated with risk of a suicide attempt during this period if the individual has contemplated suicide, perhaps due to the marked impulsiveness associated with ADHD. The likelihood of suicidal thinking and attempts appear to decline by age 27, though suicidal thinking in particular remains somewhat elevated over that seen in control samples. The risk for suicidality in ADHD cases is largely a function of the presence of major depressive disorder, and to a lesser extent CD and severity of earlier or concurrent ADHD symptoms (Barkley et al., 2008).

What of the inverse relationship of ADHD in cases of depression in adults? A study of adults with major depressive disorder found that 16% self-reported symptoms from childhood sufficient to warrant a retrospective diagnosis of ADHD, while 12% reported persistence of these symptoms into adulthood (Alpert et al., 1996). Both figures for ADHD risk are greater than population prevalence estimates for either children

or adults. But the relationship of ADHD in cases of depression seems to be considerably less striking than is the risk for depression in cases of ADHD.

In general, the weight of the evidence suggests a significant relationship between ADHD in adults and risk for depression, as it does in children with the disorder. This risk may be partly mediated by the comorbidity of both disorders with CD and antisocial personality disorder. While some genetic vulnerability to depression may exist in cases of ADHD and their biological family members, this may require exposure to repeated stress, social disruption, or other environmental disadvantage in order for depression to become manifest or to rise to the level of diagnosable major depressive disorder. These social factors are also associated with CD and antisocial personality disorder and could mediate this three-way linkage among disorders.

RUSSEL A. BARKLEY

See Also

Adolescent Depression
Childhood Depression
Externalizing Disorders
Internalizing Disorders

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Attributional Style

Attributional style, also called explanatory style, is a term used to describe the types of attributions, or causes, that people tend to give for events that happen to them. It is categorized along three dimensions: (a) internal-external (whether people tend to attribute the causes of things to themselves or others), (b) stable-transient (whether people tend to view the causes of events as remaining constant or as things that will change over time, and (c) global-specific (whether people tend to view the causes of situations as things that are present across situations or specific to individual situations).

Beck (1972) observed that when something negative happens “[a depression-vulnerable] individual interprets an experience as representing a personal defeat or thwarting; He attributes this defeat to some defect in himself. . . . He sees the trait as an intrinsic part of him. . . . He sees no hope of changing” (p. 278). Indeed, it has repeatedly been found that people who are depressed are more likely to make internal, stable, and global attributions for negative events than never-depressed people (see Sweeney, Anderson, & Bailey, 1986, for a review). For example, if a depressed person fails an exam, she might say that she failed the exam because she is stupid (internal), that she will always be stupid (stable), and that she is stupid in everything she does (global). However, in the same situation a nondepressed person might say that she failed the exam because her neighbors kept her up late the night before the exam (external), that this is unlikely to happen again

(transient), and that it will not affect her performance in other courses or other exams (specific). Conversely, depressed people are more likely to make external, transient, and specific attributions for positive events than are never-depressed individuals. If a depressed person receives an A on an exam, she might attribute the A to getting lucky (external), just this once (transient) and in just this class (specific), whereas a nondepressed person might attribute the A to being smart (internal), most of the time (stable) in most things she does (global). A meta-analysis of attributional style in children and adolescents has revealed similar findings in that higher levels of depressive symptoms are associated with internal, stable, and global attributions for negative events, and external, transient, and specific attributions for positive events (Gladstone & Kaslow, 1995).

Attributional style is most commonly assessed in adults using the Attributional Styles Questionnaire (Peterson et al., 1982) and the Expanded Attributional Styles Questionnaire-Revised (Peterson & Villanova, 1988). In children it is measured using the Children's Attributional Style Questionnaire (Kaslow, Rehm, & Siegel, 1984), the Children's Attributional Styles Questionnaire-Revised (Thompson, Kaslow, Weiss, & Nolen-Hoeksema, 1998), and the Children's Attributional Styles Questionnaire Interview (Conley, Haines, Hilt, & Metalsky, 2001). The measures ask people to imagine that a variety of hypothetical situations have happened to them and then to imagine the causes of those events. After causes are identified, people rate the imagined causes along internal-external, stable-transient, and global-specific dimensions.

The findings that depressed people make internal, stable, and global attributions for negative events are quite robust within the research literature. Some researchers have suggested that the depressive attributional style is a causal factor in depression. In addition, it has been proposed that it may characterize a "hopelessness subtype" of depression, and a depressive attributional

style is widely recognized as serving to maintain the length of depressive episodes.

There are several exceptions to the findings regarding depressive attributional style. Recent research suggests that flexibility of attributional style may be more predictive of depression than the types of attributions made (Fresco, Tytewski, & Craighead, 2007). In another study of dysphoric participants, it was found that those with predominantly hostile mood tended to make external attributions, while those with predominantly sad mood evidenced the more typical internal, stable, and global depressive attributional style (Scott, Ingram, & Shadel, 2003). Thus, it is possible that even within depression, mood states are important to consider when assessing attributional style.

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See also

Attributional Theories of Depression
Cognitive Theories of Depression
Hopelessness

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Attributional Theories of Depression

According to both the reformulated learned helplessness theory (Abramson, Seligman, & Teasdale, 1978) and the hopelessness theory (Abramson, Metalsky, & Alloy, 1989) of depression, different individuals possess different attributional styles: habitual ways of perceiving the causes of negative events.

Reformulated Learned Helplessness Theory

The reformulated learned helplessness theory (Abramson et al., 1978) operationalizes a pessimistic attributional style as the tendency to attribute the causes of negative events to internal, global, and stable factors. In contrast, the theory operationalizes an optimistic attributional style as the tendency to attribute the causes of negative events to external, specific, and unstable factors. Individuals with a pessimistic attributional style are posited to be at risk for developing depressive symptoms following negative events because they are more likely than other individuals to make depressogenic attributions for such events. Making depressogenic attributions increases the likelihood of developing helplessness expectancies—the belief that one has no control over the occurrence of future events. Once helplessness expectancies develop, depression is inevitable, as helplessness expectancies are viewed as a proximal sufficient cause of depression by the theory.

Hopelessness Theory

According to the hopelessness theory of depression (Abramson et al., 1989), individuals with a pessimistic attributional style exhibit the tendency to attribute the causes of negative events to global and stable factors, whereas individuals with an optimistic attributional style exhibit the tendency to attribute such causes to specific and unstable factors. According to the hopelessness theory, individuals with pessimistic attributional style are more likely than individuals without such styles to make depressogenic inferences about the causes of negative events. Making such inferences increases the likelihood of developing hopelessness—the belief that bad events will occur and good events will not occur coupled with the belief that one can do nothing to change the likelihood of such outcomes. Once hopelessness develops, depression is inevitable, since hopelessness is viewed as a proximal sufficient cause of depression by the theory. Although the focus of this entry is on attributional style as a vulnerability factor to depression, it is important to note that the hopelessness theory elaborates on the reformulated learned helplessness theory by delineating two additional cognitive styles that play a role in the etiology of depression. Individuals with pessimistic cognitive styles about consequences and the self exhibit the tendencies to view negative events as having many disastrous consequences and to view the self as flawed and deficient following negative events. In contrast, individuals with optimistic cognitive styles about the self and consequences exhibit the tendencies to infer that negative consequences will not follow from negative events and to believe that the occurrence of negative events in their life does not mean that they are flawed in any way.

Assessing Attributional Style

Traditionally, most researchers examining the attributional vulnerability hypothesis of the reformulated learned helplessness and hopelessness theories of depression have used

some variation of the Attributional Style Questionnaire (ASQ; Peterson et al., 1982) to assess attributional style. The ASQ is a self-report questionnaire that consists of 12 hypothetical life events: 6 negative events and 6 positive events. Participants are asked first to imagine that each event happened to them and next to write down a cause for each event's occurrence. Participants subsequently rate the cause on a 1 to 7 scale along three separate dimensions: internal versus external, global versus specific, and stable versus unstable. Separate composite scores are computed for attributional style for negative and positive events. For both composite scores, higher scores represent more internal, global, and stable attributional styles. As both the reformulated learned helplessness and the hopelessness theories of depression operationalize vulnerability to depression as consisting of the manner in which individuals tend to perceive the causes of negative events, the remainder of this entry focuses on attributional style for negative events. At the same time, it is important to note that although attributional style for positive events has not been implicated in playing a role in the initial development of depression, subsequent research and theory has suggested that it plays a role in the recovery from depression (Needles & Abramson, 1990).

In order to increase the reliability of the original ASQ, expanded versions of the measure containing 12 (Metalsky, Halberstadt, & Abramson, 1987) or 24 (Peterson & Villanova, 1988) hypothetical negative events have been developed. Similarly, to assess cognitive styles about consequences and the self, in addition to attributional style, Abramson, Alloy, and Metalsky (1988b) developed the Cognitive Style Questionnaire. Consistent with the etiological chains proposed by both the reformulated learned helplessness and hopelessness theories of depression, these newer measures only assess attributional style for negative events.

To date, the majority of research examining the attributional vulnerability hypothesis in youth has utilized the Children's Attributional Style Questionnaire

(CASQ; Seligman et al., 1984). The CASQ is a forced-choice questionnaire containing 48 items, each consisting of a hypothetical positive or negative event and two possible causes of the event. Children choose the option that best describes the way they would think if that event happened to them. The two choices for each item hold constant two dimensions of attributional style while varying the third. In order to increase the reliability of the CASQ as well as its appropriateness for use with younger children, researchers have recently developed the Children's Attributional Style Interview (CASI; Conley, Haines, Hilt, & Metalsky, 2001). Similar to the ASQ, the CASI allows youth to generate their own causal attributions and then rate each attribution on continuous scales of internality, stability, and globality. In order to make the CASI appropriate for young children, events are illustrated pictorially, and children rate their attributions using a sliding pointer on large, colorful scales with icons representing the concepts of internality, stability, and globality.

Prospective Studies Examining the Association Between Explanatory Style and Depression

Initial research examining the reformulated learned helplessness and hopelessness theories of depression focused on examining either the cross-sectional association between attributional style and depressive symptoms or the main effect of attributional style on change in depressive symptoms over time. Critical reviews of the literature, however, argued that such early studies provided an inadequate examination of the theories, as they failed to examine the theories' central hypothesis: individuals with a pessimistic attributional style are only more likely than other individuals to experience increases in depressive symptoms in the face of negative events; in the absence of such events, individuals with a pessimistic attributional style are no more likely than others to exhibit depression

(Abramson, Alloy, & Metalsky, 1988a; Alloy, Hartlage, & Abramson, 1988). In response to such critiques, the field saw a major shift in methodologies used to test these theories, and prospective diathesis-stress designs became the gold standard. Recent comprehensive reviews of the literature have demonstrated that although inconsistencies exist in the findings of studies, the majority have provided support for the hypothesis that a pessimistic attributional style confers vulnerability to the development of depressive symptoms following the occurrence of negative events in children (Abela & Hankin, 2008), adolescents (Abela & Hankin, 2008), and adults (Abela, Auerbach, & Seligman, 2008).

JOHN R. Z. ABELA AND BENJAMIN L. HANKIN

See also

Attributional Theories of Depression
Cognitive Theories of Depression
Hopelessness

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Automatic and Controlled Processing in Depression

John, a 37-year-old man, has suffered several depressive episodes throughout his life. After a problematical marriage, he and his wife, Anne, divorced. He worked as a receptionist when, 3 months ago, the last depressive episode took place. "I think the reason for this last episode was that I felt lonely and there were too many women working at my office. I used to see lots of women but I seemed invisible to them. Furthermore, while trying to work or to chat with any female colleague, I could not get rid of the scene of my last argument with Anne, and this happened over and over, invading my mind without my being able to do anything to avoid it." When asked about the meaning of this situation, John told us: "It reminded me over and over what I will never be able to get: I will never have a wife, no woman likes me. . . . It's just impossible. My life has been a failure and will always be a failure. . . . It's obvious, I'm not good-looking, I look so miserable . . . pathetic, it's always been that way."

In his cognitive model of depression, Aaron Beck (1976) suggested that depressed people generate negative products such as images (e.g., “the scene of our last farewell appeared over and over”), thoughts (e.g., “I’m not good-looking”), and inferences (e.g., “I will never have a wife”) automatically. These automatic products, which are usually assessed via the verbalizations of the depressed person, would be the reflection of underlying automatic processes. Beck’s ideas regarding the processes by which automatic products are generated paralleled the definitional criteria for automatic versus controlled processing developed by cognitive theorists in the 1970s. Automatic processes are operations that (a) take place without requiring attention or conscious awareness, (b) occur in parallel without interfering with other operations or stressing the capacity limitations of the cognitive system, and (c) occur without intention or control. However, effortful processes (a) require attention and thereby take place serially, inhibit other pathways, and are influenced by cognitive capacity limitations; (b) improve with practice; and (c) can be used to produce learning (Hartlage, Alloy, Vázquez, & Dyckman, 1993).

Different lines of empirical research have consistently found that, in fact, depressed persons have difficulty planning, initializing, and monitoring complex goal-directed behaviors in the face of distracting negative information (see Hartlage et al., 1993). Thus, in a broader sense, depressed individuals seem to show a reduced executive control, which leads them, for example, to being unable to control or adequately redirect their attention when negative thoughts or images appear (e.g., “I could not get rid of the scene of my last argument with Anne”) or even to retrieve rather overgeneral negative autobiographical memories (e.g., “My life has been a failure”).

Are automatic processes also affected in depression? Although the experimental evidence is less robust in this case, there is also empirical evidence suggesting that, in depressed persons as well as in depression-prone individuals, negative cognitions are

more easily activated than in normal participants (Lau, Segal, & Williams, 2004). Although clinical observations are not a direct way to measure cognitive processes, John’s description of his own mental experiences describes many instances in which negative cognitions seem to be easily activated, and, moreover, he also seems to have difficulties to inhibit those processes once initiated (i.e., problems with controlled or effortful processes). This distinction of a dual processing mechanism has proved to be a valuable tool to understand diverse findings in the literature on various cognitive processes in depression (attention, memory, thinking, etc.).

According to the differentiation of automaticity and control, the statement that biases in depression and anxiety operate on different processing levels that correspond to different cognitive tasks has become a dominant theme. Thus, it has often been stated that anxiety states are more closely related to biases in the automatic processing of threatening material (particularly reflected in attentional tasks), whereas depressive states are characterized by biases in operations of controlled processing (especially reflected in biases of memory; e.g., Matt, Vázquez, & Campbell, 1992). However, some caution is required when establishing a division of this kind. In fact, in most cognitive tasks, there are aspects that require both controlled and automatic processing. For example, although attention has traditionally been considered an automatic process, it also implies effortful processing, such as disengagement. Actually, depressed patients as well as recovered depressed patients seem to have difficulty moving attention away from negative information once it is presented (Joorman & Gotlib, 2007). Moreover, the data about this depression-versus-anxiety dichotomy reflecting different ways of processing are not as conclusive as has often been stated.

Vulnerability and Automatic and Controlled Processing

Frequently, the onset of a depressive episode is found to be associated with a stressful

event; then again, people who have suffered depressive episodes in the past seem to be more sensitized to the appearance of stressors, so that minor events, of apparently little consequence, can be responsible for triggering a depressive episode. In the case of John, seeing women triggers a series of automatic thoughts about himself and his life, facilitating a depressive mood, which in turn fuels those negative cognitions in a perpetuating way. Segal, Williams, Teasdale, and Gemar (1996) invoked the concepts of kindling and sensitization to explain this heightened susceptibility to recurrence of previously depressed individuals. In the process of kindling, the continued reactivation of negative memory structures would produce dense interconnections, so that activation of one element in the array is likely to activate the entire structure; this means that only minimal cues are needed to activate the array of depressive constructs. In the process of sensitization, the repeated activation of depressotypic constructs during previous depressive experiences produces the lowering of the activation threshold for these structures. Hence, the phenomena of sensitization and kindling (see Monroe & Harkness, 2005) can be interpreted in terms of a decrease in the threshold of automatic activation of cognitive processes and, in parallel, a greater difficulty for the executive processes to counteract them, which can create escalating spiraling cycles of negative cognition-emotion.

Finally, although more investigation is needed about the precise role of the cognitive mechanisms in the onset and relapse of depression and its relation with the processes of kindling and sensitization, the distinction between controlled and automatic processes is probably a way to fruitful analysis. The results of some recent high-risk prospective studies show that the onset of depression is more likely when there is a confluence of negative cognitive schemas (about oneself, the world, and the future) together with a tendency to process information ruminatively (i.e., Alloy et al., 2006). Thus, more spontaneity in generating negative thoughts and more difficulty in controlling them and redirecting processes

toward specific contents seem to be powerful candidates to explain the risk of onset, relapse, and recurrence of depression.

Implications for Therapy

The importance of working with automatic thoughts was emphasized by cognitive depression therapy from the beginning. The goal is, firstly, to make clients aware of the automatic thoughts that come into their minds and of the underlying dysfunctional beliefs. A common way of achieving this is to record automatic negative thoughts. Secondly, such thoughts are challenged with alternative, more adaptive and functional thoughts. The idea is to get clients to have “second thoughts” about thoughts that automatically come to their minds and that, following repeated and disciplined practice, these automatic thoughts will be countered. However, there are few data about the cognitive mechanisms involved in effective therapeutic interventions and, especially, whether there are real changes in the *ways* of processing (Beevers, 2005). Future research will have to show whether the efficacy of cognitive interventions in depression is based on the direct reduction of the automaticity of the negative cognitive processes, or on the teaching of metacognitive skills that serve to render this type of automatic processing more accessible to effortful reflection. Approaches such as mindfulness-based cognitive therapy for depression, or acceptance and commitment therapy help patients to deliberately monitor and observe their thinking patterns when they feel sad and to respond to these thoughts and feelings in a way that allows them to disengage from the cognitive consequences of automatically activated mood-related rumination (Segal, Williams, & Teasdale, 2002). Given that these automatic processes seem to be related to depressive relapse, effective prophylactic interventions should involve attempts to deautomatize such processes. Future investigation must attend to the cognitive mechanisms that underlie therapeutic improvement and determine the most feasible and effective ways (i.e., reducing automaticity vs. increasing executive

control) to improve interventions and reduce the likelihood of relapse.

CARMELO VÁZQUEZ AND LAURA HERNANGÓMEZ

See also

Attention
Memory Processes
Mindfulness-Based Cognitive
Therapy

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Automatic Thoughts

Automatic thoughts are an important part of Beck's cognitive model of depression. Automatic thoughts, called self-talk by some theorists, reflect the subconscious mono-

logue in which individuals are constantly engaged. In addition to self-talk, automatic thoughts can also consist of images. The idea that these thoughts are subconscious suggests that individuals have little ongoing awareness of these thoughts, but that they easily can be brought into conscious awareness. In depression, automatic thoughts are negative in nature and reflect deeper levels of cognitive activity, such as the operation of cognitive self-schemas. Early in the process of cognitive therapy, individuals are taught to monitor these thoughts, and once they can be monitored, the therapist teaches the depressed patient to challenge, and presumably alter, automatic thoughts. Modifying such thoughts has therapeutic value in its own right, but another reason for focusing on such thoughts is to gain access to the negative cognitive schemas of the depressed patient, with the idea that these schemas can be modified in a more adaptive manner.

As a cognitive variable comprising self-statements, the assessment of automatic thoughts must rely on self-report questionnaires. The Automatic Thoughts Questionnaire (ATQ-N) was developed by Hollon and Kendall (1980) as a way to measure these thoughts. Examples of items include statements such as “I feel like I am up against the world” and “I've let people down.” Individuals are asked not whether they believe these statements to be true or not, but rather to rate the frequency that these, and similar thoughts, occur over a period of time. The ATQ-N includes 30 such statements, with each statement rated on a 1 (never) to 5 (all time) scale, with scores ranging from 30 to 150. A variety of studies have shown that the ATQ-N is a valid and reliable measure.

The ATQ-P was developed by Ingram and Wisnicki (1988) as a counterpart to the ATQ-N, with a focus on the frequency of positive thoughts. Like the ATQ-N, the ATQ-P includes 30 statements (e.g., “My future looks bright”) to which respondents rate the frequency of occurrence on a scale of 1 to 5. The ATQ-Revised (ATQ-R) was developed by Kendall, Howard, and Hays (1989), also

as a way to examine positive thoughts. Unlike the ATQ-P, the ATQ-R adds 10 positive statements to the ATQ-N. Ingram, Kendall, Siegle, Guarino, and McLaughlin (1995) compiled data that showed the ATQ-P to be reliable and valid, and Burgess and Haaga (1994) reviewed data suggesting that the ATQ-P and the ATQ-R are roughly equivalent measures positive automatic thoughts.

RICK E. INGRAM

See also

Aaron Beck
Cognitive Behavioral Therapy
Cognitive Theories of Depression

Rumination
Self-Focused Attention

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